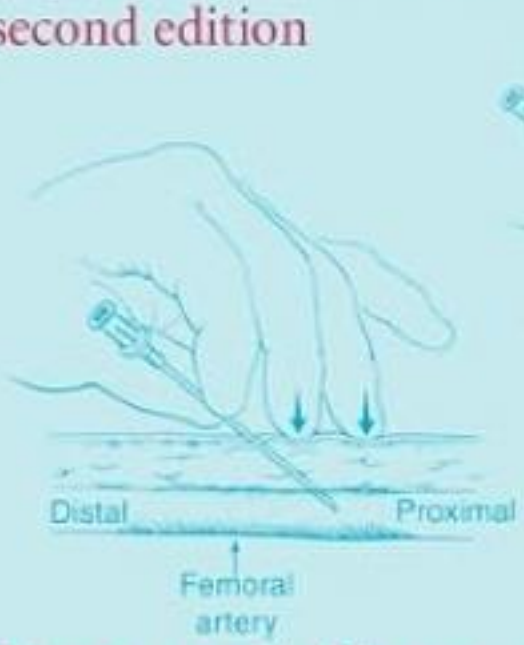
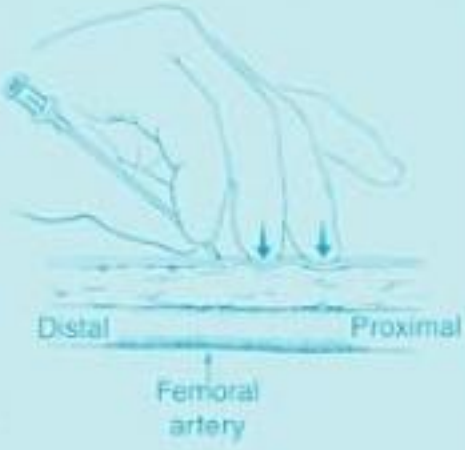
The Interventional Cardiac Catheterization HANDBOOK

second edition





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To
my wife, **Margaret**, and daughter, **Anna Rose**,
who have made my work the joy that it is

PREFACE

to the Second Edition

Seven years have elapsed since the first edition of this book, a companion to *The Cardiac Catheterization Handbook* for diagnostic catheterization techniques. This handbook is intended to provide a general approach to cardiovascular interventional procedures, emphasizing the mostly commonly used strategies and techniques currently available. By design we have limited the presentations of novel or unusual devices that may have only transient and niche application to our common interventional problems.

The beginning operator will benefit by gaining a detailed understanding of guide catheter selection, methods of guidewire use, and balloon and stent delivery. Often more important than equipment selection is the recognition and management of complications. Potential problems related to optimal stent placement and deployment, maintaining patency of side branches, reducing embolization, and no-reflow are discussed. In the era of costly drug-eluting stents, the approach to multivessel disease with selection of appropriate lesions by objective data using fractional flow reserve has distinct clinical and economic advantages. A detailed examination and discussion of intravascular ultrasound imaging and fractional flow reserve for lesion assessment is presented.

Care of the interventional patient after the procedure has also been updated to include techniques of best vascular access and closure with vascular sealing devices. A review of basic angiographic views best used for various interventional presentations has been extended from the standard diagnostic methods.

Sections on special techniques such as valvuloplasty, alcohol septal ablation for hypertrophic obstructive cardiomyopathy, thrombus aspiration and distal embolic protection, atherectomy,

balloon pericardiocentesis, foreign body retrieval, and closure of percutaneous septal defects have been added and updated. Several new sections dealing with brachyvascular therapy, drug-eluting stents and their biologic mechanisms, the pharmacotherapy of thrombosis, and management of difficult angioplasty situations have been designed to assist in answering issues raised in certifying examinations and reviews of critical clinical problems.

It is my hope that the useful fundamentals presented here will carry forward to new catheter-based approaches to improve the training of our interventional fellows, nurses, and technicians, and provide a helpful review for experienced physicians in their quest to provide the best care for our patients.

I would like to thank my coauthors who assisted in the preparation of this book, the cardiology fellows in Saint Louis University, the nurses and technicians in the J. Gerard Mudd Cardiac Catheterization Laboratory, and my assistant, Tamara Musgrove, and Sherry Karstens for manuscript preparation.

Morton J. Kern
October 1, 2003
St. Louis, Missouri

PREFACE

to the First Edition

As a companion to *The Cardiac Catheterization Handbook*, we have attempted to provide an introduction to the complex techniques of coronary and peripheral arterial intervention. We have directed the materials toward trainees in interventional cardiology who have completed their basic diagnostic catheterization training and who likely have seen or have been superficially exposed to angioplasty. As in *The Cardiac Catheterization Handbook*, we have also included illustrations and explanations for the more junior cardiologists, nurses, technicians, and associated industry personnel unfamiliar with these techniques who require a background and reference in interventional cardiology.

Several basic aspects of cardiac intervention, such as arterial access and arteriography, are reviewed with the understanding that the detailed descriptions have been provided in *The Cardiac Catheterization Handbook* or other more complete catheterization reference texts. This work emphasizes the approach, indications, contraindications, and methods of performing most of the standard interventional techniques available at the time of publication. This work is not intended to be a comprehensive presentation of all aspects of interventional cardiac catheterization wherein the reader is referred to more definitive works. Likewise, the author's bias is presented with anticipation that most coronary angioplasty methods have stood the test of time and that the approach for stent placement has gained wide acceptance. The brief discussion of laser angioplasty represents the limited experience of most interventionalists with an expensive and evolving technique that has not settled into the daily repertoire of our laboratory.

This work could not have been produced without the steadfast and excellent help of Donna Sander and the inspiration

and motivation from the fellows-in-training who continue to make the catheterization laboratory a valuable service for patients and stimulating for both attending and fellowship physicians alike. I would like to acknowledge the contributions of my coworkers in the J. Gerard Mudd Cardiac Catheterization Laboratory, with a special thank you to Margaret and Anna Rose for patience and forbearance in both St. Louis and Paris.

Morton J. Kern

July, 1996

INTRODUCTION TO INTERVENTIONAL CARDIOLOGY

Morton J. Kern

The discipline of interventional cardiology is now a subspecialty certified by the American Board of Internal Medicine. Extensive databases can be examined and applied by practitioners in the field. Interventional cardiology combines technically mastered skills and extensive cognitive information. Several major aspects and the application of the knowledge base are key to excellent outcomes in clinical practice.

THE SELECTION OF PATIENTS FOR INTERVENTIONAL PROCEDURES

The interventional physician should have a complete understanding of the indications and practice guidelines for percutaneous coronary interventions (PCI), coronary bypass graft surgery, and medical therapy for both coronary and peripheral arterial disease. Practitioners should know the natural history of patients treated for ischemic heart disease by each interventional modality for various patient subsets. The physician should also know the survival benefit and evidence for symptomatic improvement with these techniques. Specifically, the physician should know the following:

- The chance of successful PCI
- The benefit to the patient if the procedure is successful
- The occurrence of various complications
- The methods for dressing complications should they arise
- The overall risk to the patient.

These facts must be integrated into judgments that are specific for a given patient for noncoronary intervention such as

balloon valvuloplasty and renal and peripheral artery stenting, and the interventional cardiologist should understand the indications for the procedures, their complications, and outcomes.

TECHNICAL CONSIDERATION FOR THE PERFORMANCE OF PCI

No area of cardiology has had greater need for online decision making while performing a procedure than the PCI method. Similar to the surgical techniques, the planning and execution of PCI require extensive understanding of the options, limitations, and alternative methods of proceeding if the initial approach fails.

In addition, one must be prepared to take immediate measures to avoid complications and to manage complications promptly and effectively should they arise. Experience with different types of guiding catheters, guidewires, balloon catheters, stents, intravascular ultrasound imaging, and a host of FDA-approved nonballoon interventional devices is required. The operator must know the equipment's physical and material properties and the handling characteristics of intravascular devices, both large and small. Skill in the techniques for bending and shaping guidewires and maneuvering them throughout the three-dimensional structure of the coronary arteries is necessary. Knowing the characteristics of balloon catheters and stents, including profile, tractability, compliance, and strength, is essential for optimal success.

INTERVENTIONS AND THE VASCULAR HEALING RESPONSE

Understanding plaque biology and the vascular response to injury is important for device selection. The biologic process of vascular healing and restenosis should be appreciated in regard to the mechanical damage and stretch inflicted on the target vessel. The effect of the depth of vascular injury, the occurrence of elastic recoil, fibrointimal proliferation, migration of cells, matrix formation, and chronic vascular constriction converge to produce a restenotic lesion. The influence of growth factors, vasoactive substances, oxidative stress, and genetic signals in this healing process must be appreciated for best outcomes.

These issues impact directly on the use of drug-eluding stents and their ultimate results.

MANAGING COMPLICATIONS

Most complications can be prevented or at least minimized with proper planning, execution, and patient selection. When complications arise, prompt recognition and decisions to minimize the consequences are required. Newly forming thrombus, dissection, wall hematoma, and vasospasm require differentiation and treatment. Intravascular ultrasound imaging is a useful technique for diagnosing intravascular pathology. The use of intravascular ultrasound and translesional physiology, specifically fractional flow reserve (FFR), for selecting which lesions do and do not require treatment should be part of the complete interventional cardiologist's practice.

The knowledge and technical skill required for performance of interventional procedures greatly exceeds that required for diagnostic catheterization. Such knowledge and technique is acquired only through performance of many procedures under expert supervision. The proper use of guidewires, balloons, stents, and other interventional devices will reduce the potential complications and provide the operator with knowledge of how to manage dissection, thrombus formation, severe myocardial ischemia, emboli, and so on.

Although learning the general coronary anatomy is a component of the 3-year cardiology curriculum, to perform interventional procedures optimally the operator must know more detail. The relationship of the coronary artery ostia with an abnormal aortic root and other of the multiple anatomic variations must be understood. For example, decision analysis and a grading angiographic data often require supplemental views before deciding on coronary interventions. Specialized radiographic projections will display many details not analyzed through a routine diagnostic angiography, including lesion length, eccentricity, filling defects suggestive of thrombus, parent vessel angulation, bifurcation point location, and plaque mass. These influence the crucial decisions on how to proceed. Knowledge of coronary anomalies, coronary steal, the function of collaterals on flow dynamics, and myocardial perfusion is also important. Physiologic parameters, which

include pressure gradients across coronary and other vascular or valvular obstructions and, at times, the phasic coronary flow velocity measured with sensor-tip guidewires to determine myocardial flow reserve for a given region, are also helpful for accurate decisions.

EXPANDED KNOWLEDGE OF PCI FOR ACUTE MYOCARDIAL INFARCTION

The operator should know the indications for thrombolysis compared to acute catheterization and primary PCI, indications for PCI combined with agents facilitating thrombolysis, and indications for urgent bypass surgery in such individuals.

Decisions about PCI in high-risk subgroups, including patients with cardiogenic shock, should be made using supporting data involving myocardial viability, stunning, hibernation, and mass of myocardium at risk during the procedure.

The operator should know how to manage patients with acute hemodynamic instability with appropriate vasoactive drugs, intra-aortic balloon pumps, cardiopulmonary support, and emergency pacing, and the indication for emergency coronary artery bypass graft surgery. Interventional cardiologists should also have an understanding of the clotting cascade, platelet function, thrombolysis, and the function of antithrombus and antiplatelet agents. Pharmacologic methods that alter clot formation such as antiplatelet antibodies, specific antithrombus and peptides, are important in thrombus-prone patients. The operator should know how to manage hemorrhagic complications, including techniques with femoral artery compression, the use and results of vascular closure devices, and percutaneous techniques for diagnosing and closing pseudoaneurysms. Vascular access and puncture site closure devices and the associated complications must be well understood. Recognition of bleeding complications at various sites, especially retroperitoneal, gastrointestinal, and pulmonary or cerebral hemorrhage is mandatory. The interventionalist should know the best tests to measure hemostasis. Decisions about which patients require urgent vascular surgery consultation are essential in the practice of interventional cardiology.

EXPANDED KNOWLEDGE OF RADIATION SAFETY AND RADIATION VASCULAR BIOLOGY

Given the increase exposure time during interventional cardiology procedures, the operator should have an increased understanding of radiation physics and measures to assure optimal radiation safety in the laboratory. The physician should use methods to reduce radiation exposure to the patient and the technical staff. Using radiation to treat in-stent restenosis with brachytherapy relies on an understanding of both radiation biology and physics. Although every interventional cardiologist will not be performing every interventional procedures, these physicians and their staff should have a broad understanding of the field to be effective caregivers as well as consultants to other cardiologists.

The ever-expanding fund of knowledge that supports interventional cardiology has led to the American Board of Internal Medicine and the American College of Cardiology establishing guidelines and requiring at least one extra year of specialized training for certification in this field. The appendix lists the requirements for board certification in interventional cardiology.

Suggested Readings

American College of Cardiology/American Heart Association. ACC/AHA guidelines for percutaneous coronary intervention (Revision of the 1993 PTCA guidelines)—a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee to Revise the 1993 Guidelines for Percutaneous Transluminal Coronary Angioplasty) endorsed by the Society for Cardiac Angiography and Interventions. *Circulation* 2001;37:2215–2238.

American College of Cardiology. Recommendations for the assessment and maintenance of proficiency in coronary interventional procedures: statement of the American College of Cardiology. *J Am Coll Cardiol* 1998;31:722–743.

APPENDIX IA: CERTIFICATION IN INTERVENTIONAL CARDIOLOGY

BACKGROUND

Certification in interventional cardiology is a program for Diplomates in cardiovascular disease, and is designed to recognize excellence among physicians who are specialists in interventional cardiology. Participation is voluntary. Certification is not required of practitioners in this field, and the Board's certificate does not confer privilege to practice.

CERTIFICATION REQUIREMENTS

All candidates for certification in interventional cardiology must hold a current ABIM certificate in cardiovascular disease and a valid, unrestricted license to practice medicine. Candidates with restricted, revoked, or suspended licenses in any jurisdiction will not be admitted to examination. In addition, candidates must meet training requirements, clinical competence requirements, and pass a secure examination.

TRAINING PATHWAY

The training pathway is available only to candidates who completed acceptable interventional cardiology fellowship training in 1997 or later. This pathway requires 12 months of satisfactory fellowship training in interventional cardiology in addition to the required 3 years of cardiovascular disease training.

Interventional cardiology training taken July 1, 2002 and thereafter must be accredited by the Accreditation Council for Graduate Medical Education (ACGME). Interventional cardiology training undertaken prior to July 1, 2002 must be conducted within an accredited cardiovascular disease fellowship program.

During training in interventional cardiology, the fellow must have performed at least 250 therapeutic interventional cardiac procedures, documented in a case list and attested to by the training program director. In addition, the training program director must judge the clinical skill, judgment, and technical expertise of the fellow as satisfactory.

To receive credit for performance of a therapeutic interventional cardiac procedure in the training pathway, a fellow must meet the following criteria:

- Participate in procedural planning, including indications for the procedure and the selection of appropriate procedure or instruments
- Perform critical technical manipulations of the case. (Regardless of how many manipulations are performed in any one "case," each case may count as only one procedure.)
- Be substantially involved in postprocedural management of the case
- Be supervised by the faculty member responsible for the procedure. (Only one fellow can receive credit for each case even if others were present.)

Program directors will be asked to attest to the performance of at least 250 therapeutic interventional cardiac procedures for each candidate who received training in their program.

Beginning with the November 2000 examination, candidates who have been out of formal training for 3 or more years as of June 30 of the year of examination must document post-training performance as primary operator of at least 150 therapeutic interventional cardiac procedures in the 2 years prior to application for certification.

PRACTICE PATHWAY

Candidates who have been admitted previously to the interventional cardiology examination through the practice pathway and have not yet achieved certification may be admitted to future interventional cardiology examinations beyond 2003, when the practice pathway is no longer available for first-time admission. These candidates must meet the Board's requirements for licensure and professional standing and provide documentation of performance as primary operator of 150 therapeutic interventional cardiac procedures in the 2 years prior to application for examination.

CLINICAL COMPETENCE REQUIREMENTS

The Board will require substantiation by local authorities or the program director that the candidate's clinical competence

as an interventional cardiology consultant is satisfactory and that the candidate is in good standing in the medical community.

CERTIFICATION EXAMINATION

The Certification Examination in Interventional Cardiology will be a comprehensive 1-day examination of multiple-choice questions in the single-best-answer format with an absolute standard for passing. The examination will assess the candidate's knowledge and clinical judgment in aspects of interventional cardiology required to perform at a high level of competence. These include:

Case Selection (25%)

- Indications for angioplasty and related catheter-based interventions in management of ischemic heart disease, including factors that differentiate patients who require interventional procedures rather than coronary artery bypass surgery or medical therapy
- Indications for urgent catheterization in management of acute myocardial infarction, including factors that differentiate patients who require angioplasty, intracoronary thrombolysis, or coronary artery bypass surgery
- Indications for mitral, aortic, and pulmonary valvuloplasty in management of valvular and congenital disorders, including factors that differentiate patients who require surgical commisurotomy or valve repair or replacement
- Indications for interventional approaches to management of hemodynamic compromise in patients who have acute coronary syndromes, including the use of pharmacologic agents, balloon counterpulsation, emergency pacing, and stent placement.

Procedural Techniques (25%)

- Planning and execution of interventional procedures, including knowledge of options, limitations, outcomes, and complications as well as alternatives to be used if an initial approach fails
- Selection and use of guiding catheters, guidewires, balloon catheters, and other FDA-approved interventional devices, including atherectomy devices and coronary stents
- Knowledge of intravascular catheter techniques and their risks

- Use of antithrombotic agents in interventional procedures
- Management of hemorrhagic complications.

Basic Science (15%)

- Vascular biology, including the processes of plaque formation, vascular injury, vasoreactivity, vascular healing, and restenosis
- Hematology, including the clotting cascade, platelet function, thrombolysis, and methods of altering clot formation
- Coronary anatomy and physiology, including angiographic data such as distribution of vascular segments, lesion characteristics, and their importance in interventions; alterations in coronary flow due to obstructions in vessels; the assessment and effect of flow dynamics on myocardial perfusion; the function of collateral circulation; and the effect of arterial spasm or microembolization on coronary flow.

Pharmacology (15%)

- Biologic effects and appropriate use of vasoactive drugs, antiplatelet agents, thrombolytics, anticoagulants, and antiarrhythmics
- Biologic effects and appropriate use of angiographic contrast agents.

Imaging (15%)

- Specific applications of imaging to interventional cardiology, including identification of anatomic features and visualization of lesion morphology by angiography and intravascular ultrasonography
- Radiation physics, radiation risks and injury, and radiation safety, including methods to control radiation exposure for patients, physicians, and technicians.

Miscellaneous (5%)

- Ethical issues and risks associated with diagnostic and therapeutic techniques
- Statistics, epidemiologic data, and economic issues related to interventional procedures.

Successful Diplomates will be awarded an ABIM Certificate of Added Qualifications in Interventional Cardiology. The certificate will bear dates limiting its validity to 10 years. Recertification will be required for renewal of the certificate.

RECERTIFICATION

To maintain certification in interventional cardiology and to qualify for recertification, Diplomates must maintain a valid ABIM certificate in cardiovascular disease.

COMPUTER-BASED TESTING

The ABIM is beginning to phase in computer-based testing. By 2005, all ABIM recertification examinations will be administered via computer only; all certification examinations will be administered via computer only by 2006.

EXAMINATION REGISTRATION

The Certification Examination in Interventional Cardiology is offered in November of each year. Registration for the examination extends from January 1 through April 1 of the year of examination. Late registration is available through June 1; however, a non-refundable penalty fee of $300 will be charged for applications postmarked between April 2 and June 1. Candidates may register through the "Online Services" feature of the ABIM web site, www.abim.org, during the registration period. To obtain a paper application, please contact the ABIM:

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1

BASIC CORONARY BALLOON ANGIOPLASTY AND STENTING

Morton J. Kern

INTRODUCTION

On September 16, 1977, Andreas Grüentzig performed the first human percutaneous transluminal coronary angioplasty (PTCA) in Zurich, Switzerland. Until then, coronary artery bypass surgery was the only alternative to medicine for the treatment of coronary artery disease. Over the last 26 years, new developments have resulted in a dramatic growth of percutaneous coronary intervention (PCI) as one of the most successful methods of coronary revascularization. In 2002 approximately 750,000 patients underwent PCI in the USA alone. PCI is the treatment of choice for discrete single- and double-vessel coronary lesions in patients with good left ventricular function and plays an important role in complex revascularization in patients with multivessel coronary artery disease and depressed left ventricular function. Today there are many techniques to open a narrowed artery, not only of the coronary arteries but also of the peripheral and great arteries of the body. The use of various techniques, which include balloons, stents, cutters, lasers, grinders, suckers, filters and other tools, are collectively called PCI. Percutaneous transluminal coronary angioplasty (PTCA) will be used to describe information and techniques related to use of the balloon inflation technique alone that was first employed by Grüentzig.

This chapter will present the basic method and mechanisms of balloon angioplasty and stenting as an introduction to the practice of interventional cardiology. The various techniques of

percutaneous coronary revascularization can be placed into niche applications for specific devices (Table 1-1).

OVERVIEW OF THE BASIC PCI METHODS

Percutaneous coronary intervention was derived from the basic procedures used for diagnostic cardiac coronary angiography. PCI begins with vascular access and uses the same techniques for the insertion of an arterial sheath through the arm (radial artery) or leg as Seldinger's method (needle and guidewire). Specialized larger-lumen "guiding" catheters engage the coronary artery in the same manner as those used for diagnostic coronary angiography with relatively minor differences.

Figure 1-1 shows how to perform PCI. A guiding catheter is first seated in the coronary ostium. A thin, steerable guidewire is introduced into the coronary artery and positioned across the stenosis into the distal aspect of the artery. An angioplasty catheter, which is considerably smaller than the guiding

Table 1-1

Niche Applications of PCI Devices

Lesion Type	Balloon/ Stent	Rotoblator	DCA	TEC	Special Device
Type A	+++	++	–	–	–
Complex	++	++	+	–	–
Ostial	++	++	–	–	–
Diffuse	+	+++	–	–	–
Total occlusion	++	+	–	–	–
Calcified bifurcation	±	++	+	–	–
SVG Focal	+++	±	+	±	–
SVG Diffuse	+	±	–	++	–
SVG Thrombotic	±	–	–	++	Angio Jet
Complication dissection	+++	–	±	–	–
Acute Occlusion	++	–	–	–	–
Thrombosis	+	–	–	+	Angio Jet
Perforation	±	–	–	–	Covered stent, perfusion balloon

+++ highly applicable; ++ somewhat helpful; + applicable; ± marginal; – not applicable.
DCA, directional coronary atherectomy; TEC, transluminal extraction catheter.

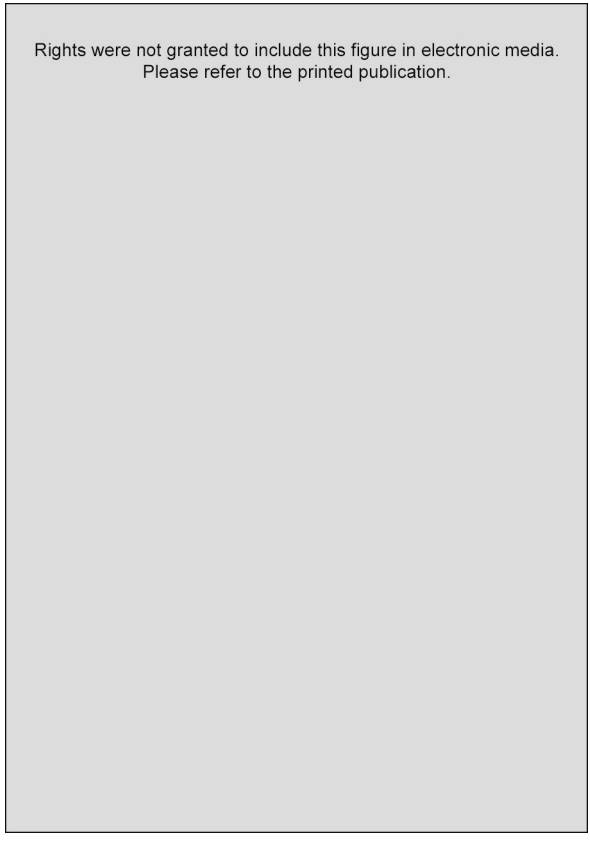


Fig. 1-1 How angioplasty and stenting works. **A,** The artery is filled with atherosclerotic material, compromising the lumen. A cross-section of the artery is shown on the right side. **B,** A guidewire is positioned past the stenoses through the lumen. **C,** A balloon catheter is advanced over the guidewire. **D,** The balloon is inflated. **E,** The balloon is deflated and withdrawn. **F,** The balloon catheter is exchanged for a stent (on a balloon). **G,** The stent is expanded. **H,** The expanded stent remains in place after the deflated balloon is withdrawn. (Reproduced with permission from 'Your PTCA, our Guide to Percutaneous Transluminal Coronary Angioplasty', American Heart Association, 2001.)

catheter, is inserted through the guiding catheter and is positioned (in the artery) across the stenotic area by tracking it over the guidewire. Once correctly placed within the area to be treated, the balloon on the PCI catheter is inflated several times for periods ranging from 10 seconds to several minutes. The

inflation and deflation of the balloon in the blocked artery restores blood flow to an area of the heart previously deprived by the stenosed artery. The stent on a balloon catheter is also deployed in the same manner. The definitions of a successful PCI procedure are summarized in Box 1-1.

Figure 1-2 shows the components of the PCI system. There are three major difficulties with PCI: (1) stable guide catheter positioning; (2) negotiating tortuous vessel segments with the guidewire; (3) delivering the stent through tortuous segments. To complete the PCI, the operator must control the three principal movable components (guide catheter, balloon catheter, and guidewire).

After the balloon catheter is positioned, and compresses the stenotic material, a stent will then be delivered. After the PTCA, the balloon is exchanged for a catheter carrying a stent. The stent is a metal scaffold, compressed on another balloon catheter and delivered exactly as the first balloon catheter was delivered. The stent should be precisely positioned and is inflated with the same pressure gauge syringe (to high pressure) for 10–20 seconds. A full opening of the stent is important to a good result. After the stent is expanded into the artery wall, the balloon is deflated and the delivery catheter and guidewire are removed. After final angiography is performed, the guide catheter is removed. The arterial sheath is secured, to be removed later, or removed and the puncture site sealed in the laboratory. The patient is then transferred to his room. If no complications occur, the patient is discharged the next morning. The patient commonly returns to work shortly (<3 days) thereafter.

MECHANISMS OF ANGIOPLASTY

Several mechanisms of angioplasty have been proposed.

- **Disruption of plaque and the arterial wall.** The inflated balloon exerts pressure against the plaque and the arterial wall, causing fracturing and splitting. Concentric (round or circumferential) lesions fracture and split at the thinnest and weakest points. Eccentric lesions split at the junction of the plaque and the normal arterial wall. Dissection or separation of the plaque from the vessel wall releases the restraining effect caused by the lesion and results in a larger lumen. This is the major mechanism of balloon angioplasty.
- **Loss of elastic recoil.** Balloon dilatation causes stretching

Box 1-1

Definitions of PCI Success

PCI success may be defined by angiographic, procedural, and clinical criteria.

Angiographic Success

A successful PCI substantially enlarges the vessel lumen at the target site. Prior to stents, success was the achievement of a minimum stenosis diameter reduction to <50% with grade 3 TIMI flow (assessed by angiography). With coronary stents, success is a minimum stenosis diameter reduction to <20%.

Procedural Success

A successful PCI should achieve angiographic success without in-hospital major clinical complications (e.g., death, myocardial infarction [MI], emergency coronary artery bypass surgery) during hospitalization. MI is often defined as the development of Q-waves in addition to a threshold value of creatine kinase (CK) elevation has been commonly used. The significance of enzyme elevations in the absence of Q waves is controversial. Several reports have identified non-Q-wave MIs with CK-MB without Q-waves as an associated complication of PCI.

If serial determinations are performed, an abnormally high value (CK-MB >1 times normal) can be expected in 10–15% of PTCA procedures, 15–20% of stent procedures, 25–35% of atherectomy procedures, and more than 25% for any device used in saphenous vein grafts or long lesions with a high atherosclerotic burden, even in the absence of other signs and symptoms of MI. There is no accepted consensus on what level of CK-MB index (with or without clinical or ECG findings) is indicative of a clinically important MI following the interventional procedure.

Cardiac troponin T and I as measurements of myocardial necrosis are more sensitive and specific than CK-MB. However, prognostic criteria based on troponin T and I have not yet been developed. In patients in whom a clinically driven CK-MB determination is made, a CK-MB of more than three times the upper limit of normal constitutes a clinically significant MI.

Clinical Success

A clinically successful PCI is anatomic and procedural success with relief of signs and/or symptoms of myocardial ischemia after recovery from the procedure. The long-term clinical success requires that the patient has persistent relief of signs and symptoms of myocardial ischemia for more than 6 months. Re-stenosis is the principal cause of lack of long-term clinical success when short-term clinical success has been achieved.

Modified from Smith SC Jr, Dove JT, Jacobs AK, *et al.* ACC/AHA guidelines for percutaneous coronary intervention (revision of the 1993 PTCA guidelines)—executive summary: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee to revise the 1993 guidelines for percutaneous transluminal coronary angioplasty) endorsed by the Society for Cardiac Angiography and Interventions. *Circulation* 2001;103:3019–3041.

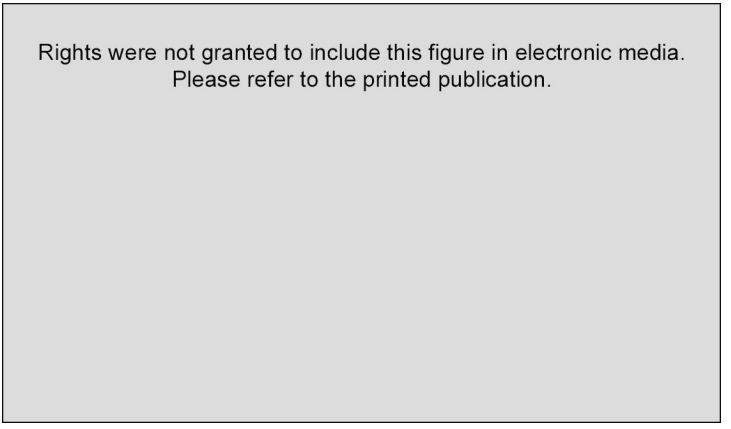


Fig. 1-2 Diagram of components of percutaneous coronary intervention equipment. (From Safian R, Freed M, eds. *The Manual of Interventional Cardiology*, 3rd edition. Birmingham, Michigan: Physicians' Press, 2001.)

and thinning of the medial wall. Stretching causes the vessel wall to lose its elastic (recoil) properties. The degree of elastic recoil is affected by the balloon/artery size ratio. Almost all vessels that have undergone PTCA have some elastic recoil. The major mechanism of stenting is elimination of elastic recoil, maintaining a large lumen over time.

- **Redistribution and compression of plaque components.** During angioplasty, balloon pressure causes denudation or stripping of the vessel wall lining (endothelial) cells and the extrusion or pushing out of plaque components. There may be some molding of the softer lipid material but this effect accounts for a very small part of the overall effect of angioplasty.

INDICATIONS FOR PCI*

Specific anatomical and clinical features for each patient should be considered for the likelihood of success, failure and risk of complications with vessel closure, vascular morbidity, mortality, and restenosis. Restenosis and incomplete revascularization must also be weighed against the outcome anticipated for CABG. The complete recommendations from the AHA/ACC/SCAI PCI guidelines are provided in Appendix I.

*See Guidelines AHA/ACC Task Force Report, *Circulation* (2000).

In General, PCI

- Angina pectoris unrelieved by optimal medical therapy
- Mild angina pectoris with objective evidence of ischemia (abnormal stress test or abnormal stress thallium) and a coronary lesion in a vessel supplying a large area of myocardium
- Unstable angina
- Acute myocardial infarction in patients as a primary approach or who have contraindications to thrombolytic therapy or who have evidence of persistent or recurrent ischemia despite thrombolytic therapy; direct PTCA without thrombolytic therapy is commonly used as the primary therapy in many circumstances
- Angina pectoris after coronary artery bypass graft surgery
- Symptomatic restenosis after prior PCI

Relative Contraindications to PCI

- Unsuitable coronary anatomy (e.g., left main, severe diffuse distal disease)
- High-risk coronary anatomy in which closure of vessel would result in death
- Contraindications to coronary bypass graft surgery (however, some patients will have PCI as their only alternative for revascularization)
- Bleeding diathesis (low platelet count, peptic ulcer disease, coagulopathy, and so on)
- Patient noncompliance with procedure and post-PCI instructions
- Multiple PCI restenoses

Complications of PCI (see also Chapter 4)

For most elective procedures:

- Death (0.1%)
- Myocardial infarction (1–3%)
- Emergency coronary artery bypass grafting (0.5–2%)
- All complications that can occur during diagnostic cardiac catheterizations can also occur during PCI:
  - —Access-site bleeding, especially with larger sheaths and prolonged anticoagulation (1:250 patients)
  - —Contrast-medium reactions
  - —Cerebral vascular accident, myocardial infarction, and so on

- Vascular injury (e.g., pseudoaneurysm of femoral artery)
- Restenosis (see Chapter 7).

Note: Restenosis at the site of PCI occurs in approximately 10–30% of patients and may lead to recurrence of anginal symptoms. Typically, restenosis occurs most frequently within the initial 6 months after PCI. This biologic effect is *not* considered a complication but rather a clinical part of angioplasty.

PCI EQUIPMENT

The most commonly used PCI equipment consists of four basic elements: a guiding catheter, a coronary guidewire, a balloon catheter, and a stent (Fig. 1-2, Table 1-2).

The Guiding Catheter

A special large lumen catheter is used to guide the coronary balloon catheter and other interventional devices to the vessel with the lesion to be dilated.

Functions of the Guiding Catheter

A guiding catheter serves three major functions during angioplasty: (1) balloon catheter delivery and guidance; (2) backup

Table 1-2

Approximate Costs of Coronary Angioplasty Equipment

Equipment	Cost ($)
Balloon dilatation catheter	500
Guiding catheter	100
Guidewire	100
Exchange guidewire (300 cm)	100
Indeflator	50
Y connector	15
Sheath introducer	10
Torque tool	10
Nonballoon devices	
Stent (noncoated)	1200
Stent (drug-eluting)	2800
Directional atherectomy catheter	1200
Rotoblator	1200

support for balloon advancement; and (3) pressure monitoring. Construction of a guiding catheter is shown in Figure 1-3.

Balloon Catheter Delivery and Guidance. To deliver the balloon catheter to the coronary ostium, the guiding catheter should be seated with the tip parallel to the artery (coaxial). Coaxial alignment permits safer transmission of force needed to advance the balloon across a stenosis. This act may require guide catheter repositioning or deep seating into the artery.

Adequate contrast injection through the guide catheter is critical to position the balloon and depends on the size of the guide catheter lumen with the angioplasty device in place. A guiding catheter must be large enough to permit adequate contrast administration with the PCI catheter in place to opacify the target vessel and visualize the lesion. Large, nonballoon PCI devices (rotoblator, directional coronary atherectomy, Angio-Jet Aspiration catheter, or some stents) in small guide catheters may not allow adequate vessel visualization during angiography. This problem has been overcome with larger lumen, small guide catheters, and power injectors in some labs.

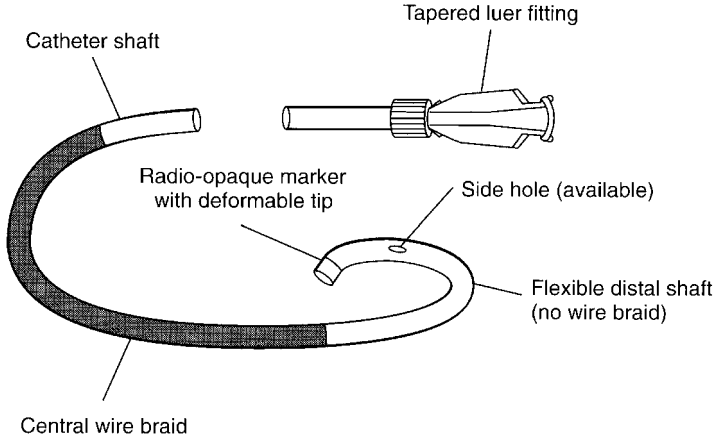


Fig. 1-3 Construction of a guiding catheter. The features noted differentiate it from diagnostic catheters. (From Avedissian MG, *et al.* Percutaneous transluminal coronary angioplasty: a review of current balloon dilation systems. *Cathet Cardiovasc Diagn* 1989;18:263.)

Operators should select a guide catheter with a lumen diameter large enough to allow adequate contrast flow around the PCI device to obtain a clear angiographic image of the lesion. As balloon and PCI catheters have become smaller, the size of internal diameter of the guiding catheter has become less important for achieving adequate visualization. A large guide catheter lumen, however, is critical to facilitate easy passage of atherectomy devices, and double balloon/stent systems for complex or bifurcation lesions.

Backup Support for Balloon Catheter and Stent Advancement. Support or "backup" for stent advancement is achieved after seating (cannulation) the guide catheter in the coronary ostium. The guiding catheter provides a platform from which one can push the stent over the guidewire through the artery and across the stenosis.

Inadequate backup support will result in failure to cross a lesion and an unsuccessful procedure. Backup support requires a combination of correct coaxial (in-line with the artery ostium) alignment, as well as the ability to provide carefully controlled advancement (deep seating) of the guiding catheter into the coronary ostium.

The improved quality and size of currently used stents have reduced the need for robust backup support in most situations. For more complex and technically difficult lesions, the choice of an appropriate guiding catheter for adequate support and lesion visualization remains essential (see Chapter 3). Although commercially formed catheters are generally adequate, a guiding catheter will rarely need to be reshaped in the catheterization laboratory using a heat gun for successful coronary cannulation and backup support.

When there is insufficient backup in crossing a very tight stenosis, the guiding catheter may be disengaged from the coronary ostium and backed out into the aortic root. When pressure is applied to the stent catheter during attempt to cross the lesion, repositioning the guide catheter in a stepwise fashion as the stent is advanced may overcome this loss of support. However, aggressive intubation of the coronary ostium may damage the vessel, stopping the procedure prematurely, and may require additional stenting for an ostial dissection.

Deep seating of the guide catheter is achieved by manipulating the guide catheter over the balloon catheter shaft, past the aortocoronary ostium and farther into the vessel, to obtain increased backup support for crossing difficult lesions. This maneuver typically is used as a last resort because of the increased chance of guide catheter-induced dissection of the left main or proximal vessel.

Pressure Monitoring. The guiding catheter measures aortic pressure during the case. Pressure wave damping may occur during coronary artery engagement if there is plaque in the coronary ostium. In addition, pressure measured proximal to the stenotic area can be compared to distal transstenotic pressure measured with a pressure sensor guidewire for assessment of lesion severity before and after PCI. Some catheters have side holes near the tip to permit perfusion into the artery when the catheter is deeply seated and obstructing flow.

Guide Catheter Construction

Catheter Characteristics. Compared to the diagnostic catheters, the guiding catheters have thinner walls, larger lumens, and stiffer shafts. A large catheter lumen is achieved at the expense of catheter wall thickness and thus may result in decreased catheter wall strength, less torque control, or catheter kinking. The guiding catheters are generally stiffer to provide backup support during the PCI catheter advancement into the coronary artery and, therefore, respond differently to manipulation than diagnostic catheters. The guiding catheter tip is not tapered. Pressure-wave damping upon engaging the coronary ostium is seen more often than with similar-size diagnostic angiographic catheters. Some guide catheters have relatively shorter and more flexible tips to decrease catheter-induced trauma.

Side Holes. Guiding catheters with small side holes permit blood to enter the coronary artery when the ostium is blocked by the guide catheter. Side holes are used when the guide catheter either partially or totally occludes blood flow into the coronary artery. The guide catheter coronary occlusion is noted by the change in the arterial pressure waveform to one of "damping." Catheter side holes eliminate or reduce ischemia

when the guiding catheter is seated in a small artery. However, side holes may lead to inadequate artery visualization from loss of contrast media exiting the catheter before entering the artery. Although side holes may provide reliable aortic pressure, coronary flow can still be compromised during the angioplasty procedure. The guide catheter and side holes act as a "second stenosis" at the coronary ostium.

Small-Shaft-Diameter Catheters. The most frequently used size of guiding catheter is currently 6 French. The use of smaller-diameter guide catheters, conceptually, will result in fewer vascular complications and allow earlier ambulation of patients. However, this advantage is offset by the compromised quality of coronary angiograms with smaller catheter lumen sizes. Small-size (≤5F) guide catheters do not allow for the use of some stents. 7 or 8F guide catheters are used for complex procedures involving larger PCI devices or bifurcation lesions.

Balloon Dilatation Catheter Systems

Types of Balloon Catheter. There are three types of PCI balloon catheter (Fig. 1-4): over-the-wire, monorail, and fixed-wire balloon catheters. The over-the-wire and monorail balloons, but not fixed-wire balloons, are also used to deliver stents that are mounted by the manufacturer on a specific balloon. The advantages and limitations are summarized in Table 1-3.

Over-the-Wire Angioplasty Balloon Catheters. A standard over-the-wire angioplasty balloon catheter has a central lumen throughout the length of the catheter for the guidewire and another separate lumen for balloon inflation. These balloons are approximately 145–155 cm long and may be designed to be used with guidewires of various dimensions (1.010–0.018 inches). In these systems, the guidewire and the balloon catheter move independently. The major advantage is the ability to maintain distal artery access with the guidewire beyond the lesion while exchanging one over-the-wire balloon catheter for another. To exchange balloons, the balloon is tracked over the wire to a distal position. The wire may then be removed from the balloon. The wire may then be reshaped and reintroduced through the central lumen or exchanged for a longer guidewire (300 cm) to maintain distal position while

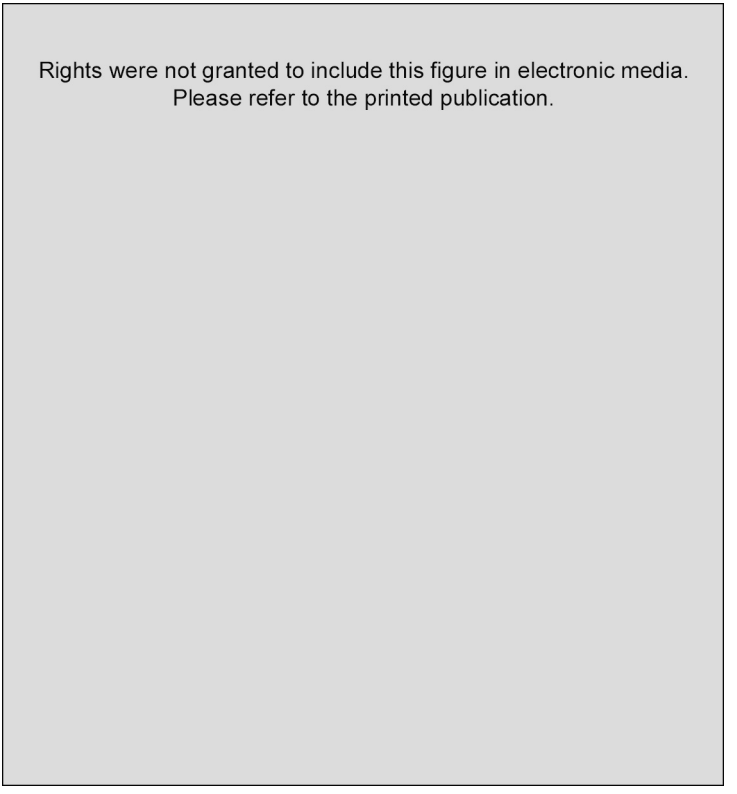


Fig. 1-4 Three common types of coronary balloon angioplasty catheter design. (Modified from Freed M, Grines C, eds. *Manual of interventional cardiology*. Birmingham, MI: Physicians' Press, 1992: 29.)

the balloon catheter is completely withdrawn over the guidewire and another balloon catheter is introduced over the same long guidewire for additional dilatations. An alternative method of balloon catheter exchange is to "trap" or secure a regular guidewire (145 cm) in the guide while the balloon catheter is exchanged (see exchange methods later). Over-the-wire catheters can accept multiple guidewires, which allows for exchanging additional devices that may require stronger, stiffer guidewires.

Over-the-wire angioplasty balloon catheters have several limitations. These catheters are, in general, slightly larger than

Table 1-3

Advantages and Limitations of Angioplasty Balloon Types

Advantages	Limitations
Over the wire	
Distal wire position	Two experienced personnel required
Distal port available for pressure measurement or contrast media injection	± Larger profile
Accepts multiple guidewires	
Rapid exchange	
Distal wire position	Exchanging balloons at hemostatic valve may be technically demanding
Enhanced visualization	
Low-profile balloons	
Single-operator system	
Fixed wire	
Enhanced visualization	Lack of through lumen
Single-operator system	Inability to recross lesion without removing system
Use with small guiding catheters	
Low-profile balloons	

Modified from Kern MJ, ed. *The cardiac catheterization handbook,* 2nd ed. St Louis, MO: Mosby, 1995.

the rapid-exchange (monorail) and fixed-wire catheters. Additional personnel may be required to help with long guidewire exchanges.

Rapid-Exchange (Monorail) PCI Balloon Catheters. "Rapid-exchange" monorail catheters were developed to improve the exchanging of angioplasty balloon catheters by single operators. Rapid-exchange catheters have only a short (~30–40 cm) length of the catheter shaft containing two lumens. One lumen runs the entire length of the catheter and is used for balloon inflation. The other lumen, which extends through only a portion of the catheter shaft, houses the guidewire. Because only a limited portion of the balloon requires dual lumens, rapid-exchange catheters are smaller in diameter than over-the-wire balloon catheters. Figure 1-5

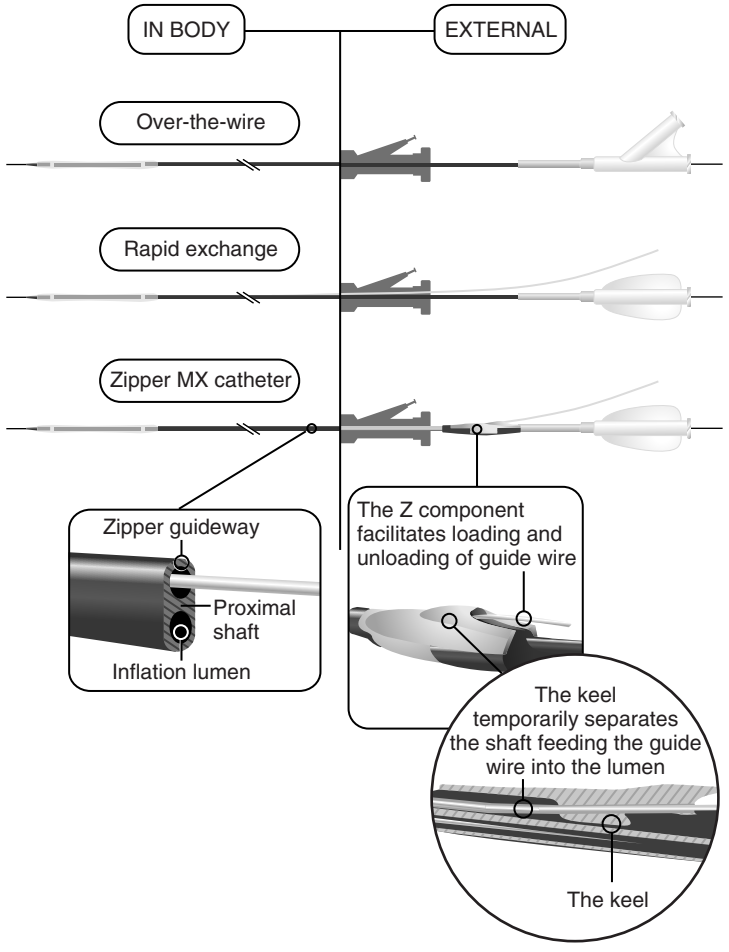


Fig. 1-5 Zipper balloon (Medtronic, Minneapolis, MN) allows variable length monorail to be converted to over-the-wire method at any time.

shows a novel type of "convertible" monorail using a zipper-type technique. Figures 1-6 and 1-7 show OTW and monorail balloon catheters.

Rapid-exchange balloon catheters address certain inherent limitations of over-the-wire catheters. First, over-the-wire balloon exchanges require a long (or extension) guidewire, an unnecessary maneuver for the rapid-exchange type. Second, a single operator can use rapid-exchange balloon catheters

Fig. 1-6 Quantum Maverick OTW. Over-the-wire "Quantum" Maverick Balloon. (Courtesy of SciMed-Boston Scientific, Boston, MA.)

without the aid of other assistants to maintain distal guidewire position.

Limitations of monorail catheters include the need for more expertise in manipulation of the guidewire, balloon catheter, and guiding catheter. Excessive blood loss at the rotating hemostatic valve during removal of the balloon catheter (back-out) maneuver may occur but valved "Y" connectors have reduced this problem. More caution when moving the balloon is needed. If the monorail balloon is advanced beyond the wire, the wire may come out of its short lumen, necessitating reassembly of the balloon and guidewire, especially when monorail catheters with relatively short "rails" are used.

If the balloon catheter requires force to advance beyond a lesion, a loop of guidewire may sometimes form outside the guide catheter in the aorta. This loop is nearly invisible but should be considered if the operator advances the catheter without seeing motion at the balloon tip.

Fixed-Wire Angioplasty Balloon Catheters. The fixed-wire catheter has the balloon mounted on a central hollow wire with a distal flexible steering tip. The proximal end of the catheter consists of a single port connected to a thin metal tube (hypotube). A core wire extends from the hypotube to the end of the distal steerable tip. This assembly is coated with a thin plastic shaft that enhances flexibility. Fixed-wire balloons have only one enclosed lumen for balloon inflation.

The on-the-wire balloon catheter is a fixed wire system, where the guidewire cannot be advanced independently of the balloon and the balloon cannot be exchanged without

Fig. 1-7 Maverick 2 Monorail. Monorail balloon catheter. (Courtesy of SciMed-Boston Scientific, Boston, MA.)

removing the entire system. Since the wire is attached to the distal end of the balloon, there is no central balloon lumen, resulting in a lower total profile than an over-the-wire or monorail system. Its principal advantages relate to its low profile, enabling passage through very tight stenoses, and good contrast visualization of the lesion being dilated around the balloon catheter.

The small shaft size provides excellent coronary visualization. Because the balloon is mounted on the distal guidewire, the device was designed to be used by a single operator. Fixed-wire balloon catheters are particularly useful for distal lesions, subtotal stenoses, and lesions located in tortuous vasculature.

The limitations of fixed-wire catheters include lack of the inherent safety advantage of over-the-wire and rapid-exchange systems because there is no movable wire available to exchange for a stent if a dissection occurs. To exchange this catheter the catheter must be removed completely. The lesion is then recrossed with a new guidewire and balloon catheter. A dissected lesion may not permit recrossing with a guidewire or advancement of another balloon catheter. Further attempts at recrossing may even close the vessel. In such cases, an angioplasty guidewire can be introduced alongside the fixed-wire balloon catheter before removing the catheter to assist in placing another balloon.

Characteristics of Balloon Catheters. The plastic material of the balloon determines its compliance and tensile strength. The main differentiation among balloons is related to compliant or noncompliant materials. Inflation of a compliant balloon above nominal pressure (i.e., a set pressure for a known balloon size) will lead to further expansion of the balloon approximately 10–20% over the rated diameter. Noncompliant balloons, on the other hand, remain very close to their rated diameter even when inflated several atmospheres above nominal pressure. The advantages and disadvantages of balloon materials remain controversial. A compliant balloon may be more cost-effective and result in fewer balloons being used in a given case. However, a compliant balloon may result in oversizing, particularly on second and third inflations, resulting in dissections. The complication rate of such mechanisms has not been tested in randomized clinical trials.

After most stents are deployed, post-deployment, high-pressure inflations are performed with low-compliance or noncompliant balloons. Most balloons are coated with low-friction surface polymers to facilitate lesion crossing.

The mechanical aspects of balloon inflations apply the following fundamental principles.

- Overinflation at balloon ends. According to Laplace's law, wall stress increases with radius. At a given pressure, a larger balloon undergoes more wall stress than a smaller balloon, promoting balloon rupture. The arterial segment next to the lesion has a larger luminal radius than the lesion. Thus, artery sites adjacent to the lesion may be traumatized.
- At high pressures, balloons weaken over time. The balloon burst pressure may be decreased during subsequent inflations of the same balloon. When inflating a balloon above the rated burst pressure, consider limiting the number and duration of inflations.
- Balloon diameters always increase with increasing pressure. Noncompliant balloons will grow in diameter by less than 10% over nominal pressures. Compliant balloons may increase by more than 20%. The balloon diameter-pressure relation is usually linear, reflecting the compliance characteristics. Figure 1-8 shows the balloon during inflation and a graph for pressure versus diameter.
- Balloons do not return to their original dimensions after deflation. At any given pressure, the balloon diameter during a subsequent inflation will be larger than during the first inflation. When dilating two lesions with a compliant balloon, consider approaching the narrower lesion first.

Selection of Balloons. The selection of a balloon catheter is highly subjective and less critical in the era of stents. The balloon size is selected to achieve a 1:1 size match with the vessel. Balloon-to-artery ratios of more than 1.2:1 are associated with increased complications. Longer balloons (30–40 mm) are useful for dilating long and diffuse narrowings. Short (10–15 mm) balloons are used for stent re-expansion to avoid stretching the uninvolved vessel wall.

The balloon size is determined using the distal arterial reference segment diameter as gauged by the size of the guiding catheter (e.g., 7F guide = 2.31 mm, 8F = 2.64 mm,

Fig. 1-8 **A**, Balloon size changes proposed to occur during increasing inflation pressure when using compliant balloon material. (From *Clinical issues in angioplasty balloon material: a review of the literature regarding polyethylene terephthalate (PET)*, USCI Division of C.R. Bard, Inc.) **B**, Diameter–pressure relationships of three balloon materials. All three balloons have a nominal size of 3.0 mm at 6 atm. (From Raymenants E, Bhandari S, Desmet W, *et al.* The impact of balloon material and lesion characteristics on the incidence of angiographic and clinical complications of coronary angioplasty. *Cathet Cardiovasc Diagn* 1994;32:303–309.)

9F = 2.97 mm, 11F = 3.63 mm). Visual estimation of artery diameter is less accurate than quantitative angiographic approaches but it is the method used by most interventionalists. From intravascular ultrasound (IVUS) studies, most stents selected by visual sizing are 0.5 mm smaller than true vessel dimensions.

Balloon Inflation Strategies. Stenosis resolution occurs when the balloon pressure eliminates the balloon indentation caused by the stenosis called the "waist." Unstable or thrombotic lesions are generally soft and are associated with a lower balloon inflation pressure than chronic, stable lesions. Most coronary lesions respond to inflation pressures of more than 10 atm. Calcific or fibrotic lesions may require higher inflation pressure (12–17 atm) to eliminate the balloon waist. Because stenting after PTCA is now routine, issues regarding optimal balloon inflation strategies are relatively unimportant. Balloon inflations are generally brief (<60 sec). No consensus on the optimal duration of inflation exists. The balloon should be inflated long enough to permit elastic tissue to relax or stretch.

However, in lesions not receiving a stent, high pressures in compliant balloons may produce an oversized balloon-to-artery ratio, associated with an increased incidence of dissection and complications. Low-pressure inflations may reduce complications. Most procedures start with low pressures but operators often feel compelled to use higher pressures to achieve satisfactory angiographic results.

Additional Issues in Choosing Catheters. Important technical considerations for selecting PCI systems include catheter profile, trackability, pushability, and ease of exchange.

- **Device profile.** The size of a deflated catheter has been emphasized in catheter selection. Stent profiles are now very small (profile size of 0.035 and 0.033 inch). There appears to be no practical difference among equipment sizes. Stent profile alone is not the only factor in facilitating a stent to cross a lesion.
- **Trackability** is the ability to advance the stent through the vessel to reach the lesion and is a function of friction related to both the guidewire and the delivery catheter. A stiffer guidewire will allow easier trackability. Although stent systems are marketed based on their ability to track and conform to the vessel, trackability is difficult to measure in an objective manner. For a balloon to track it must be able to transfer force through the shaft of the balloon catheter ("pushability"). There may be little difference in the pushability of the majority of the available systems. Resistance to balloon catheter forward motion may occur as a result of

guidewire-balloon friction, balloon-guide catheter friction, or balloon-artery friction.

- **Ease of device exchange.** The monorail catheter system is the quickest and easiest to exchange. The standard over-the-wire balloon requires the placement of a long guidewire, or the attachment of an exchange system to the end of the guidewire, or a guidewire trapping system. A rapid-exchange system reduces x-ray exposure time, because fluoroscopy is not required if the wire is fixed during catheter removal.

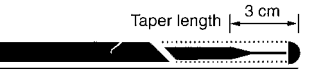
PCI Guidewires

PCI guidewires are small-caliber (0.010–0.018 inch) steerable wires, advanced into the coronary artery or its branches beyond the lesion to be dilated. A J-tip of varying degree, usually shaped by the operator, allows steering across side branches through tortuous artery curves (Fig. 1-9).

Guidewires are made with an inner core wire and an outer spring tip. The shorter the distance between the end of the central core and the spring tip the stiffer and more maneuverable the wire. Differences in core construction affect guidewire handling. Important considerations when selecting a guidewire include diameter, coating, torque control, flexibility, malleability, radio-opacity, and trackability. The diameter for the most commonly used coronary guidewires is 0.014 inch, although diameters from 0.010 to 0.018 inch are available. Larger-diameter guidewires have better torque and backup support, while small-diameter wires are more maneuverable. Custom tip shaping will help steer the guidewire. Several helpful shapes are shown in Table 1-4.

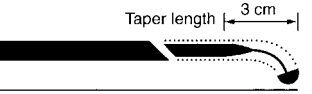
Guidewire Characteristics. The selection and placement of a guidewire distal to the stenosis depend on the clinical situation and the operator's experience and skills. The following terms are applied to angioplasty guidewires.

- **Stiffness** of the guidewire determines specific performance. Soft wires are safer and easier to advance through tortuous artery branches. Stiff wires torque better and are often useful for crossing difficult or total chronic occlusions. Extra stiff guidewires provide better support for intracoronary stent placement in highly tortuous arteries.
- **Steerability** is defined as the ability to turn and advance the wire through tortuous segments and side branches by



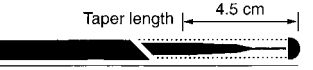
Standard tip flexibility

Catalog number	Diameter, inches	Length, cm	Taper length, cm	Tip shape
502-596	0.014	175	3.0	Straight
502-595	0.014	300	3.0	Straight



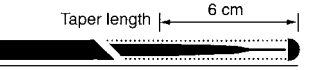
Standard tip flexibility

Catalog number	Diameter, inches	Length, cm	Taper length, cm	Tip shape
502-597	0.014	175	3.0	J-Curve



High tip flexibility

Catalog number	Diameter, inches	Length, cm	Taper length, cm	Tip shape
502-598	0.014	175	4.5	Straight



Very high tip flexibility

Catalog number	Diameter, inches	Length, cm	Taper length, cm	Tip shape
502-599	0.014	175	6.0	Straight

Fig. 1-9 Angioplasty guidewires.

rotation of the wire. Steerability is an important feature of a guidewire.

- **Flexibility** is determined by the distance from the end of the central core to the distal spring tip of the wire and is important in avoiding vascular trauma when crossing and re-crossing lesions.
- **Malleability** is the ability to shape the spring tip and maintain a desired tip shape. Repeated attempts with different wire tip configurations may be required to cross distal stenoses. The manufacturer preforms some guidewire tip shapes. Guidewire tip shaping is accomplished by bending the wire between the thumb and index finger, rolling the guidewire tip over a needle, or bending the wire tip at the end of an introducer tool. In general, the length of the distal bend in a large vessel should approximate half the usual diameter of the vessel (about 2 mm). A larger bend may be needed to reach a takeoff. When steering the wire into an

abruptly angled branch, a double 45° bend is often helpful (Table 1-4).

Radio-opacity, Marker Bands and Special Coatings. Visualization of the guidewire is provided by a radio-opaque coating usually applied only to the distal part of the wire. The limited radio-opaque segment permits lesion visualization without obscuring useful angiographic detail, such as small dissections. Calibrated radio-opaque marker bands are used to gauge lesion length. Angioplasty balloons usually have two markers, one at each end of the balloon. Small balloons, e.g., 1.5 mm diameter, have one central marker. These markers may be confused for markers on some guidewires.

A variety of different wire coatings increase ease of wire movement within the balloon catheter and artery. Some coated plastic tipped wires, especially with hydrophilic tips, have a higher likelihood to perforate.

Exchange and Extension Guidewires. An exchange guidewire is similar to those mentioned above, except that its length is 280–300 cm. This long wire replaces the initial 140 cm wire when an exchange of the balloon catheter is necessary (e.g., upsizing balloon or insertion of stent). Alternatively, a 120–145 cm extension wire can be connected to a companion 145 cm guidewire, thus creating a long exchange guidewire to allow balloon catheter exchanges.

Types of Guidewire Exchange Systems. Three different systems are available to lock a short guidewire in place to permit over-the-wire PCI catheter exchanges (Table 1-5):

- The trapper exchange system uses a specially constructed balloon-on-a-wire that is not long enough to leave the end of the guide catheter. When it is inflated within the guide catheter, it traps the guidewire against the inside wall of the guide catheter, permitting the PCI catheter to be pulled off without moving the guidewire. A different PCI catheter can then be advanced over the wire and the trapper balloon can be deflated and removed. The PCI catheter is then re-advanced into the artery. If the trapper is removed too quickly, air embolization can occur.
- A ferromagnetic guidewire can be secured in place with a strong magnet (SciMed). The magnet is attached to the

Table 1-4

Guidewire Tip Curves that can Facilitate Difficult Anatomic Problems During PTCA

Rights were not granted to include this table in electronic media.
Please refer to the printed publication.

From Voda J. Angled tip of the steerable guidewire and its usefulness in percutaneous transluminal PTCA. *Cathet Cardiovasc Diagn* 1987;13:204–210.

Table 1-5

Exchange Devices

Company	Name	Description
Guidant	Anchor	Device for exchanging balloons and other devices over any non-hydrophilic guidewire ≥0.018 inch, by wrapping an Elastinite core wire around the guidewire and fixing it in the distal guidewire catheter
SciMed	Trapper	A small inflatable balloon that traps the guidewire inside the guiding catheter, allows rapid exchange with over-the-wire balloons
SciMed	Magnet	Clip on magnet to secure exchange of Choice (and other SciMed) guidewires

Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*, Birmingham, MI: Physicians' Press, 1999.

Y connector. The guidewire lies within the cylindrical magnet while the PCI catheter is withdrawn and another is inserted over the wire still being held by the magnet.

- Lastly, the wire can be "anchored" by another locking system (Anchor, Guidant) in which a thin hypotube with a coiled internal "anchor" wire is advanced inside the guide. On release out of its housing tube, the anchor wire coils around the PCI guidewire, locking it in place. The PCI catheter can then be exchanged as described above.

Accessory Equipment

Adjustable Hemostasis and Rotating Y-connector Valve. The Y connector is attached to the guide catheter to permit introduction of a PCI catheter into the guide while allowing contrast injection through the guide catheter. The end of the Y connector has a rotating connection and a valve. The valve minimizes back bleeding from the guide catheter while the PCI catheter is inserted. The Y connector also permits pressure monitoring through the guiding catheter, regardless of PCI catheter position.

Balloon Inflation Devices. A disposable syringe device is used to inflate the balloon on the PCI catheter. A pressure gauge or display indicates the precise inflation pressure in atmospheres (atm or torr) or pounds/square inch (psi). Typically, the

balloon is inflated with sufficient pressure (4–12 atm) to fully expand the stenosis indentation ("dumbbell" or "waist") of the partially inflated balloon. Occasionally, some lesions involved with calcified or high fibrotic lesions may require very high inflation pressures (>14 atm) to expand the balloon and eliminate the "dumbbell." Overinflation of the balloon increases the risk of artery dissection.

Guidewire Torque (Tool) Device. A small, cylindrical pin vise clamp slides over the end of a guidewire, permitting the operator to perform fine steering manipulations of the guidewire.

Guidewire introducer
A very thin, needle-like tube with a tapered conical opening on one end helps the operator to insert the guidewire through Y connector valves or into balloon catheters.

Arterial Sheaths. In the past long (23 cm) arterial sheaths were thought necessary to protect the femoral artery and improve guide catheter handling. Currently, long sheaths are rarely needed. However, long-length femoral arterial sheaths allow one to repeatedly negotiate a tortuous or diseased iliac artery more easily than with the standard 15 cm sheath used for diagnostic catheterization. A long sheath may also improve the torque control for some guiding catheter maneuvers but does add friction if kinked.

STENTS

The implantation of coronary stents has superseded traditional balloon angioplasty and is one of the most basic parts of PCI. Stents reduce coronary restenosis, initially shown from the results of the Belgium–Netherlands stent (BENESTENT) and the stent restenosis study (STRESS) trials. Premounted stents on balloon catheters have a capacity to expand a lesion depending not only on the diameter of the stent but also on the amount of friction (flaring of the distal struts as it contacts the lesion), flexibility of the stent and delivery balloon, and resistance of the artery to expansion. The multitude of stent designs has arisen because of the need to overcome patent designs, physiologic mechanisms, and materials applications and most importantly to increase the ability of the operator to

deliver the stent. The ability of a stent to cross a lesion depends on several factors, such as the profile of the balloon/delivery system, the friction between the system and the artery, the friction of stent struts and the lesion, and the flexibility of the system. Stent delivery depends on both flexibility and profile to achieve the goal of reliable delivery without sacrificing radial strength and length of lesion scaffolding. Characteristics of an ideal stent are listed in Box 1-2.

Types of Stent

Mechanism of Stent Expansion. Stents are classified based on their mechanism of implantation and are either self-expanding or balloon-expandable. Nearly all commonly used coronary stents are balloon-expandable. Some stents for saphenous vein graft stenting are self-expanding.

Composition. Stents vary in their composition and may be made of stainless steel, cobalt-based alloy, tantalum, or nitinol, and further may or may not have inert or active coatings or biodegradable coatings and/or drug delivery systems.

Stent Dimensions and Designs. For native coronary arteries, expanded stent diameters range from 2.5 to 5 mm. Stent lengths vary from 8 to 33 mm. For saphenous vein grafts, larger diameters (>5 mm) of stents may be used. Few stents are

Box 1-2

Ideal Stent Characteristics

- Biocompatible
- Conformity to tortuosity
- Flexibility
- High radial strength
- Low metallic surface area
- Low profile
- Radio-opaque
- Secure delivery system
- Side branch access
- Thromboresistant
- Trackability

specifically designed for particular lesions. A unique covered stent with a polytetrafluoroethylene (PTFE)-covered stent, by JOMED, is specifically designed for applications of coronary ruptures, aneurysms, and degenerated saphenous vein grafts. Stents are designed using a mesh structure, coil, slotted tube ring, multicellular design, or unique custom design.

Three stents in common use, from which next-generation stents have been developed, are the Bx velocity stent (Cordis), the multilink Penta (Guidant), and the NIR stent (Boston Scientific). All three stents superseded the initial Palmaz–Schatz design and have themselves been superseded by more recently released designs. Current stents achieve near perfect stent attachment to the delivery balloon, eliminating the problem of stent migration and loss. There is a minimal delivery balloon extension from the stent edge, limiting adjacent vessel trauma during high-pressure stent balloon inflation. There is relatively low compliance of the delivery balloons, assisting in a homogeneous stent implantation. All three stents are applicable for general use in nearly all circumstances. Each stent has specific characteristics that permit the operator to differentiate among them for their selection. Figure 1-10 shows cell designs of different available stents.

- The Bx velocity stent has three different cell configurations—six cells for a vessel diameter of 3 mm, seven cells for vessels up to 4 mm, and nine cells for vessels of 5 mm.
- The multilink tetra, penta, and zeta stents are similar to the Bx velocity with a catheter 143 cm long, compared to 138 cm or 135 cm for other systems. The penta stent has a modified link pattern, which improves flexibility and scaffolding in vessels up to 4 mm in diameter.
- The NIR stent also provides excellent lesion coverage and, because of cell geometry, is thought to reduce plaque prolapse in vessels 4 mm or more in diameter. The NIR stent has a seven or nine cell structure and is useful in saphenous vein grafts. A balloon covering the end of the stent by a "sox" is a feature unique to this stent delivery system.
- Compared to open cell designs, the BeStent (Medtronic) stent has an extra strut between interlocking junctures, providing greater coverage and greater support. The S7 stent (Medtronic) has ring segments providing increased flexibility, conformability, and low friction for this stent.

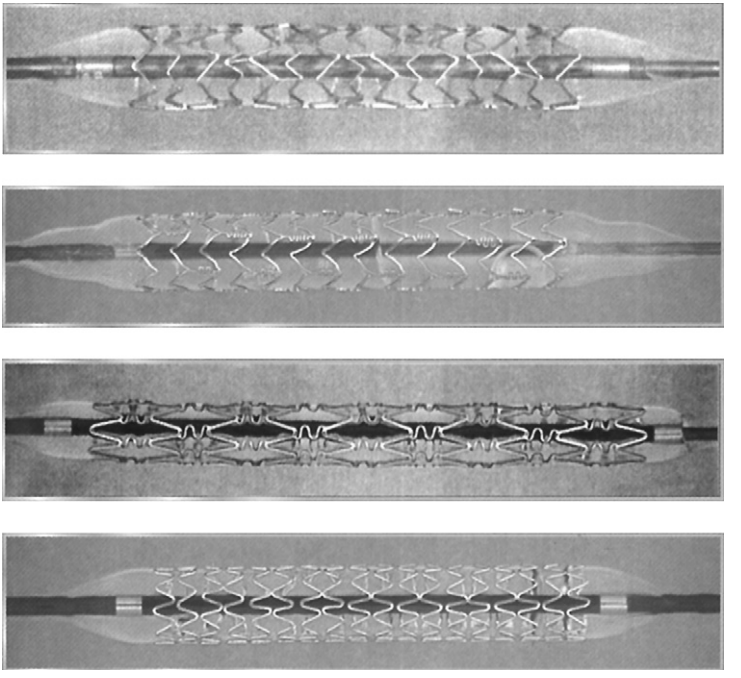


Fig. 1-10 **A,** Cell characteristics of different commercially available stents.
Continued

Table 1-6 lists and compares features of several commercially available stents.

Indications and Contraindications for Stenting

In addition to indications for PCI in general, extensive evidence from observational and randomized trials to support the use of coronary stenting for five major indications:

- Treatment of abrupt or threatened vessel closure during angioplasty
- Primary reduction of restenosis and de novo focal lesions in vessels greater than 2.5 mm in diameter
- Focal lesions in saphenous vein grafts
- Total coronary occlusions
- Urgent treatment of acute coronary occlusion for myocardial infarction.

Figure 1-11 is an example of stent placement in right coronary angioplasty (RCA).

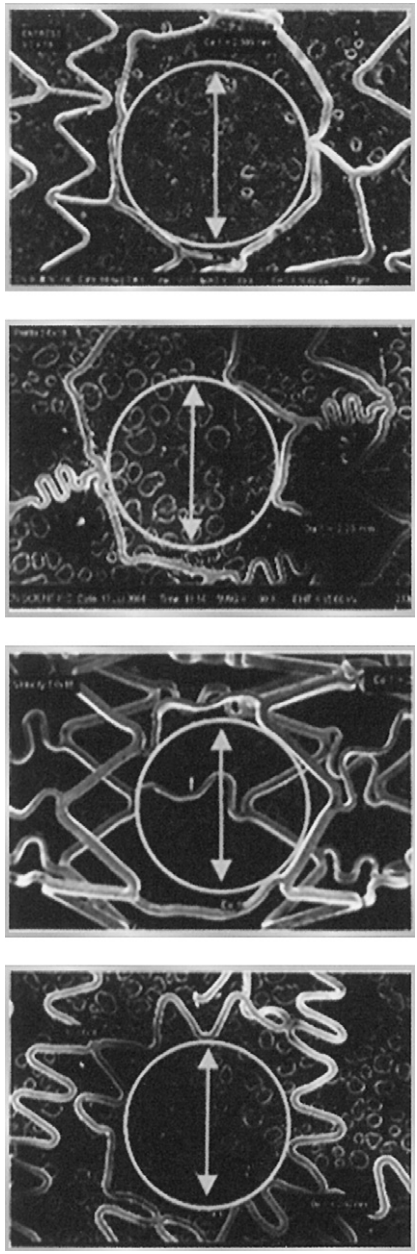


Fig. 1-10, cont'd **B,** Cell characteristics of different commercially available stents.

Continued

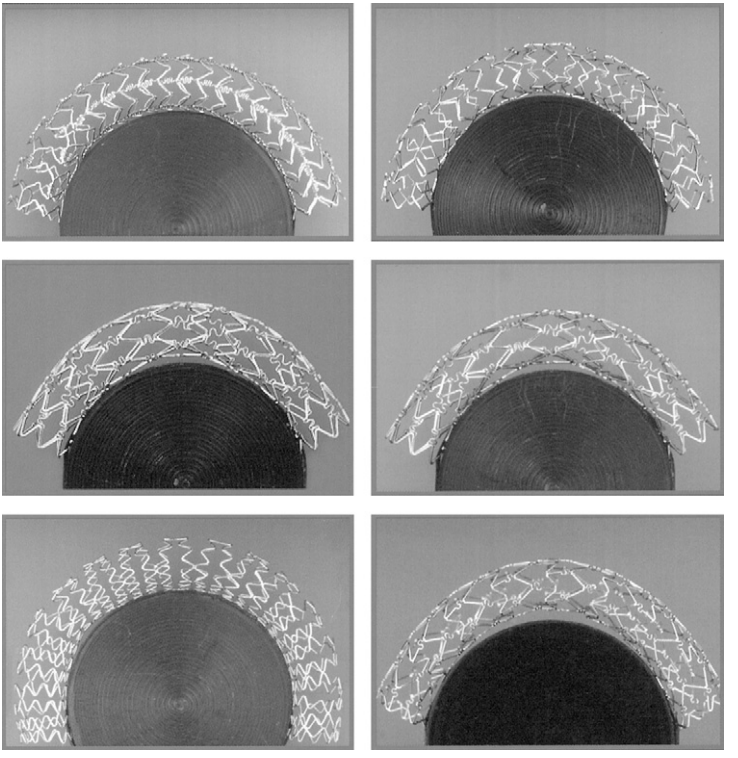


Fig. 1-10, cont'd C, Cell characteristics of different commercially available stents.

Contraindications can be divided based on patient and anatomic factors.

Patient Factors. When anticoagulation is not used, patient contraindications are similar to those for coronary angioplasty. When anticoagulation is used, contraindications include:

- Gastrointestinal bleeding that prevents 4–5 hours of anticoagulation during or following the stent procedure
- Inability to take antiplatelet therapy
- Conditions prone to hemorrhage; intracranial hemorrhage, recent surgery, or bleeding diathesis.

Anatomic Factors.

- Small vessels, less than 2.5 mm
- Vessels with poor distal runoff

Table 1-6

Stent Consumer's Guide

Product	Manufacturer	Deliverability	Scaffolding	Side-Branch Access	Accurate Positioning	Large Vessels	Small Vessels	MRI Safe
AVE S670	Medtronic	++++	+ (19%)	+++	++	++	+	+
AVE S7	Medtronic	++++	+++ (14%)	++	++	+++	+	+
Biodivysio	Biocompatibles	++	++++	+	+	++	++	+
Bx Velocity/Sonic	Cordis, Johnson & Johnson	+++	+++ (>20%)	++	++	+++	+	+
JoStent graft	Jomed	+	++++	NA	++++	+++	0	+
Multilink Penta	Guidant	+++	+++ (19%)	++	++	+++	+	+
Express	Boston Scientific	+++	+++	++	++	+++	+	+
Biodivysio SV	Biocompatibles	+++	+++	+	+	NA	++	+
Multilink Pixel	Guidant ACS	+++	+++	++	+	NA	++	+

++++ excellent; +++ very good; ++ good; + acceptable; 0 unsuitable; NA not applicable.
From Colombo A, Stankovic G, Moses JW. Selection of coronary stents. *J Am Coll Cardiol* 2002;40:1021–1033.

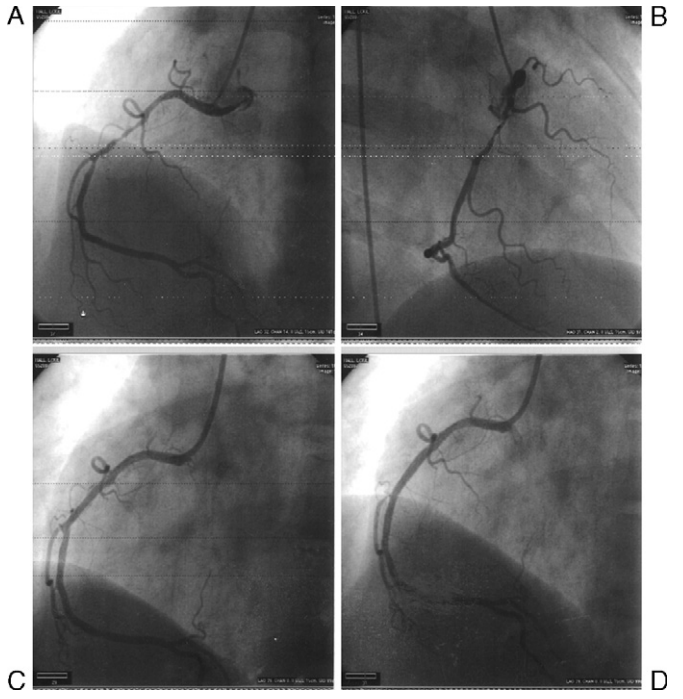


Fig. 1-11 Stent placement in right coronary angioplasty. **A,** Cineangiographic frames of right coronary artery before PCI with a stenosis in the proximal segment (left anterior oblique view). **B,** Right coronary artery (RCA) in right anterior oblique view. **C,** RCA after balloon angioplasty. **D,** RCA after stent placement. Note compression of plaque into ostium of RV marginal branch.

- Vessels supplying poorly functional or nonfunctional myocardium
- Heavily calcified vessels.

Complex Stenting

(See below for specific lesion techniques.)

Stenting for patients with any of the following characteristics should be carefully considered and will be discussed in detail in Chapters 8 and 9:

- Long lesions requiring more than one stent per lesion
- Small coronary artery reference vessel diameters (<3 mm)
- Significant thrombus at the lesion site

- Lesions in saphenous vein grafts, the left main coronary artery, ostial locations, or bifurcation lesions
- Restenotic lesions
- Diffuse disease or poor outflow distal to the identified lesion
- Very tortuous vessels in the region of the obstruction or proximal to the lesion
- Significant impairment of left ventricular function—patients with impaired left ventricular function are at increased risk of complications associated with interventional techniques.

Box 1-3 lists proven and unproven indications for stenting.

Stent Implantation Technique

Delivery of stent to the lesion is performed after balloon angioplasty and has also been performed directly without preceding balloon dilation (called "direct" stenting). Stent implantation technique differs from balloon angioplasty technique in two major respects: (1) Choosing the correct stent size and length is critical, since a stent becomes a permanent implant; (2) Potential problems of stent delivery and implantation before the procedure such as vessel calcification, tortuosity and length of lesion, generally not problems for

Box 1-3

Proven and Unproven Indications for Stenting

Proven indications for stenting

- Native coronaries ≥3.0 mm in diameter
- Stenotic vein grafts
- Restenotic lesions after nonstent interventions
- Chronic total occlusions
- Acute myocardial infarction

Currently unproven situations for stenting

- Small vessels
- Long lesions and diffuse disease
- Bifurcation lesions
- Ostial lesions
- Unprotected left main stenting
- Multivessel stenting vs. coronary artery bypass grafting.

From Ashby, DT, Dangas G, Mehran R, Leon MB. Coronary artery stenting. *Catheter Cardiovasc Intervent* 2002;56:83–102.

balloon catheters, are problems for stents and must be considered beforehand. Stent implantation can be performed equally well from the femoral or radial approach with 6 French sheaths and guide catheters. 7 or 8 French systems should be used if double balloons or stents are anticipated for bifurcation lesions or for rotoblator.

Guiding Catheter for Stents. As for balloon catheter delivery, coaxial guiding catheter support remains essential for effective stent delivery. Correct guide catheter selection is especially important when stent implantation is performed in an angulated circumflex or shepherd's crook right coronary artery, tortuous vessels, distal lesions, or vessels with long dissections. In these anatomic conditions, Amplatz, Voda, Q or similar wide-curve configurations will provide good support for stent delivery. Stent delivery into some saphenous vein graft conduits, especially to the circumflex or left anterior descending artery, may require a multipurpose guiding catheter support.

Although large-lumen (>6F) guides provide better contrast delivery and visualization of the target site, power injection of contrast facilitates visualization and reduces the procedure time and contrast load using guide catheters of less than 6 French.

Guidewires for Stenting. Routine stent implantation procedures can be easily performed with regular support guidewire. Extra-support guidewires (0.014 inch) provide a good "rail" when stent implantation is undertaken in lesions with extreme angulation or tortuosity and for lesions with long dissections. The extra support guidewire assists both guiding catheter stability and stent delivery. Although helpful in stent delivery, extra-support guidewires can sometimes damage the distal vessel, cause "pseudo" lesions by folding endothelial tissue in tortuous artery bends, or precipitate vessel spasm. A strategy of exchanging back to a floppy-tipped wire after stent delivery may prevent these effects.

The Predilation Stenting Method. Prior to stent implantation, balloon predilation is commonly performed. Predilation with a balloon that is slightly undersized relative to the reference vessel diameter is a safe strategy that gives the operator useful information such as the degree of difficulty involved in

negotiating the vessel curves and the pressure needed to expand the lesion. Using a slightly undersized balloon leaves an indication of the lesion so the stent can be optimally positioned. Utilizing standard techniques for balloon angioplasty, the stenotic lesion is crossed with a 0.014 inch guidewire. Once the lesion is traversed, a standard balloon angioplasty procedure is performed. The lesion may be intentionally underdilated before stenting. The balloon angioplasty catheter is withdrawn, leaving the guidewire positioned across the lesion.

Predilation also allows for the vessel to be fully re-pressurized with restored flow, which often produces vasodilation. It is not uncommon to find a vessel enlarged after balloon dilation. This enlargement results in the operator selecting a larger stent than would have been chosen initially.

The Direct Stenting Method. Direct stenting without balloon predilation is commonly performed with excellent results in most circumstances. Caution should be used when stents cannot be delivered to the lesion site because of tortuosity or calcifications. Exchange for a balloon catheter, predilation, and/or exchange for a stiff guidewire may be needed. It is disconcerting to the operator to place a stent directly in a lesion only to find that the stent cannot be fully expanded because of heavy calcification. Factors favoring direct stenting are summarized in Box 1-4.

Stent Expansion. Verify the stent position relative to the lesion before implanting the stent. After positioning the stent, recheck the position relative to the side branches and landmarks of the target lesion. Remember that the stent is a

Box 1-4

Factors Favoring Successful Direct Stenting

- Age <70
- No calcium at the target or other coronary vessels
- No severe proximal tortuosity
- Not a left circumflex artery lesion
- Proximal location

Modified from Nguyen T, *et al. J Interventional Cardiol* 2002;15:237–241.

permanent implant and time should be taken to place it correctly, thus avoiding additional and unnecessary stents. It is also important that the stent covers the entire length of the dissection or lesion without leaving any inflow and outflow obstruction.

Stent expansion should be performed under fluoroscopy to judge whether it is fully expanded and to ensure that its diameter matches the proximal and distal reference coronary artery diameter(s). Optimal implantation requires that the stent struts be in full contact with the arterial wall. If the stent is not symmetrically expanded along the long axis of the vessel, a larger balloon (up to 4 mm) or high inflation pressures (>14 atm) may be used. Ideally, the final stent diameter should match that of the referenced vessel. All efforts should be taken to ensure that the stent is not underdilated. Intravascular ultrasound (IVUS) is the only method of guaranteeing this.

Technical Notes for Stent Implantation.

- When stenting multiple lesions, the distal lesion should be treated initially, followed by the proximal lesion. Stenting in this order obviates the need to recross the proximal stent with the distal stent and reduces the chances of stent delivery failure.
- When recrossing a recently implanted stent, ensure that the guidewire traverses the stent and does not go between the stent and the vessel wall, which may result in inadvertent dislodgement of the stent during further balloon/stent passage.
- If there is stent inflow or outflow obstruction or residual distal vessel narrowing, a freshly prepared balloon catheter can be advanced into and through the stented area for further dilatations. Re-wrapping previously used balloons should be considered.
- Eliminate any inflow or outflow narrowing by additional balloon inflations or stent implantation (especially if the stent margin has a dissection).
- An acceptable angiographic result is a residual narrowing of less than 10% by visual estimate, but a truly optimal result must be confirmed by IVUS.
- Vasospasm may occur during the procedure when inflation pressures of more than 15 atm were used for stent optimization. This phenomenon is self-limiting, always resolves with time or after intracoronary nitroglycerin, and

has not been associated with any unfavorable clinical events. Extraordinarily high-pressure inflations (>16 atm) are generally unnecessary and have been associated in some reports with stent overexpansion and higher in stent restenosis rates.

Optimizing Stent Implantation. The concept of optimization is to expand the stent to the maximal extent that it is safe to dilate without vessel injury. Optimal stent expansion is determined by the ratio of the stent lumen cross-sectional area (CSA) relative to the vessel CSA at the stent site and also relative to the reference lumen CSA. The essential features of the stent optimization technique are:

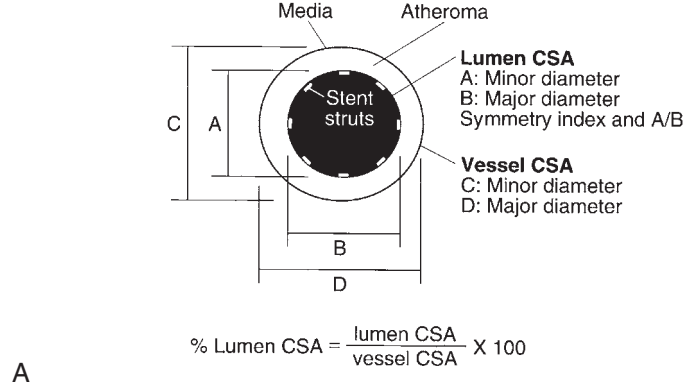
- Evaluation of the dimensions of reference vessel and implanted stent by IVUS
- Selection of an appropriately-sized, noncompliant balloon based on IVUS target vessel diameter at the stent site
- Perform high-pressure balloon dilatation of the stent (usually above 12 atm) or dilation with a larger balloon.

IVUS Optimization Based on the Reference Lumen. Successful stent expansion is achieved when (1) there is no significant difference between the lumen diameters of the stent and the reference site (particularly the distal reference) and (2) there is complete apposition of the stent to the vessel wall. For small vessels, the IVUS criterion of achieving a final stent lumen CSA larger than the distal reference lumen CSA are strongly recommended. In larger (>2.5 mm) vessels, a final stent lumen CSA greater than the distal reference CSA is accepted with optimal stent apposition. This is accepted because the reference sites in large vessels commonly have less disease in the reference segments than do the small vessels. This also makes the achievement of a final stent lumen larger than the distal CSA more difficult to achieve in large vessels than in small vessels. Typically, a final stent lumen CSA of 80% of the distal reference vessel is accepted.

IVUS Optimization Based on the Reference Vessel Area. Using criteria based only on IVUS vessel area has the inherent flaw of not incorporating stent expansion relative to the reference lumen CSA. The use of a criterion of 50% of the average vessel area would leave a significant number of patients with a stent

that was underexpanded compared to the distal reference lumen. The use of a criterion of 60% of the average would position the final stent lumen between the CSAs of the proximal and distal reference lumen (Fig. 1-12). The use of reference vessel criteria has the disadvantage of requiring multiple additional measurements, in contrast to using the reference lumen criterion, which requires only a few.

IVUS Optimization Based on Final Balloon Size. A simplified guideline for assessing final stent lumen uses the balloon



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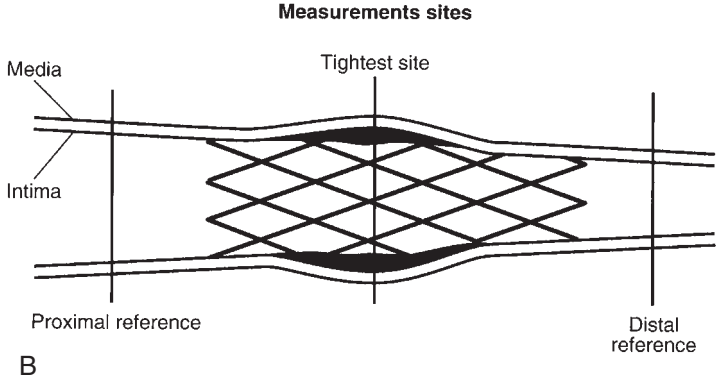
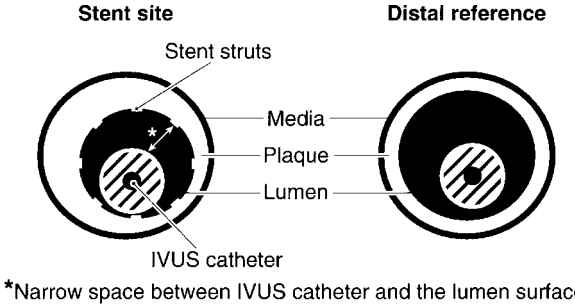


B

Fig. 1-12 Intravascular ultrasound imaging (IVUS) measurements after stent placement. **A,** Vessel structure in cross-sectional areas (CSA) A, B, C, D diameters by IVUS. **B,** Measurements sites along the course of the vessel. The most severe narrowings (tightest site) in the proximal and stent segments are compared to the distal reference site.

Continued



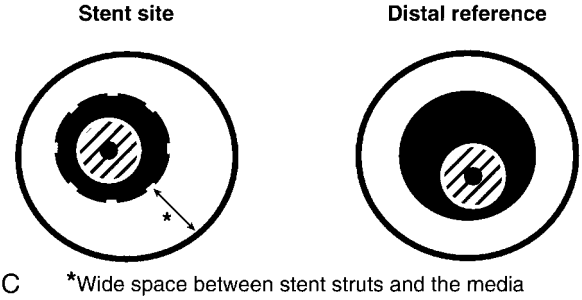


Fig. 1-12, cont'd C, Intravascular ultrasound imaging comparison of area of deployed stent relative to distal reference area and amount of plaque in each segment.

chosen for final stent optimization. The interventionist usually selects an appropriate-sized balloon based on visual estimation of the diameter of the reference vessel. The minimum cross-sectional area of the stent lumen should be greater than 70% of the calculated cross-sectional area of the balloon selected, based on the angiogram. This simplified criterion provides a safety buffer in small vessels, where the risk of stent thrombosis is higher, and is less strict for larger vessels, where the risk of stent thrombosis is reduced.

Stent Expansion Strategies. There are two methods of optimizing stent expansion and improving the cross-sectional area of the stent lumen: (1) high pressure and (2) a large-diameter balloon. When an oversized balloon is used, there is

an increased likelihood of coronary vessel rupture or dissection. Using high pressure with a balloon that is appropriately sized to the vessel allows stent expansion to occur within the natural confines of the vessel. To avoid complications, the balloon: angiographic reference vessel ratio should be approximately 1.0. If a balloon:vessel ratio is more than 1.0, a short, noncompliant balloon with medium pressure (12–16 atm) is preferable. When a balloon larger than the angiographic vessel diameter is used for final stent optimization, it should never be larger than the distal IVUS minimum vessel diameter (measured media to media). When there is a large differential between the size of the proximal and distal vessels, as may occur in the left anterior descending artery before and after the second diagonal, careful balloon selection is important. Generally, using slightly lower pressure in the distal part of the stent segment and a higher pressure for the proximal portion of the stent is all that is necessary. Care should be taken not to dilate beyond the distal edge of the stent with an oversized balloon. Occasionally, if there is significant vessel tapering, dilation with two balloons of different diameters should be considered.

Noncompliant balloons are preferable to compliant balloons for final dilations for several reasons. Noncompliant balloons will expand and dilate uniformly, even in focal areas of resistant lesions, and are more likely to maintain a uniform diameter even at high pressures. Thus noncompliant balloons allow for optimal stent expansion without overexpansion of the balloon in adjacent unstented segments, which contributes to dissection. Additionally, experience with IVUS has shown that 25% of stents have improved stent expansion with an increase in pressure from 15 to 18 atm or more.

Asymmetric Expansion. Stent expansion should be symmetric in soft plaque, especially soft plaque with lipid pool. Very hard plaque (fibrotic or calcified), seen in approximately 20–30% of lesions, is not easily compressed by the balloon/stent, resulting in asymmetric stent expansion into the normal arc of the vessel. In lesions with a significant arc (≥270°) of dense or hard fibrocalcific disease, asymmetric stent expansion occurs with a minimum to maximum lumen diameter ratio (symmetry index) of less than 0.7. In such lesions, further inflation leads

to focal overstretching in the less diseased arc of the vessel. The symmetry index can worsen after further dilation, especially if an oversized balloon is used (Fig. 1-13). Using a balloon that is 0.25–0.5 mm smaller than the size of the vessel, and very high pressures, may improve the symmetry index but will not necessarily increase the CSA of the lumen at the stent site.

Asymmetric overexpansion is associated with a risk of vessel rupture. The risk is highest if a larger balloon is used. If the stent lumen CSA is acceptable relative to the distal lumen CSA and the stent is well apposed, avoid efforts to make stent symmetry perfect.

Incomplete Stent Expansion. Adequate stent expansion is dependent on the plaque burden. Optimal stent expansion in lesions with 50–70% diameter stenosis or lesions with a spiral dissection can be easily accomplished because there is not much atheroma. In lesions with more than 90% diameter stenosis optimal stenting is more difficult to achieve and is associated with a higher percentage of asymmetric stent expansion. Incomplete stent expansion (i.e., when the stent struts do not contact the intimal surface) can occur, particularly in ectatic vessels (at poststenotic dilation or aneurysm sites) and in the ostial left anterior descending artery (LAD), where the operator is cautious about performing a high-pressure balloon inflation in the left main trunk (Fig. 1-14). In the latter case,

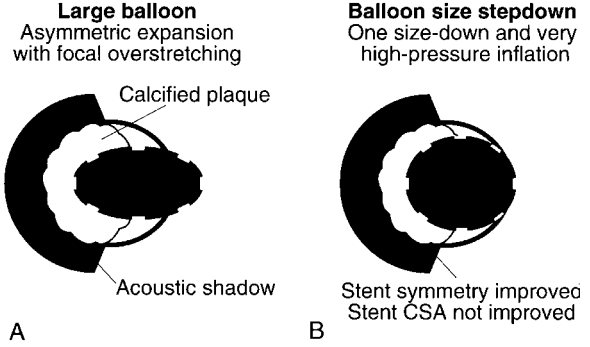


Fig. 1-13 Balloon inflation strategy based on intravascular ultrasound imaging after stent placement. **A,** Asymmetric stent expansion may require larger balloon. **B,** Stent symmetry is improved but cross-sectional area is not increased; use smaller balloon at very high inflation pressure.

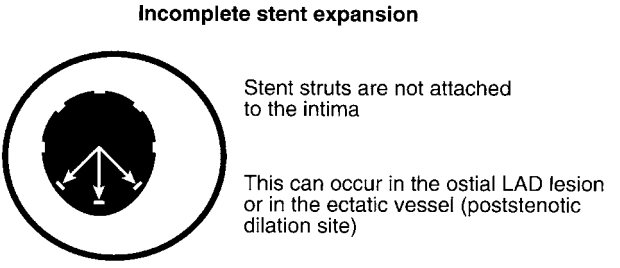


Fig. 1-14 Intravascular ultrasound image of incomplete stent expansion.

dilation of the ostial lesion with only the shoulder of the balloon does not provide sufficient expansion force to implant the stent fully. Box 1-5 suggests two ways to redeploy stent after initial failure to expand.

Dissection at the Stent Margin. Stent dilations sometimes cause a plaque fracture or dissection at the edge of the stent and vessel, which requires additional stents to stabilize the newly produced dissection (Fig. 1-15). Plaque fracture may result from misplacement of the balloon post dilation, especially if the balloon is clearly oversized relative to the angiographic vessel size. Plaque fracture can also occur even when the balloon is positioned within the stented segment, especially in calcific lesions or vessels. In more elastic or soft lesions, this is less likely to occur but it can be seen at the stent margins when the stents are deployed on bend lesions.

Plaque Prolapse. Plaque prolapse through stent struts may occur in 5% of coil-type stent implantation. Although the coiled stents have advantages in flexibility, the stent structure provides less complete radial support to the vessel wall. Further dilation does not improve the stent lumen CSA. An additional stent within the primary stent is necessary.

Box 1-5

Redeployment of a Stent after First Failure of Stent Expansion

- Increase the balloon inflation pressure to maximum
- Change current balloon to a high pressure and noncompliant balloon, inflate with maximal high pressure possible

Modified from Nguyen T, *et al. J Interventional Cardiol* 2002;15:237–241.

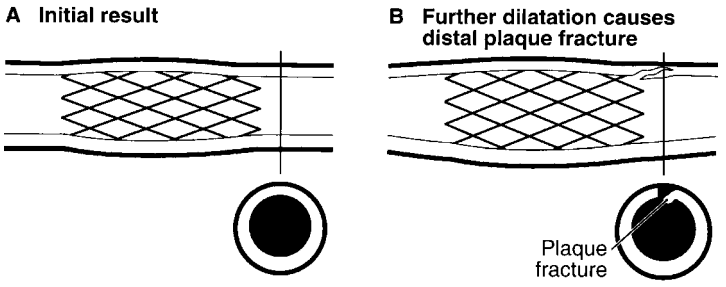


Fig. 1-15 Overexpansion of stent may cause distal dissection.

MANAGING COMPLICATIONS DURING STENT DELIVERY AND IMPLANTATION

Stents are used to treat challenging anatomic and clinical subsets. The complex nature of the procedure predisposes to unique complications and technical challenges. Complications of stenting implantation can be broken into six major categories.

Delivery Failure

Failure to deliver the stent is most often due to:

- Suboptimal guide catheter support
- Failure to predilate a significant coronary lesion
- Unsuspected proximal tortuosity or calcification of the vessel with unanticipated vessel rigidity and acute angulation.

A significant obstruction proximal to the target lesion may likewise prevent delivery of the stent to the offending narrowing. Failure of adequate guidewire support and stent–vessel mismatch also contribute to failure to deliver the stent to the target vessel.

For these reasons, predilatation has advantages for stent delivery in most circumstances. A pre-deployment balloon that tracks easily to the lesion dilates the lesion simply, provides evidence of good guide catheter support and bodes well for the delivery of the stent to the lesion. Difficulties with advancing the balloon, guide catheter instability, and difficulty in dilating through tortuous segments, on the other hand, herald the onset of stent delivery problems.

In arteries that are highly tortuous and have multiple bends and folds, guidewire selection is an important factor in stent

delivery success. Extra support guidewires may not be ideal for initially crossing lesions, producing folds and pseudostenosis, and conventional guidewires that are softer may permit delivery of the stent system without encountering the pseudostenosis (Box 1-6).

Box 1-6

Technical Manipulations when a Stent Fails to Advance

General

Best technical manipulation—secure a more stable guide position or, if possible, the guide can be deep-seated safely. A potential late complication is ostial stenosis due to endothelial trauma

Constant forward pressure on the stent catheter while pulling the wire back to decrease friction inside the stent catheter lumen and to straighten the stent catheter

Additional proximal segment dilation or plaque removal to facilitate stent advancement

Wire Manipulations

Advance a second stiffer wire to straighten the artery (the buddy wire technique). This stiff wire can cause wire bias

Advance the stent on the second stiffer buddy wire. Occasionally stents may actually advance more easily over a softer wire

Shape the wire along the curve of the artery to lessen wire bias so there is less friction or resistance at the outer curve of the vessel and the path of the wire is more coaxial with the path of the vessel

Stent Manipulations

If the problem is due to tortuosity of the proximal segment, change the stent to a shorter one

Select a different type of stent with better flexibility

Bend the stent to conform it along the curve of the artery

Guide Manipulations

Change to a guide with a different curve, to achieve better backup, and more coaxial to allow less friction at the ostium

Larger or smaller guide to achieve better backup

Modified from Nguyen T, *et al. J Interventional Cardiol* 2002;15:237–241.

Expansion Failure or "Persistent" Stent Narrowing

Inability to fully expand the stent after implantation may be due to:

- Tissue prolapse through cell sites
- Calcification or rigid vessels
- Dissection at stent margins
- Unsuspected thrombus formation within or adjacent to the stent, which may appear as narrowings related to stent implantation.

Failure to remove the balloon from the stent will provide an artifactual appearance of "material" within the stent. During the balloon inflation phase of stent implantation, full expansion of the balloon should always be observed. If an indentation persists, higher balloon inflation pressures or a larger, short balloon should be used. Failure of full stent expansion is usually the result of an inadequate predilatation approach. In cases where stent deployment appears suboptimal, intravascular ultrasound will confirm the mechanism of persistent narrowing due to tissue prolapse, incomplete apposition, heavy calcification, or, in some cases, thrombus.

Loss of Access to the Stent

Loss of guidewire access to a stent may result in a complication, especially if the stent has been inadequately expanded or when new lesions have been produced distal or proximal to the implanted stent. Re-crossing a recently deployed stent is facilitated by using a soft guidewire with an exaggerated tip loop to prolapse through the stent. Care should always be taken so that the wire does not enter under a stent strut between the strut and the arterial wall. Once the guidewire has crossed the stent, a second problem may be encountered of inability to advance a balloon for high-pressure post-stent deployment.

Re-crossing stents with balloons may be difficult when the proximal border of the stent is on a tortuous vessel segment, forcing the tip of the dilatation balloon into the vessel wall where it is blocked by the stent struts. Several approaches can be used to overcome this problem. The guide catheter

can be repositioned in a more coaxial manner. A stiffer guidewire can be advanced to reshape the curve of the artery. The balloon can be withdrawn slightly, rotated and re-advanced during inspiration or coughing (the balloon's profile should be as low as possible). Several operators have recommended putting a curve onto a stiff part of the guidewire and using it to advance across a tortuous segment proximal to a stent and placing a curve on the balloon by forming it with the finger and using a technique similar to that of putting a gentle curve on a guidewire. Box 1-7 summarizes several technical manipulations that may be employed to re-cross a deployed stent.

Table 1-5 lists unique guidewire tip shapes that will help in difficult PCI situations.

Box 1-7

Techniques Facilitating Recrossing of a Stented Area by a Balloon or Another Stent

General

Best technical manipulation—steer the wire into a different direction, or to a different branch to lessen wire bias and increase more wire centering

Rotate the balloon catheter while advancing it and let the catheter enter the stent by itself through its rotational energy (like torquing the Judkins Right catheter).

Guidewire Manipulations

Bend the wire and place the bent segment near the ostium of the stent to be crossed to position the wire more at the center of the entrance of the stented segment and to decrease wire bias

Insert a second stiffer wire to straighten the vessel

Change the current wire to a stiffer one

Balloon/Stent Manipulations

Use a shorter balloon or stent

Use a more flexible balloon or stent

Use a fixed-wire balloon to cross the stent

Use a fixed-wire balloon to track alongside a buddy wire

Mount a stent on a balloon with the tip partially inflated

If only the balloon needs to enter the stented segment, inflate the balloon with 1–2 atm so the balloon centers the wire in the lumen and facilitates the crossing of the wire and balloon

Modified from Nguyen T, *et al. J Interventional Cardiol* 2002;15:237–241.

Malpositioned or Embolized Stent

Several techniques for recovery of damaged or embolized stents have been proposed. These include loop snares, basket retrieval devices, biliary forceps, biopsy forceps, and other specifically designed retrieval systems.

Stent Perforation

Consider using a covered stent (see Chapter 4).

Stent-Related Dissection, Thrombosis and Ischemia

The following factors are associated with an increased risk of stent thrombosis:

- Inadequate stent expansion
- Dissection, not covered by the stent
- Poor distal runoff or infarct in related vessels
- Presence of thrombus
- Subtherapeutic anticoagulation
- Vessels <2.5 mm in diameter.

Subacute thrombotic occlusion may occur in a small proportion of patients, usually about the third to fifth day after the implantation, but may also happen during the week following discharge. Risk factors for subacute occlusion are noted above. The risk of subacute thrombosis is increased when multiple overlapping stents are used, probably because of the abnormal rheologic (flow) environment produced. Subacute occlusion is treated with repeat balloon dilatations and continuation antiplatelet agents.

Ruptured or Tethered Balloon

Loss of inflation pressure during expansion of the stent can indicate balloon perforation. Exchange the ruptured balloon for a new one. If balloon rupture occurs after the ends of the stent are flared and anchored in the artery wall, the balloon can be deflated, rotated two or three times inside the stent, and gently pulled back inside the sheath and removed. A new balloon catheter is introduced through the sheath and positioned inside the partially expanded stent. Inflation of the new balloon catheter then completes the expansion and deployment of the stent. Alternatively, a rapid high-pressure inflation can deploy the partially opened balloon/stent enough to fully expand the stent and withdraw the balloon.

A **tethered balloon** may be caught on the edge of the stent. The ends of the stent may not have been expanded and anchored securely in the arterial wall. The balloon should be deflated, advanced slightly to the stent edge, rotated, and gently withdrawn.

Safety of Magnetic Resonance Imaging After Stent Implantation

A magnetic resonance imaging (MRI) scan should not be performed until the implanted stent (only if ferromagnetic) has been completely endothelialized (6–8 weeks), in order to minimize the risk of migration of the stent under a strong magnetic field. The stent may cause artifacts in MRI scans due to distortion of the magnetic field. Most stents may be nonferromagnetic.

Stent Implantation Shortly Before Noncardiac Surgery

Catastrophic outcomes have been reported for stenting after noncardiac surgery. Kaluza *et al*. noted that patients who underwent coronary stent placement less than 6 weeks before noncardiac surgery requiring general anesthesia had a high incidence of myocardial infarction, bleeding, and death. Among 40 consecutive patients, there were 7 myocardial infarctions, 11 major bleeding episodes, and 8 deaths; 4 patients expired after undergoing surgery 1 day after stenting. Stent thrombosis accounted for most of the fatal events, with the time between stenting and surgery as the main determinant of the outcome. It is recommended that elective noncardiac surgery be postponed for 2–4 weeks after coronary stenting, which should permit completion of the mandatory antiplatelet regimen and stent re-endothelialization, reducing the risk of stent thrombosis and bleeding complications.

CLINICAL PROCEDURE FOR PCI

PCI has been performed in laboratories with and without on-site surgical backup. Criteria for performance of PCI without surgical backup are provided in Boxes 1-8 and 1-9.

Pre-PCI Workup

Noninvasive Testing for Ischemia.

- Electrocardiogram (ECG) (evidence of resting ischemia/recent infarction)

Box 1-8

Criteria for the Performance of Angioplasty at Hospitals without On-Site Cardiac Surgery

The operators must be experienced interventionalists who regularly perform elective intervention at a surgical center (75 cases/year). The institution must perform a minimum of 36 primary PCI procedures per year.

The nursing and technical catheterization laboratory staff must be experienced in handling acutely ill patients and comfortable with interventional equipment. They must have acquired experience in dedicated interventional laboratories at a surgical center. They participate in a 24-hour, 365-day call schedule.

The catheterization laboratory itself must be well equipped, with optimal imaging systems, resuscitative equipment, and intra-aortic balloon pump (IABP) support, and must be well-stocked with a broad array of interventional equipment.

The cardiac care unit nurses must be adept in hemodynamic monitoring and IABP management.

The hospital administration must fully support the program and enable the fulfillment of the above institutional requirements.

There must be formalized written protocols in place for immediate (within 1 hour) and efficient transfer of patients to the nearest cardiac surgical facility that is reviewed/tested on a regular (quarterly) basis.

Primary intervention must be performed routinely as the treatment of choice around the clock for a large proportion of patients with acute myocardial infarction, to ensure streamlined care paths and increased case volumes.

Case selection for the performance of primary angioplasty must be rigorous. Criteria for the types of lesion appropriate for primary angioplasty and for the selection for transfer for emergency aortocoronary bypass surgery are shown in Box 1-9.

There must be an ongoing program of outcomes analysis and formalized periodic case review.

Institutions should participate in a 3–6-month period of implementation, during which time development of a formalized primary PCI program is instituted that includes establishing standards, training staff, detailed logistic development, and creation of a quality assessment and error management system.

From Smith SC Jr, Dove JT, Jacobs AK, *et al.* ACC/AHA guidelines for percutaneous coronary intervention (revision of the 1993 PTCA guidelines)—executive summary: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee to revise the 1993 guidelines for percutaneous transluminal coronary angioplasty) endorsed by the Society for Cardiac Angiography and Interventions. *Circulation* 2001;103:3019–3041.

Box 1-9

Patient Selection for Angioplasty and Emergency Aortocoronary Bypass at Hospitals without On-Site Cardiac Surgery

Avoid intervention in hemodynamically stable patients with:

- Significant (60%) stenosis of an unprotected left main coronary artery upstream from an acute occlusion in the left coronary system that might be disrupted by the angioplasty catheter
- Extremely long or angulated infarct-related lesions with TIMI grade 3 flow
- Infarct-related lesions with TIMI grade 3 flow in stable patients with three-vessel disease
- Infarct-related lesions of small or secondary vessels
- Lesions in other than the infarct artery.

Transfer for emergency aortocoronary bypass surgery patients with:

- High-grade residual left main or multivessel coronary disease and clinical or hemodynamic instability
  —After angioplasty or occluded vessels
  (Preferably with intra-aortic balloon pump support)

Adapted from Wharton TJ Jr, McNamara NS, Fedele FA, *et al.* Primary angioplasty for the treatment of acute myocardial infarction: experience at two community hospitals without cardiac surgery. *J Am Coll Cardiol* 1999;33:1257–1265.

- Exercise stress with/without perfusion imaging or echo left ventricular (LV) wall motion as indicated; pharmacologic stress study (e.g., dipyridamole); two-dimensional echocardiogram (as indicated for assessment of LV function or valvular heart disease)
- Coronary angiography evaluation (may require in-laboratory translesional physiology assessment of flow or pressure for objective evidence of ischemia before intervention).

Note: The order of the tests depends on the clinical presentation.

Pre-PCI Preparation

- Patient preparation (intravenous access, meds, consent)
- Patient and family teaching (procedure, results, complications)
- Cardiothoracic surgeon consultation, particularly for high-risk, multivessel disease or decreased LV function

- Appropriate laboratory work (type and cross-match, complete blood cell and platelet counts, prothrombin time (PT), partial thromboplastin time (PTT), electrolytes, blood urea nitrogen (BUN), creatinine).

Patient Preparation in Catheterization Suite

- ECG (inferior and anterior wall leads): 12-lead (radiolucent) ECG
- IV lines
- Skin-prepare both inguinal areas or wrist for radial artery (venous access for temporary pacing no longer routine; consider for high-risk patient, acute myocardial infarction, left bundle branch block requiring RCA PCI; rotoblator) or thrombus aspiration device).
- Aspirin (325 mg PO): failure to administer aspirin before PCI is associated with a two to three times higher acute complication rate
- Calcium antagonists are no longer routine unless used for hypertension or known coronary spasm.
- Diphenhydramine (25 mg IV or PO)
- Heparin 40–70 u/kg bolus (or smaller bolus if IIb/IIIa blocker used). Target activated clotting time (ACT) >200 sec. Heparin is an important component for PCI, despite dosing uncertainties and an unpredictable therapeutic response with the unfractionated preparation. Higher levels of anticoagulation with heparin are roughly correlated with therapeutic efficacy in the reduction of complications during coronary angioplasty, albeit at the expense of bleeding complications at high levels of heparin. Weight-adjusted heparin provides a clinically superior anticoagulation method over fixed heparin dosing.
- Clopidogrel 375 mg loading dose and then 75 mg PO daily preceding procedure
- Consider glycoprotein IIb/IIIa blockers
- Demerol (25–50 mg IV) or fentanyl (50–100 mg IV).

Guiding Coronary Arteriograms

Perform after giving 100–200 mg intracoronary nitroglycerin

- Define coronary anatomy and collateral supply (if any)
- Store guiding shots to use as reference map for balloon/stent positioning

- Selective device size—use known guide catheter diameter used to select the balloon/stent diameter.

Note: 8F = 2.87 mm, 6F = 2.0 mm. (Size of PCI device based on distal artery normal reference segment; balloon:artery ratio <1:1.2.)

PCI Catheter Technique

1. Guiding catheter selected for angle of vessel takeoff and optimum backup
2. Seat guiding catheter. Coaxial alignment is best. Side holes for pressure damping
3. Guide wire to target vessel stenosis and distal position
4. Balloon/stent inserted through hemostasis valve on guide catheter.

Crossing and Dilating the Lesion with Balloon/Stent

1. Advance balloon/stent into center of lesion
2. Maintain centering of the balloon (use radio-opaque marker(s) on balloon)
3. Inflate balloon
4. Use adequate inflation pressure (to remove "dumbbell" indentation of lesion on partially inflated balloon). A balloon may be inflated for 30–120 seconds as tolerated; stents for 10–20 seconds.

Assessing the Result of the Dilatation

- Enlarged artery lumen (≤20% residual lesion)
- Good angiographic flow (TIMI 3)
- Observe for adverse angiographic markers (thrombus or dissection)
- No residual ischemia (ECG changes with or without chest pain).

Considerations for Additional Stenting

- Distal segment recoil
- Large dissection
- Slow flow (may need measurement of fractional flow reserve [FFR] or IVUS to establish presence of dissection)
- Ischemia

Postprocedure Angiograms and Sheath Care

- Remove guidewire for final images after additional intracoronary nitroglycerin
- Femoral angiography before vascular closure device selection (perform right anterior oblique view for right femoral artery)
- Vascular closure device
- Alternatively, suture arterial and venous sheaths in place. Remove in 4 hours
- No prolonged (>6 h) heparin infusions unless there are special circumstances.

Post-Procedure Out of Lab

- Teaching on hospital course and bleeding problems, late complications, re-stenosis
- Notification of departments, intensive care (or other appropriate patient care area), operating room and surgical team stand down
- Lab and ECG
- **Post-procedure evaluation of ischemia:** After PCI, chest pain may occur in as many as 50% of patients. ECG evidence of ischemia identifies those at significant risk of acute vessel closure. When angina pectoris or ischemic ECG changes occur after PCI, the decision to proceed with further interventional procedures, coronary artery bypass graft surgery, or medical therapy should be individualized, based on factors such as hemodynamic stability, amount of myocardium at risk, and the likelihood that the treatment will be successful. Following PCI, in-hospital care should monitor the patient for recurrent myocardial ischemia, achieve puncture site hemostasis, and detect and prevent contrast-induced renal failure.

Post-PCI Medications

- Aspirin (325 mg PO daily)
- Clopidogrel 325 mg (load) and 75 mg/d, PO for at least 2–4 weeks (3–6 months for drug-eluting stents). Consider initiating statin drugs. Restart antihypertensives or antianginal drugs depending on clinical needs
- Restart antianginal drugs. Initiate statin therapy.

Appropriate secondary atherosclerosis prevention programs should be started involving adherence to recommended

medical therapies and behavior modifications to reduce morbidity and mortality from coronary heart disease.

Patients with renal dysfunction and diabetes should be monitored for contrast-induced nephropathy. In addition, those patients receiving higher contrast loads or a second contrast load within 72 hours should have their renal function assessed. Whenever possible, nephrotoxic drugs (certain antibiotics, nonsteroidal anti-inflammatory agents, and cyclosporin) and metformin (especially in those with pre-existing renal dysfunction) should be withheld for 24–48 hours after PCI.

Follow-up Schedule

- Exercise treadmill test (not before 6 weeks after PCI). Exercise treadmill testing or radionuclide scintigraphy may be performed about 6 weeks after angioplasty to establish functional status. Most ischemic tests are sufficiently specific to screen asymptomatic patients for restenosis. Angiography, and potentially repeat PCI, is reserved for patients with positive results of functional tests or recurrent ischemic symptoms. There is no indication for exercise testing within 2 weeks after the procedure. Because myocardial ischemia, whether painful or silent, worsens prognosis, some physicians advocate routine testing. However, the American Heart Association and American College of Cardiology (AHA/ACC) practice guidelines for exercise testing favor selective evaluation in patients considered to be at particularly high risk (e.g., patients with decreased LV function, multivessel coronary artery disease, proximal left anterior descending disease, previous sudden death, diabetes mellitus, hazardous occupations, and suboptimal PCI results). For many reasons, stress imaging is preferred to evaluate symptomatic patients after PCI. If the patient's exertional capacity is significantly limited, coronary angiography may be more expeditious to evaluate symptoms of typical angina. Exercise testing after discharge is helpful for activity counseling and/or exercise training as part of cardiac rehabilitation. Neither exercise testing nor radionuclide imaging is indicated for the routine, periodic monitoring of asymptomatic patients after PCI without specific indications.
- If symptoms or signs of ischemia, repeat coronary angiography.
- Return to activities of daily living. Most patients are able to return to work within 1–2 days after PCI. Factors preventing

rapid return to work include access site complications and persistent symptoms. A functional (ischemic testing) evaluation for patients with multivessel coronary angioplasty or incomplete revascularization after angioplasty will indicate the limitations, if any, on work status.

Risk-Factor Modifications

All patients should be instructed about risk-factor modification and medical therapies for secondary atherosclerosis prevention before leaving the hospital. The interventional cardiologist should emphasize these measures directly to the patient and family. Failure to do so suggests that secondary prevention therapies are not important. The interventional cardiologist should contact the primary care physician regarding the secondary prevention therapies initiated and those to be maintained, including aspirin therapy, hypertensive control, diabetic management, aggressive control of serum lipids to a target low-density lipoprotein goal of less than 100 mg/dL following AHA guidelines, abstinence from tobacco use, weight control, regular exercise, and ACE inhibitor therapy as recommended in the AHA/ACC consensus statement on secondary prevention.

MEDICAL THERAPY AFTER PCI

Anticoagulant Drugs

Anticoagulant drugs (heparin, enoxaparin) are needed only for the brief intraprocedural period. Unless indicated by unusual circumstances (e.g., continued intracoronary thrombus formation) only bolus heparin without later IV infusions is used. In some labs, low-molecular-weight heparin (enoxaparin) is replacing bolus unfractionated heparin for PCI.

Warfarin is not used for PCI but may be needed for other reasons such as atrial fibrillation or severe LV dysfunction. Orally administered anticoagulants (warfarin) after PCI are no more effective than aspirin for preventing restenosis or abrupt closure.

Antiplatelet Agents

Platelet deposition on balloon-damaged intima is partially inhibited by selected antiplatelet regimens (**aspirin and clopidogrel/ticlopidine**). Acute re-occlusion is more frequent

in patients who have not received aspirin before angioplasty. Late stent thrombosis is also more frequent in patients not receiving clopidogrel.

Antiplatelet agents of the theinopyridine family (clopidogrel or ticlopidine) inhibit platelets by blocking adenosine diphosphate (ADP)-stimulated aggregation and are highly effective for preventing subacute thrombotic occlusion after stenting. A rare associated side effect of ticlopidine and less so of clopidogrel is thrombotic thrombocytopenia purpura. Clopidogrel appears to be the currently preferred oral antiplatelet drug. Recommended antiplatelet regimens

Box 1-10

Considerations for the Assessment and Maintenance of Proficiency in Coronary Interventional Procedures

Institutions

Quality assessment monitoring of privileges and risk stratified outcomes
Provide support for a quality assurance staff person (e.g., nurse) to monitor complications
Minimal institutional performance activity of 200 interventions per year with the ideal minimum of 400 interventions per year
Interventional program director who has a career experience of more than 500 PCI procedures and is board certified by the American Board of Internal Medicine (ABIM) in interventional cardiology
Facility and equipment requirements to provide high resolution fluoroscopy and digital video processing
Experienced support staff to respond to emergencies
Establishment of a mentoring program for operators who perform fewer than 75 procedures per year by individuals who perform 150 procedures per year.

Physicians

Procedural volume of 75 per year
Continuation of privileges based on outcome benchmark rates with consideration of not granting privileges to operators who exceed adjusted case-mix benchmark complication rates for a 2-year-period
Ongoing quality assessment comparing results with current benchmarks, with risk stratification of complication rates
Board certification by ABIM in interventional cardiology

From Hirshfeld JW, Elllis SG, Faxon DP, *et al. J Am Coll Cardiol* 1998;31:722–743.

include aspirin (80–365 mg/day) and clopidogrel (75 mg PO daily).

The intravenous glycoprotein-receptor-blocking platelet drugs, abciximab, tirofaban, and eptifibitide, block the final common pathway of platelet activation of the platelet receptor (called glycoprotein IIb/IIIa) and are highly effective in blocking platelet adhesion (sticking to vessel wall) and aggregation (clumping together). Reduced acute and subacute adverse event rates are reported for all three drugs. All high-risk interventions should consider using abciximab with heparin.

TRAINING FOR CORONARY ANGIOPLASTY

Advances in interventional procedures have maintained high and durable success rates despite increasingly complex procedures. The need for appropriate training and guidelines for the procedure is obvious. Recent guidelines for the assessment and proficiencies of coronary interventional procedures have been summarized in a report from the joint task force from the AHA/ACC (Box 1-10).

To be eligible for ABIM board certification in interventional cardiology requires documentation of training in an accredited

Table 1-7

Recommendations for Clinical Competence in Percutaneous Transluminal Coronary Angiography: Minimum Recommended Number of Cases per Year

	Bethesda Conference	Society for Cardiac Angiography	ACC/ AHA	ACP/ ACC/ AHA	ACC/ AHA (1993)
Training					
Total number of cases	125	125	125	125	125
Cases as primary operator	75	75	75	75	75
Practicing					
Number of cases per year to maintain competency	–	50	52	75	75

fellowship program during which a minimum of 125 coronary angioplasty procedures including 75 performed with the trainee as primary operator (Table 1-7).

RECOMMENDATIONS FOR PCI AT HOSPITALS WITH AND WITHOUT SURGICAL BACKUP

According to AHA/ACC/SCAI recommendations, guidelines for PCI at hospitals without surgical backup are recorded in Boxes 1-8 and 1-9.

Suggested Readings

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2

ARTERIAL AND VENOUS ACCESS AND HEMOSTASIS FOR INTERVENTIONAL PROCEDURES

Morton R. Rinder and Morton J. Kern

VASCULAR ACCESS

Techniques for vascular access for interventional procedures are identical to those used for diagnostic catheterization. The approaches, technique, assessment, and methods have been described in detail in *The Cardiac Catheterization Handbook* (Mosby), but will be reviewed here with specific emphasis on interventional procedures. The site and type of access, either femoral or radial artery, are determined by the anatomic and pathologic conditions under consideration. Review of the previous difficulties encountered during diagnostic procedures or by other operators is helpful to avoid known pitfalls and potential complications. As with diagnostic studies, assessment of all arterial pulses before and after the procedure is mandatory. Remember, vascular access is the most common cause of procedural morbidity.

Percutaneous Femoral Artery Approach

Because femoral access is the most commonly used technique and because of the need for large-diameter interventional equipment, the femoral artery approach is often preferred to the arm approach. Conditions in which radial (or rarely brachial) artery access should be considered are listed in Box 2-1.

Technique. The proper position for puncture should be localized relative to the femoral artery bifurcation and can

Box 2-1

Conditions in Which Radial (or rarely Brachial) Artery Access Should be Considered

Claudication
Absent leg pulses
Femoral bruits
Prior femoral artery graft surgery
Extensive inguinal scarring from previous procedures
Surgery or radiation treatment
Excessive tortuous iliac and lower abdominal aorta
Abdominal aortic aneurysm
Severe back pain or inability to lie flat
Downward origin of renal arteries
Patient request

be identified by visualizing the head of the femur and the planned path of the needle by fluoroscopy. In a manner identical to diagnostic vascular access, the operator locates the artery (Fig. 2-1) and administers local anesthesia (see *The Cardiac Catheterization Handbook*, Chapter 2). Single front wall puncture is highly desirable for two reasons (Fig. 2-2):

- To reduce chances of bleeding in the setting of potent anticoagulation and antiplatelet agents
- To perform successful vascular closure—if a second site puncture occurs, a vascular closure device cannot be used with confidence in obtaining hemostasis.

For these reasons, the supervising physician or senior fellow in training or attending should ensure proper arterial access in all patients, but especially those going on to intervention. Multiple punctures will be a source of bleeding and potential complications, including retroperitoneal hematoma, femoral pseudoaneurysms, or arteriovenous fistula post-procedure.

Once the artery has been punctured, a standard 0.38 inch guidewire is inserted. A sheath and dilator assembly is advanced into the vessel and the sheath dilator is removed and the sheath flushed.

When an interventional procedure is performed on another day after the diagnostic catheterization, interventional access on the contralateral side should be considered. Puncturing the

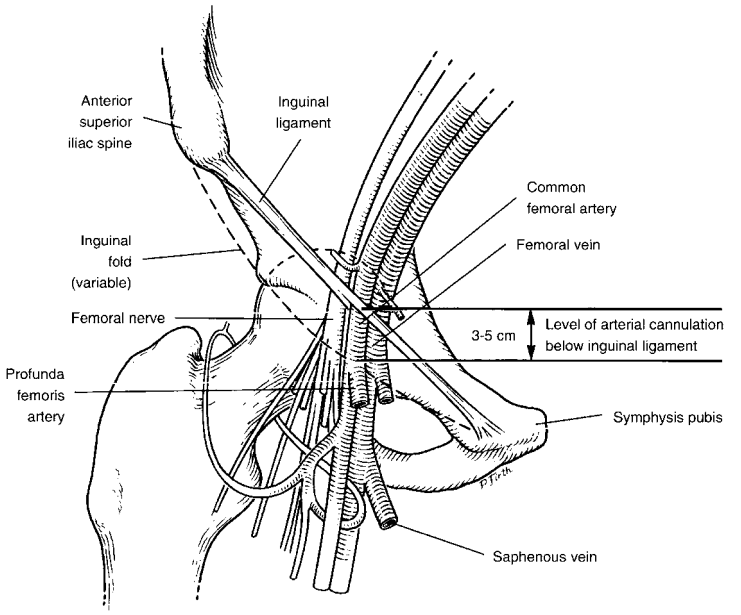


Fig. 2-1 Inguinal anatomy and guidelines for correct vascular access. (From Kulick DL, Rahimtoola SH, eds. *Techniques and applications in interventional cardiology*. St Louis, MO: Mosby, 1991: 2.)

same groin soon after diagnostic access may be associated with a higher incidence of bleeding or infection.

Key Points for Arterial Access for Interventional Procedures.

- In obese patients, place a hemostat or marker over the planned puncture site and fluoroscopically visualize the femoral head and insert the needle more caudally.
- Use a Doppler needle for deep or difficult punctures.
- Favor radial access for obese patients or those with coagulopathies (for example low platelet count, high INR, etc.).
- If anticipating need for larger PCI catheters or two catheters in the same guide, use a large (greater than) #6 French arterial sheath.

Percutaneous Femoral Vein Puncture

Femoral vein puncture is performed like the arterial puncture. Indications for femoral venous sheath placement in patients

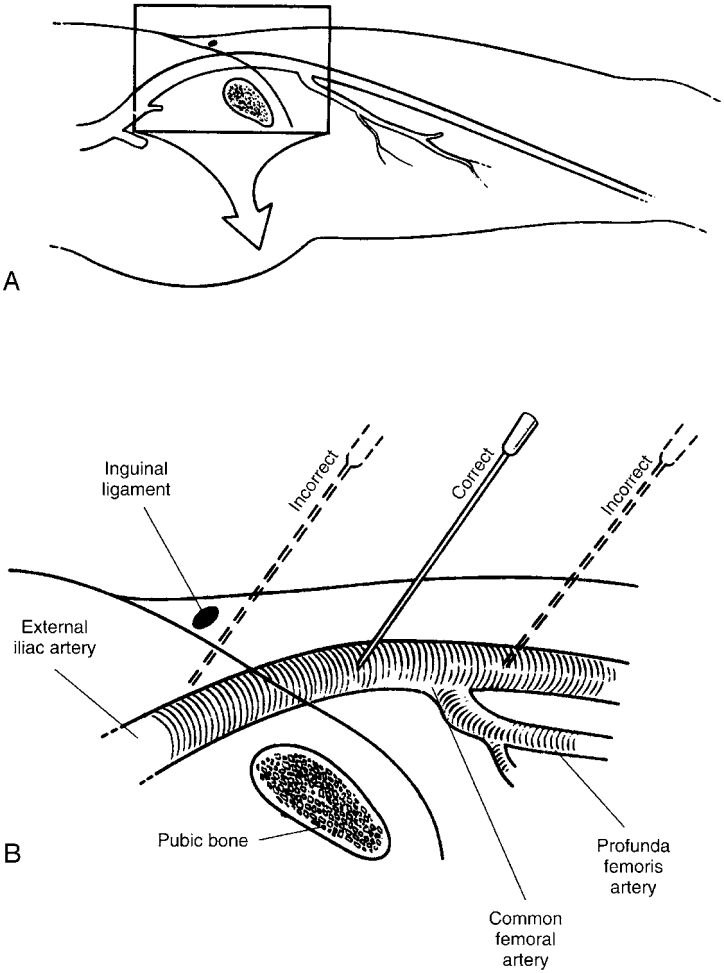


Fig. 2-2 **A, B,** Technique of single-wall arterial puncture. Parasagittal cross-sectional diagram of inguinal region at level of femoral artery. Correct needle entry position is below inguinal ligament and above femoral artery bifurcation. Correct access is particularly critical for procedures. (From Kulick DL, Rahimtoola SH, eds. *Techniques and applications in interventional cardiology*. St Louis, MO: Mosby, 1991: 3.)

undergoing interventional procedures include the need for additional intravenous access, a temporary pacemaker or pulmonary artery pressure monitoring. Caution should be used to avoid inadvertent additional arterial puncture. For this reason, if femoral vein access is needed, start with the vein access before arterial puncture.

The Arm Approach—Radial Artery Catheterization

The technique of radial artery access for diagnostic and interventional procedures has gained worldwide acceptance. Kiemeneij of the Netherlands pioneered the radial approach for coronary interventions, increasing the success rate, improving patient comfort, and providing a method for excellent hemostasis in the fully anticoagulated patient, who must remain so after intervention.

Advantages. The radial approach has several distinct advantages.

- The radial artery is easily accessible in most patients and is not located near significant veins or nerves
- The superficial location makes for easy access and control of bleeding
- In patients with a normal Allen's test, no significant clinical sequelae occur after radial artery occlusion because collateral flow to the hand occurs through the ulnar artery
- Patient comfort is enhanced. The patient can sit up and walk immediately after the procedure.

Patient Selection and the Allen Test. Patients with a normal Allen's test are candidates for the radial approach with 5 and 6 French sheaths and catheters. The Allen's test assesses ulnar flow. The test is done as follows: The radial and ulnar arteries are simultaneously occluded while the patient makes a fist. When the hand is opened, it appears blanched. Release of the ulnar artery should result in return of hand color within 8–10 seconds. A satisfactory ulnar flow can also be documented in the setting of an abnormal Allen's test by pulse oximetry. Small or female patients are more likely to have spasm in the radial artery, which can be treated effectively with intra-arterial nitroglycerin (200 μg) or verapamil (100–200 μg). Specially coated hydrophilic sheaths are also helpful to reduce spasm on sheath insertion and removal.

Patient Preparation. The patient should be well sedated and comfortably positioned. The arm is abducted at a 70° angle on an arm board for sheath insertion. A movable arm board allows the arm to be positioned at the patient's side during the procedure. A roll of sterile towels is used to support the wrist in a hyperextended position. A topical anesthetic is helpful to decrease the amount of lidocaine needed for local infiltration over the radial pulse (Fig. 2-3). Large amounts of lidocaine may obscure the pulse and make cannulation more difficult. Before complete sheath introduction, instill a cocktail of nitroglycerin, verapamil, and lidocaine to reduce artery spasm and improve patient comfort. The sheath is flushed and cared for using the same technique as for femoral sheath care.

Equipment Selection for Radial Artery Access. Arterial puncture is best achieved with a 20 gauge needle and a 0.025 inch guidewire. A radial artery sheath system of 24 cm with a graduated dilator system over the 0.025 inch guidewire is

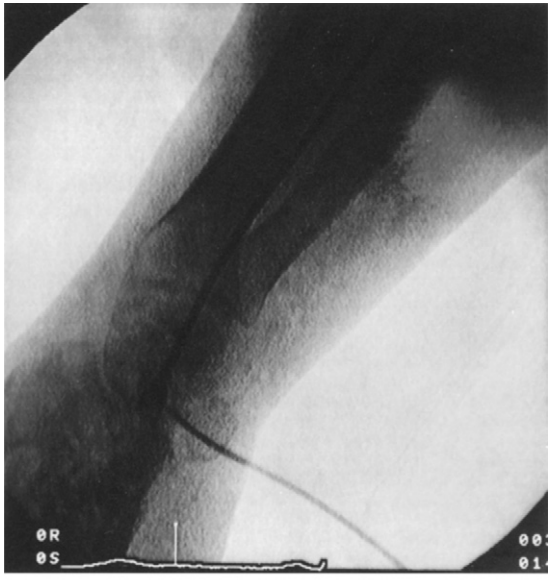


Fig. 2-3 Angiogram of radial arterial sheath placement for coronary angioplasty.

available. The long sheath technique is advocated for patient comfort and facilitates catheter manipulation. Radial artery spasm may make catheter movement difficult or impossible when a short sheath is used, and patient discomfort is more common. However, in some patients, a longer sheath may also diminish ulnar flow during the procedure.

Careful catheter selection for radial approach is important. The standard preformed diagnostic Judkins or Amplatz catheter shapes may be used but require more manipulation for selective engagement in the coronary ostium. For selective engagement of the left coronary ostium, a Judkins left 3.5 or 4.0 catheter typically is used. Use of the left radial artery approach allows for easier manipulation of the preformed Judkins shapes with minimal effort. For this approach, the left arm should be brought over the abdomen so that the operator can work from his usual position on the right side of the patient. Box 2-2 lists the commonly used catheters for approach to the radial artery.

If needed, venous access should be obtained from a brachial, internal jugular or femoral vein.

Box 2-2

Most Commonly Used Catheters for Radial Coronary Angiography

Right Coronary Artery
Multipurpose catheter
Judkins right catheter
Amplatz right catheter
Amplatz left catheter

Left Coronary Artery
Judkins left catheter (typically 3.5 cm)
Multipurpose catheter
Amplatz left catheter

Vein Grafts
Multipurpose catheter
Amplatz left catheter
Judkins right catheter

Adjunctive Medications for Radial Artery Access. After half of the arterial sheath has been inserted, a cocktail consisting of 5000 units of heparin, 2 mL of 1% lidocaine, and 200 μg of nitroglycerin is given (Box 2-3). Lidocaine improves patient comfort during catheter manipulation. An additional vasodilator such as diltiazem, verapamil, papaverine, or adenosine may be necessary to minimize spasm in the radial artery. Intra-arterial injection of 1–2 mg of verapamil through the sheath reduces painful vasospasm. Verapamil in doses up to 5 mg has been given without unwanted side effects such as hypotension or bradycardia.

Radial Artery Sheath Removal and Post Procedure Care. Before sheath removal, 1 mg of verapamil is given through the sheath to minimize spasm in the radial artery. A plastic bracelet with a pressure pad is placed around the wrist (Fig. 2-4). Gauze is wrapped around the plastic strap to prevent skin injury when the bracelet is tightened. Another folded gauze is placed under the pressure pad over the sheath insertion site. While pressing the pad over the puncture site, the sheath is gently withdrawn, the bracelet is tightened, and the pad is pressed down and locked over the puncture by tightening the bracelet bracket. The bracelet should be tight enough to ensure hemostasis but not so tight as to occlude flow to the hand. 1–2 hours later, the patient is checked and the bracelet is loosened. The bracelet can be removed later that day (more than 6 hours later

Box 2-3

Medical Regimen for Radial Catheterization

Before the Procedure
Topical anesthetic cream over the radial artery (optional)

Through the Sheath (Before Catheter Insertion)
Heparin, 2000–5000 u
Verapamil, 1–2 mg or 200–400 mg NTG
1% lidocaine, 1–2 ml

After the Procedure and Before Sheath Removal
Verapamil, 1 mg (optional)

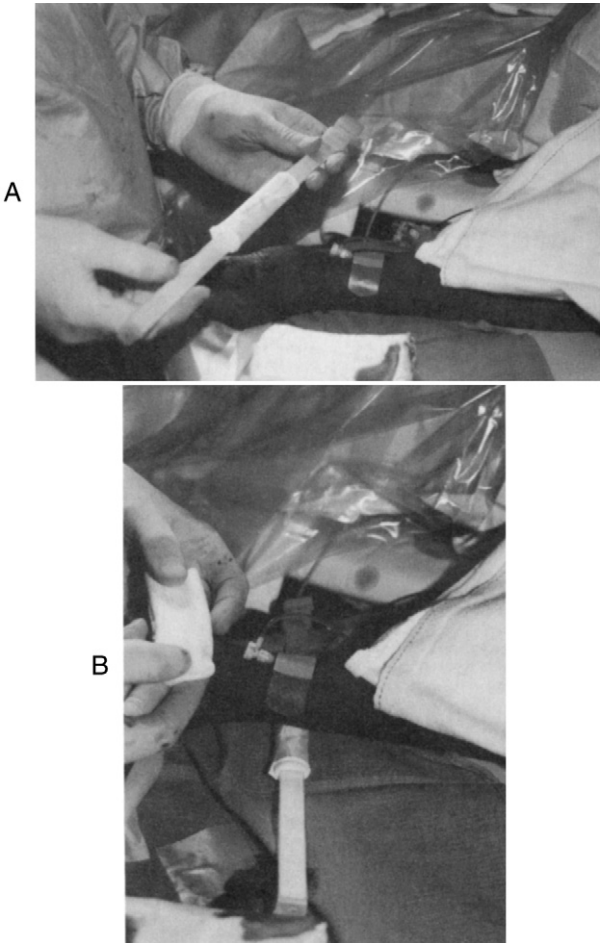


Fig. 2-4 **A,** The operator holds the plastic bracelet with gauze covering the edges. **B,** The bracelet has been placed under the wrist. Another gauze pack is folded and placed over the radial sheath beneath the pressure pad.

Continued

or next morning). Instructions to the patient should review puncture site compression with the fingers in case of late bleeding.

Key Points for Radial Artery Access.

- Always perform Allen's test
- Use adequate patient sedation and access site anesthesia
- Use clues gained during diagnostic study for left or right arm access and coronary cannulation

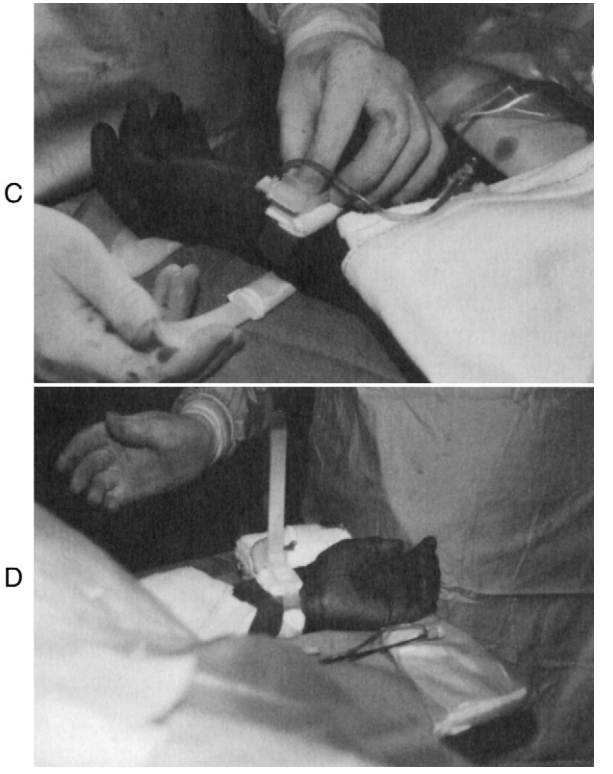


Fig. 2-4, cont'd **C,** The bracelet is engaged to hold the pad over the puncture site and while simultaneously applying pressure, the sheath is removed. **D,** The bracelet is tightened and secured over the radial puncture site. Check the hand for adequate perfusion. (From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 84, 85.)

- Work with the wrist close to the patient's body. Bring the left wrist onto the left hip for easier manipulations
- Use vasodilators, nitroglycerin, and lidocaine during sheath removal if vasospasm causes pain.

Percutaneous Brachial Artery Access

In general, percutaneous brachial artery puncture for interventional procedures is undesirable, since control of bleeding in the post-procedure period may be difficult. Brachial artery cutdown is no longer a standard technique. The brachial approach offers a closer access to the distal and descending

aortic vasculature and may be advantageous in some lower extremity or renal procedures. Approach to peripheral vascular procedures when lower extremity access is unavailable may be achieved through the brachial route.

Additional Arterial and Venous Access for High-Risk Interventions

For patients at high risk of complications who may require urgent placement of a pacemaker and intra-aortic balloon pump (IABP) or another hemodynamic support device, an additional arterial or venous access is helpful. In some patients, monitoring pulmonary artery and/or wedge pressure may help medical management during a complex procedure. For intra-aortic balloon pumping or temporary pacemaker use as a standby maneuver, a small 5 French sheath introducer can be placed in the opposite femoral artery or vein at the beginning of the procedure, permitting immediate vascular access should urgent hemodynamic or pacing support be required. Two venous cannula can be placed in one femoral vein if multiple venous catheters are anticipated. Remember, before considering IABP insertion, abdominal and iliac angiography should be performed to identify any significant peripheral vascular disease.

Overcoming Difficult Vascular Access Problems

Excessive Vessel Tortuosity. The most frequently encountered difficulty in advancing guide catheters is tortuosity of the iliac or subclavian vessels, a condition often found in elderly patients. A steerable 0.038 inch flexible guidewire (e.g., Wholey) is excellent for negotiating tortuous vessels. Its flexible, atraumatic, gently curved tip is steerable, increasing safety. In cases of extreme tortuosity, a right Judkins diagnostic catheter may be used to help direct the guidewire tip and control the advancement of the guidewire. Angiograms will delineate the arterial course and any other obstructive lesions. Once the guidewire is beyond the tortuous or narrowed segments, a long guidewire–catheter exchange will be needed thereafter. A long sheath, for example the arrow sheath, can be positioned, but they increase friction with guide catheter movement. The trade-off of multiple friction points for some straightening of the vessel against the guide catheter kinking is often worth

the effort. Catheter exchanges over a long 300 cm extra stiff exchange wire will facilitate advancement of catheters across tortuous or atherosclerotic segments.

Commonly selected equipment for tortuous vessel problems includes:

- Wholey 0.035 inch steerable guidewires
- Long 300 cm regular exchange guidewires
- Long 300 mg extra stiff exchange guidewires
- Long arterial sheaths in 23 or 90 cm.

Peripheral Vascular Disease. Peripheral vascular disease (PVD) may complicate access as well as guide catheter manipulation. Weak femoral pulses often indicate atherosclerotic obstruction at the level of the femoral, common iliac or aortoiliac bifurcation. Inability to advance the guidewire to the central aortic position requires angiography to determine further maneuvers needed to negotiate the femoral approach. In such patients, abdominal aortography and peripheral angiography are necessary to evaluate the extent of obstructive disease with focal iliac stenosis. Should a coronary intervention be required, some operators advocate iliac stent placement before proceeding with PCI. PVD may require the use of an arm approach. In patients with PVD of the lower extremities, coexistent subclavian atherosclerosis may also complicate arm access.

Inguinal Scarring or Access through Site with Previous Vascular Closure Device. Inguinal scarring may be present in patients having aortofemoral bypass surgery, femoral bypass cannula access, IABP repair or radiation therapy. In some of these patients, there may also be a synthetic arterial conduit graft. If possible, an alternative access site should be selected. Otherwise, access of a severely fibrotic or scarred groin or through a femoral bypass graft requires successive dilations with 5, 6, 7, and 8 French dilators before inserting a vascular sheath one size smaller. Most vascular closure device manufacturers indicate that re-access through a site with a recently placed closure device can be performed without a problem if the device has no internal artery fixation component. Caution should be used when re-accessing all sites but especially those closed with Angio-Seal, although no reports of Angio-Seal anchor dislodgment during re-access have been reported.

Access of sites closed with such devices after 2–4 weeks is thought to be safe. However, the contralateral femoral artery should be considered in most cases for patient comfort.

SHEATH MANAGEMENT AND HEMOSTASIS AFTER PCI

Timely and safe removal of the arterial sheath with minimal patient discomfort is an integral part of a successful coronary angioplasty procedure. Vascular access complications are the most significant cause of morbidity and increased length of hospitalization. Downsizing to less than #8 French sheath and discontinuation of post PCI heparin infusions and the use of vascular closure devices have simplified and improved sheath care and hemostasis after PCI. Although results are improving, time, personal, and other resources necessary for appropriate sheath care and hemostasis should not be minimized.

Immediate sheath removal

The arterial and venous sheaths are not routinely left in place after completing the procedure. The presence of a vascular sheath in a heavily anticoagulated patient predisposes to peri-sheath hemorrhage and local or retroperitoneal hematoma. Most laboratories remove sheaths within a few hours after the procedure or immediately remove the sheath in the laboratory and obtain hemostasis with a vascular closure device. Rare individuals may require overnight heparin infusion with next-day sheath removal.

Key Points in Post-Procedure Sheath Care and Hemostasis

- "Do it right the first time." The best results stem from meticulous arterial puncture and correct sheath placement
- After the sheath is secured in place, insert an appropriately sized obturator in the sheath to prevent sheath kinking and arterial bleeding
- Use a clear transparent dressing over the sheath for better detection of bleeding
- Inspect and palpate the puncture site and distal pulses at each post-procedure check
- If a sheath is in place to monitor arterial pressure, use a closed sterile system. Minimize blood drawing from the sheath side arm
- A downward trend in blood pressure and upward trend in heart rate are early warnings of a possible retroperitoneal

hematoma forming. Back pain, abdominal pain, and confusion are also signs associated with blood loss. Consider early CT scan.

Hemostasis after PCI Sheath Removal

Sheath removal after PCI takes place on the wards or in a special PCI unit. Sheath removal proceeds as described for diagnostic procedures. Several points should be kept in mind:

- Adjust bed height or use a foot sole so as to be able to exert maximal pressure for puncture site compression with minimal fatigue
- Ensure good intravenous access
- Give local anesthetic 10–20 ml of 1% lidocaine (to the skin around the sheath and intravenous analgesics before sheath removal)
- Have atropine ready and within reach
- Before removing the sheath, check that the heparin is stopped, the activated clotting time (ACT) is less than 150 seconds, vital signs are stable, no chest pain is present, and there are no plans for recatheterization
- If an arterial and venous sheath were used, remove the arterial sheath first. Avoid prolonged pressure on the femoral vein. Prolonged venous occlusion, especially with pressure devices, may cause venous thrombosis. Check the leg and foot for cyanosis
- The duration of pressure-holding, usually 20–45 minutes, depends on the sheath size, ACT, and ease of control of the bleeding
- When longer pressure application is needed after removal of a large sheath, intra-aortic balloon pump catheter, or cardiopulmonary support cannula, the FemoStop (Radi Medical Inc., Uppsala, Sweden) or similar compression device is the preferred method of arterial compression. Compression devices provide a stable pressure, relative patient comfort, and easy adjustment of the degree of pressure applied. Compression devices are not intended for unsupervised use. The duration of pressure application should be kept to a minimum to decrease complications such as skin necrosis, femoral nerve compression, or

venous thrombosis. There are two types of compression device:

- Some laboratories employ mechanical C-type clamps to assist in puncture site hemostasis. The clamp is effective but must be applied carefully by a trained individual and must be monitored frequently for misalignment, bleeding, or excessive pressure with limb ischemia (Fig. 2-5)
- The FemoStop system (RADI Medical; Fig. 2-6) is an air-filled, clear plastic compression bubble that molds to the skin contours. It is held in place by straps passing around the hips. The amount of pressure applied is controlled with a sphygmomanometer gauge. The clear plastic dome permits visualization of the puncture site. The FemoStop is mostly used for patients in whom prolonged compression is anticipated or if bleeding persists despite prolonged manual or C-clamp compression. The duration of FemoStop compression and time to removal of the device varies depending on the patient and staff protocols. In some hospitals, the time from application to removal may be less than 30 minutes. In other patients in whom hemostasis is required, the device may be left at a lower pressure for longer.

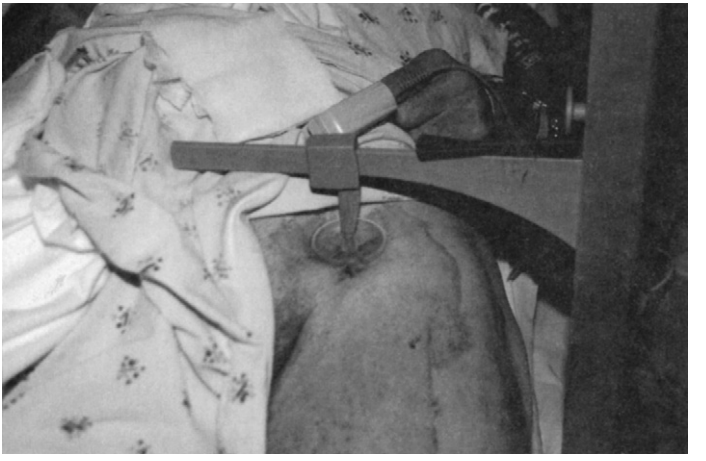


Fig. 2-5 C-clamp (Compressor™) applied to femoral puncture site. (From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 66.)

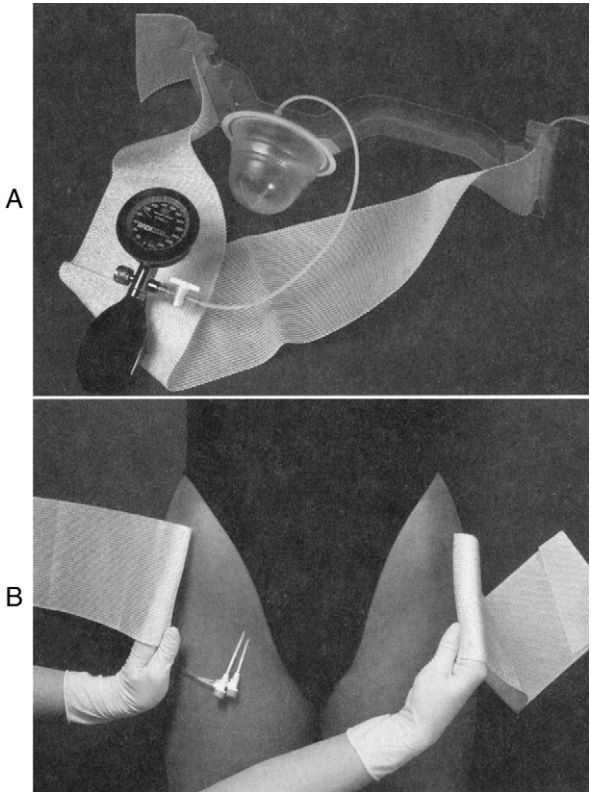


Fig. 2-6 Use of the FemoStop. **A,** Before proceeding: (1) examine puncture site carefully; (2) note and mark edges of any hematoma; (3) record current blood pressure. **B,** Step 1: Position Belt. The belt should be aligned with the puncture site equally across both hips.

Continued

Ambulation after Sheath Removal

Depending on sheath size, bed rest varies from 2 to 8 hours. Generally, ambulation can occur 6 hours after sheath removal. Ambulation should be gradual. Risk factors for pseudoaneurysm include continued anticoagulation after sheath removal, large sheaths (>10F), hematoma, and low puncture site. Palpation for pulsatile mass and auscultation for bruits should be performed before the patient is discharged.

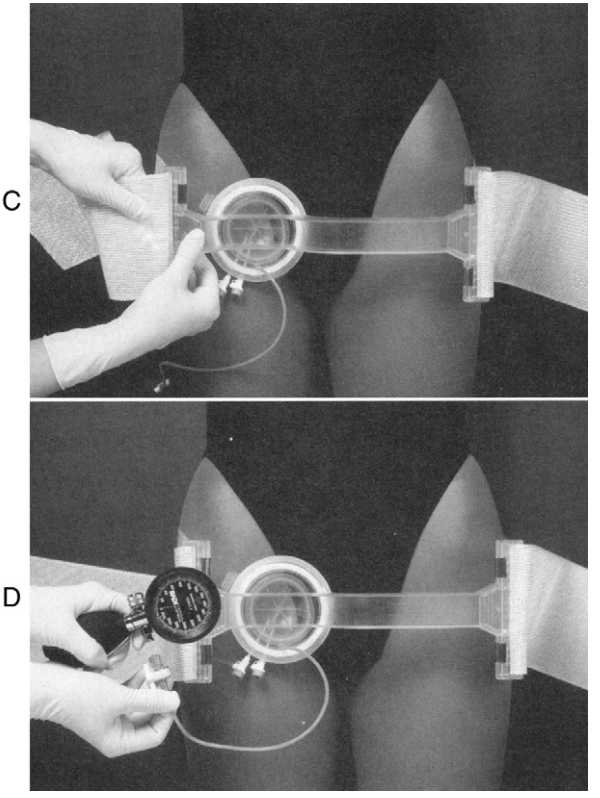


Fig. 2-6, cont'd **C,** Step 2: Center the dome and adjust belt. The dome should be centered over the arterial puncture site above and slightly toward the midline of the skin incision. The sheath valve should be below the rim of the pressure dome. Attach belt to insure a snug fit. The center arch bar should be perpendicular to the body. **D,** Step 3: Connect dome pressure pump.

Continued

Vascular Closure Devices and In-Lab Hemostasis

Immediate hemostasis can be achieved in the catheterization suite using one of several vascular closure devices. Before selecting the device, femoral angiography from an oblique projection will indicate the suitability of the device and perhaps which device should be selected. Figure 2-7 shows an anteroposterior (AP) and right anterior oblique (RAO) view of the femoral artery. Note how only the RAO view displays the bifurcation of the profunda and superficial femoral branches.

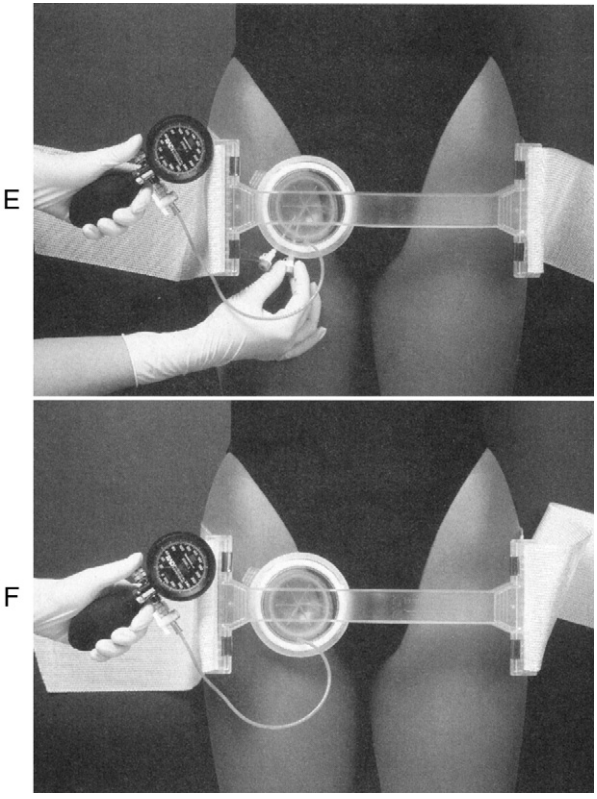


Fig. 2-6, cont'd **E,** Step 4: For a venous sheath, inflate dome to 20 or 30 mm Hg and remove sheath. To minimize formation of AV fistula, obtain venous hemostasis before the arterial sheath is removed. Step 5. For the arterial sheath, pressurize dome to 60–80 mm Hg and remove sheath and increase pressure in dome to 10–20 mm above systolic arterial pressure. **F,** Step 6: Maintain full compression for 3 minutes. Reduce pressure in dome by 10–20 mm Hg every few minutes until 0 mm Hg. Check arterial pulse. Observe for bleeding. After hemostasis is obtained, remove FemoStop and dress wound. (From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 67–69.)

Percutaneously applied devices that can safely and effectively close arteriotomies have been developed to improve patient comfort by decreasing the time patients lie flat after the procedure. The decision to use a closure device includes the fact that the device adds a small but real chance of either a complication or infection related to the device that would not

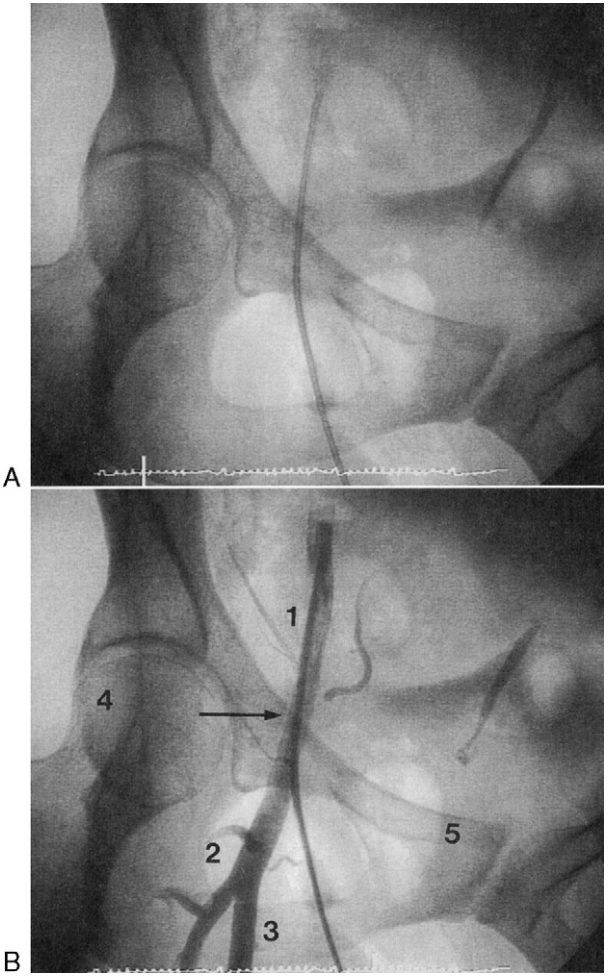


Fig. 2-7 Femoral angiogram. **A,** Angiogram of sheath in femoral artery in RAO projection. **B,** Correct positioning is seen relative to angiographic landmarks. 1, Common femoral artery; 2, bifurcation of profunda; 3, superficial femoral artery; 4, midpoint of femoral head; 5, iliac–symphysis pubis ridge (inguinal ligament line). (From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 55.)

occur with standard manual compression. Nonetheless, vascular closure device safety has been demonstrated in diagnostic catheterization and interventions. Most catheterization laboratories also report high success rates when utilizing various closure devices directly after PCI in fully anticoagulated patients receiving antithrombins, heparin, or glycoprotein receptor blockers. However, a learning curve exists for these devices. Newer-generation closure devices have steeper learning curves, faster time to deployment, and are more efficient. The four commonly used devices are shown in Figs 2-8–2-11. Collagen, either plugs (VasoSeal and Angio-Seal) or liquid (Duett), can be delivered directly to the arterial puncture site through a special sheath system (VasoSeal or Duett) or anchored inside the vessel (Angio-Seal). A percutaneous vascular suture delivery system (Perclose) also provides hemostasis. All devices facilitate early ambulation. These devices may be especially helpful in anticoagulated patients and in patients with back pain or inability to lie flat. Their advantages and disadvantages are summarized in Table 2-1. All vascular closure devices should be used with caution in patients with peripheral vascular disease or low arterial puncture at or below the femoral bifurcation.

Percutaneous Arteriotomy Closure. Perclose (Perclose Inc.)—Prostar® XL 8, Prostar® XL 10, The Closer™, The Closer-S™, The Closer-AT™ (Fig. 2-8) are suture-based devices employing differing numbers of sutures with two nitinol needles attached to each suture creating a "purse-string" method of pulling the

Table 2-1

Vascular Closure Devices

Device	Mechanism	Advantages and Limitations
AngioSeal	Collagen seal	Secure hemostasis Anchor may catch on side branch
Duett	Collagen/thrombin	Stronger collagen–thrombin seal Intra-arterial injection of collagen–thrombin
Perclose	Sutures	Secure hemostasis of suture Device failure may require surgical repair
VasoSeal	Collagen plug	No intra-arterial components Positioning wire may catch on side branch

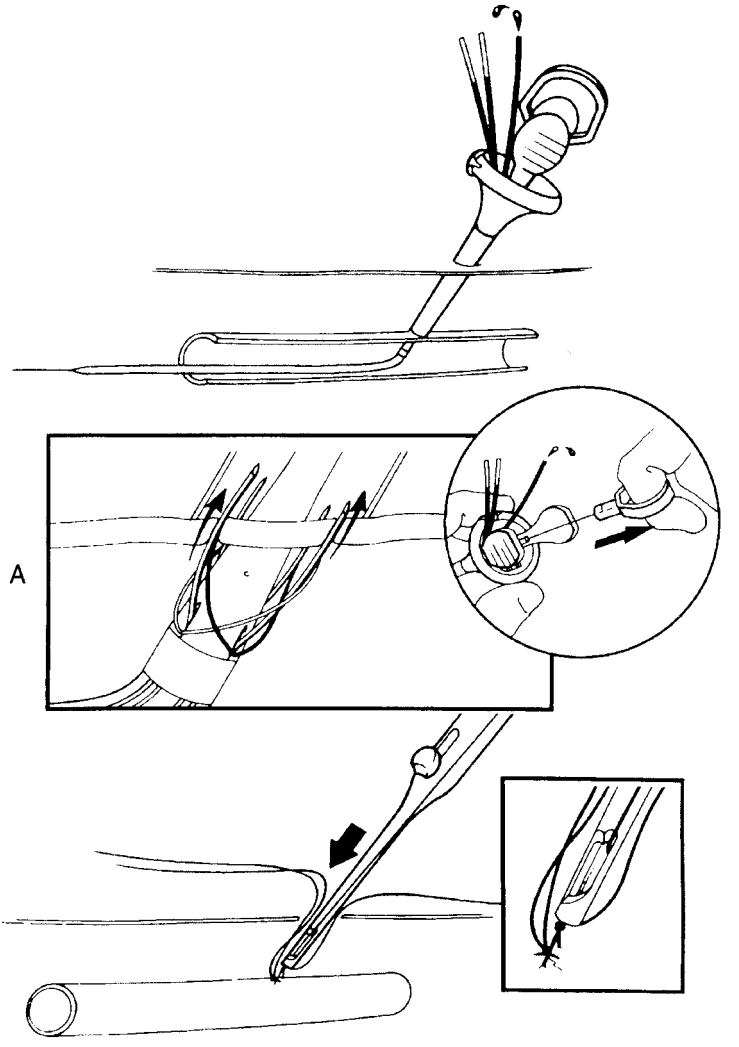
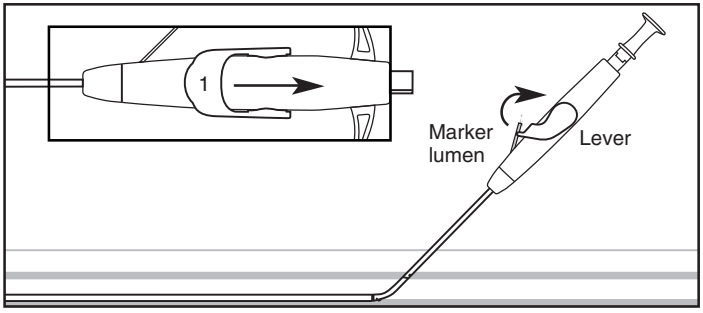


Fig. 2-8 **A,** The Perclose multiple intravascular suture device permits deployment of suture needles from within the vessel and closure of the suture from the surface of the skin. *Top panel,* The Perclose device is inserted to the level of the vessel. The fine suture needles are deployed and come through the vessel and out of the device. *Bottom panel,* The knot pusher secures the knots on top of the vessel in the subcutaneous tissue. (**A,** From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 75. Courtesy of Perclose, Menlo Park, CA.)

Continued

1. Positioning



Insert to arterial flow. Lift lever marked #1.

B

2. Needle deployment

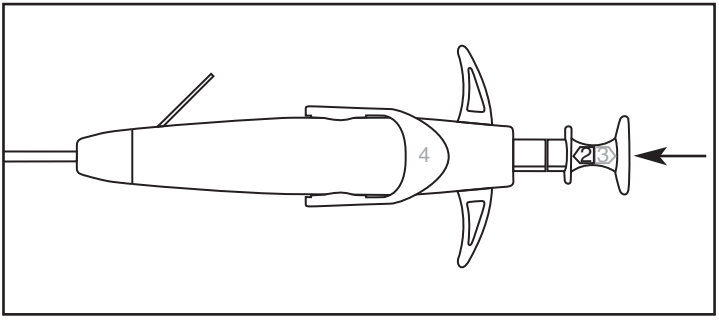


Fig. 2-8, cont'd **B,** New Perclose suture closure device.

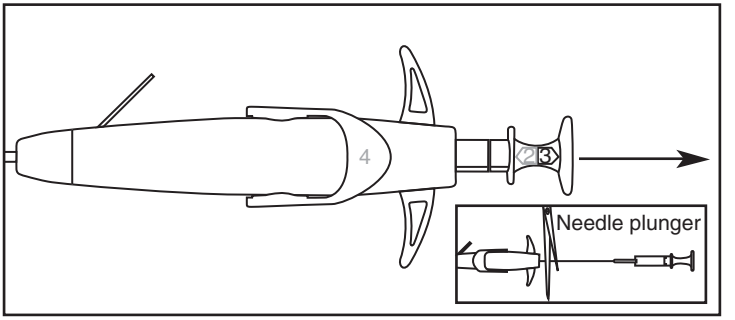
Continued

sides of the arteriotomy together. A slip knot is tied manually or automatically, allowing the operator to pull the knot down to the artery and cinch the edges of the arteriotomy together.

Helpful Hints

- With early-generation devices the operator maintains constant forward pressure while deploying the needles backwards to ensure that they exit the device, catch the walls of the arteriotomy, and then re-enter the device. However, excessive forward pressure can extend the arteriotomy outward, making closure even more perilous
- Keeping the knot loose before pulling on the "rail" suture prevents tightening of the knot before it is directly on top of the artery

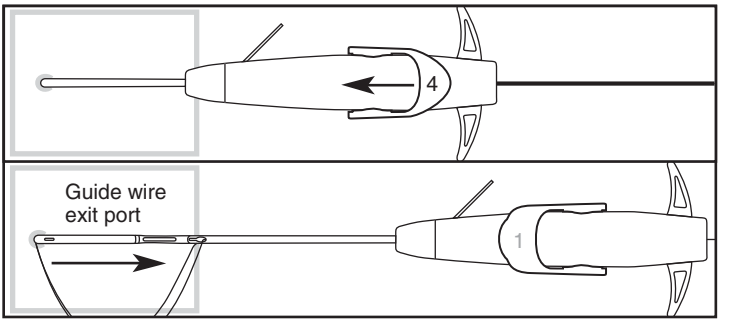
3. Plunger removal



Remove needle plunger and cut suture.

B

4. Suture harvest



Close lever marked #4 and retract device to the guide wire exit port.

Fig. 2-8, cont'd **B,** New Perclose suture device.

- Prior to closure, gentle blunt dissection is frequently necessary to allow the knot to travel smoothly through subcutaneous tissues.

Advantages.

- Suture-mediated closures have no thrombogenic material that can embolize distally
- The "Closer" devices can be used with several different sheath sizes. "Perclose" can be performed prior to sheath insertion when using very large sheaths, allowing the operator to create the purse-string before actually dilating the arteriotomy. This maneuver is currently being used in percutaneous closures of abdominal aortic aneurysms, where sheath sizes approach 22 French.

Disadvantages.

- In early generations the needles begin inside the patient and the device is designed to bring the needles through the arteriotomy into the perivascular space and back into the hub. This course has resulted in needles getting stuck within the patient and requiring surgical removal. New designs have overcome this problem
- Sutures can break if there is excessive tension on the rail suture. When this occurs, failure of the closure is imminent and will require use of external compression for hemostasis
- Arterial calcification tends to prevent needles from catching the suture in the Closer™ variation
- Currently, the device is indicated for arterial punctures in the common femoral artery only. Bifurcation closures do not respond favorably to the purse-string suture. In addition, there is no indication for superficial femoral artery sticks; however, some operators will close these if the artery is large.

Angioseal™. Angioseal STS™ 6 French, 8 French (St Jude Medical; Fig. 2-9) is a collagen sponge attached by a suture to a polyglycolic acid anchor positioned inside the arterial wall. A sandwich is created around the arteriotomy site when the intra-arterial anchor is pulled up against the inside wall simultaneously with the operator pushing the collagen sponge downward in the subcutaneous tissues. The bioabsorbable anchor softens as it warms and moistens and is completely absorbed after 10 days. The extravascular collagen sponge is absorbed over 60–90 days.

Helpful Hints.

- The latest-generation devices require several locking mechanism steps to be rigorously followed prior to anchor deployment
- Constant back pressure from the suture allows the anchor to remain snugly against the inside arterial wall. However, excessive back pressure can pull the anchor through the arteriotomy or break the suture altogether
- The introducing sheath needs to be inserted at least 2–3 cm after the back flash of blood is detected.

Advantages.

- This device is very fast and simple. No knot tying is required
- Newer generations provide better collagen coverage of the

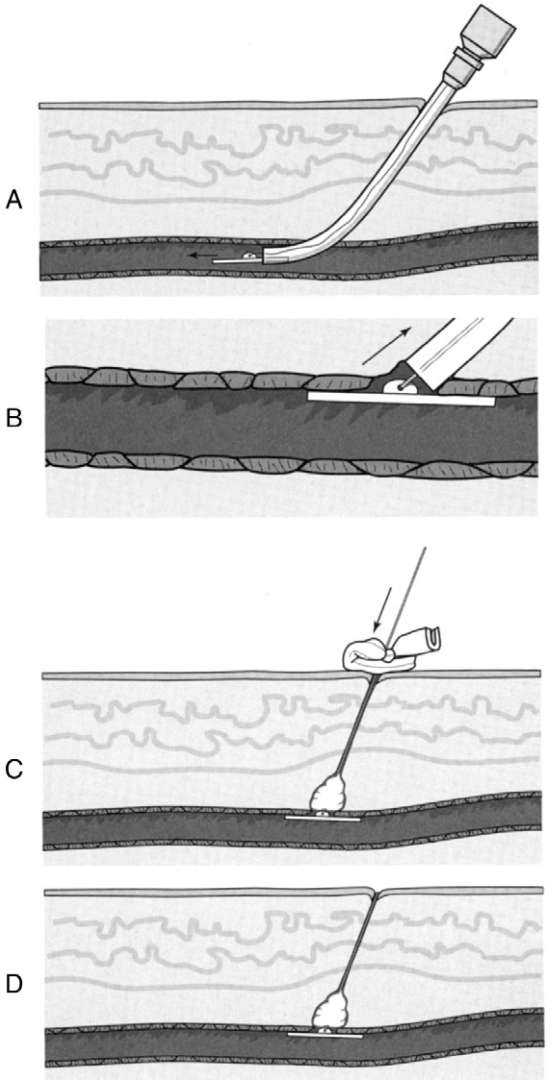


Fig. 2-9 Angioseal system. **A,** The Angioseal sheath assembly preloaded with the anchor and collagen plug is advanced into the vessel. **B,** The anchor is deployed and retraction on the system secures the anchor against the vessel wall while the sheath is removed and the collagen plug deployed outside the artery. **C,** Tamping of the suture compresses the collagen plug over the vessel. **D,** The suture is cut at the skin line leaving the subcutaneous vascular closure components hidden. (From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 72.)

arteriotomy, allowing instantaneous hemostasis and suture clipping before the patient leaves the lab.

Disadvantages.

- Since the anchor sits inside the artery, flow disruptions can occur in diseased vessels
- Potential for embolization exists, especially if the operator does not apply constant backward tension to keep the anchor fastened up against the wall
- Re-stick in the same vessel is inadvisable for 90 days but can be performed if necessary. In this case the operator should attempt to access the artery 1–2 cm above or below the site of the previous Angioseal™.

Vasoseal®. Vasoseal ES™ (Datascope, Inc.; Fig. 2-10) is a collagen plug that utilizes an intravascular positioning device but no elements are left intra-arterially once completed. The positioning device is expected to position the collagen plug immediately outside the arterial wall. The operator inserts the collagen while a second person applies occlusive pressure to the vessel. Once the collagen is inserted, pressure is slowly released to allow the blood and collagen to co-mingle and create a hemostatic plug. The collagen plug is completely absorbed within 6 weeks.

Helpful Hints.

- Blunt dissection is mandatory since the positioning sheath is designed to be much larger than the arterial sheath
- Once the first plug is inserted and meets resistance, the introducer sheath is retracted slightly in order to prevent embolization of collagen material
- Only use one plug, rather than two, in very thin patients.

Advantages.

- This sealing method can be used for any arterial stick regardless of the anatomy, pre-existing vascular disease, or calcification within the artery
- No intravascular anchor is present, so flow inside the artery is not disrupted.

Disadvantages.

- There is potential for collagen embolization with this device, as with other collagen devices

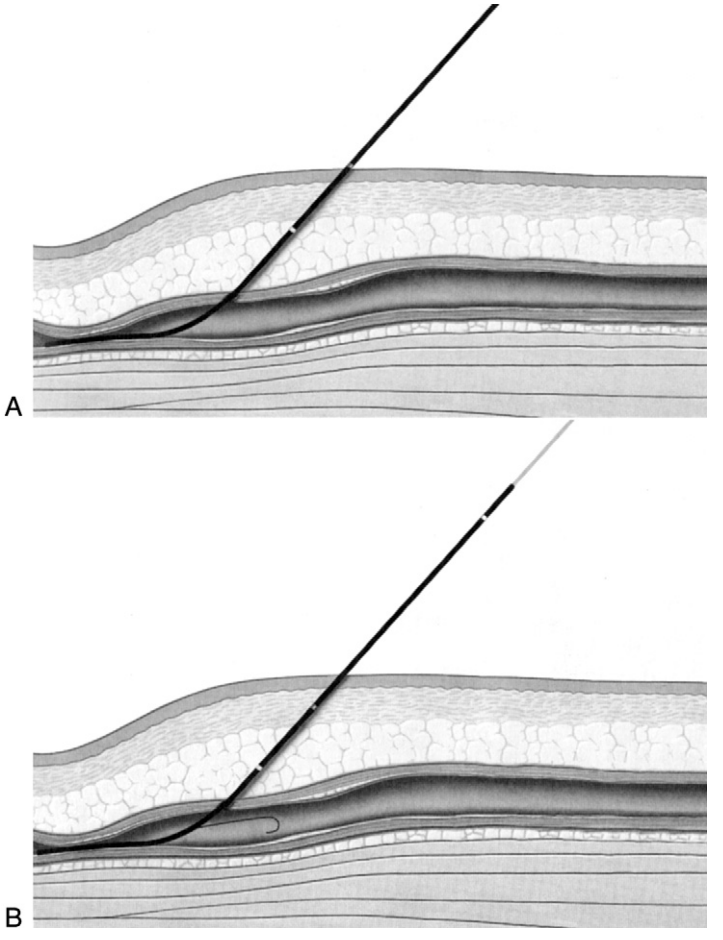


Fig. 2-10 Vasoseal vascular closure device. **A, B,** A special introducing catheter with an antegrade J wire is used to mark the artery.

Continued

- The procedure is more cumbersome than other devices, since a second assistant is required
- Re-stick in the same artery is not advised within 6 weeks. As with the Angioseal™ device, if the operator must use the same site, attempts should be made to stick the artery 1–2 cm above or below the previous site.

Duett. The Duett system (Vascular Solutions, Inc.; Fig. 2-11) utilizes the indwelling sheath, which eliminates the first step

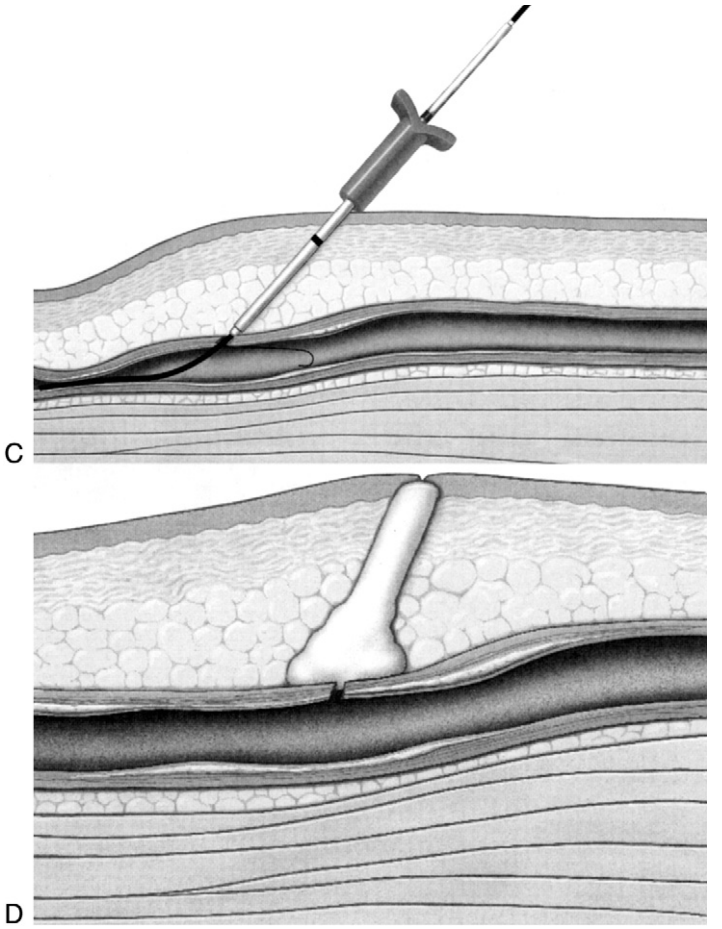


Fig. 2-10, cont'd C, A dilator and then a sheath is placed on top of the artery. **D,** A collagen plug is then inserted to achieve hemostasis. (From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 73, 74.)

involved with placement of all other devices. A special balloon catheter is inserted into the sheath and a small balloon is inflated and pulled back against the inside of the arterial wall. The sheath is then retracted until just outside the arterial lumen and a solution containing thrombin and collagen is mixed and injected through the side-port of the sheath. The sheath is retracted some more and another dose of material is

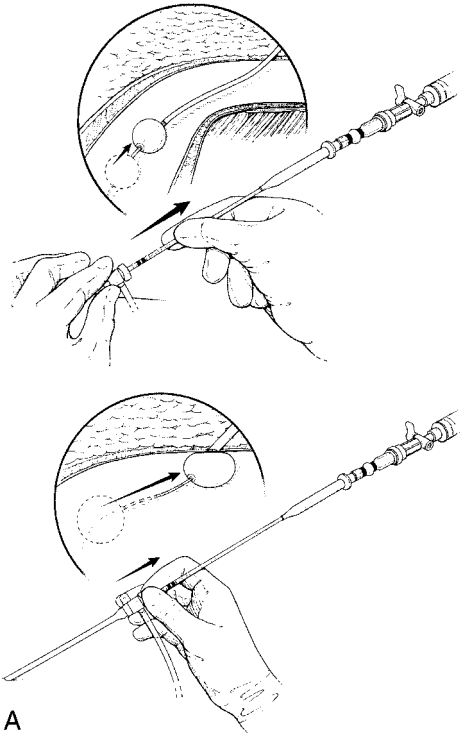


Fig. 2-11 Duett vascular closure device. **A,** A small balloon is inserted through the sheath used for the angiogram. The balloon is pulled to end of sheath.

Continued

injected. After injection an assistant keeps pressure over the site to maintain hemostasis, then slowly releases pressure over 5 minutes.

Helpful Hints.

- Maintaining proper backward tension on the balloon prevents intra-arterial thrombin injection.
- Too much balloon tension can pull the balloon through the arteriotomy.
- Do not force collagen solution into sheath. Back sheath out further then inject.
- Avoid sites that have been scarred or are fibrotic.

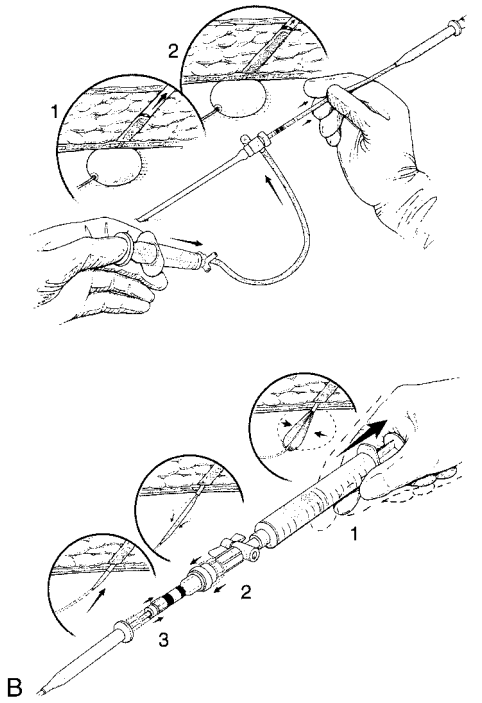


Fig. 2-11, cont'd **B,** Sheath and balloon pulled back to tamponade puncture site. Sheath is pulled back from inside of artery, aspirated to confirm sheath is out of artery and liquid collagen-thrombin mixture injected to seal outside of artery. Puncture site manually compressed. Balloon deflated and sheath/balloon assembly removed. Manual pressure maintained for 5 minutes. (From Kern, MJ. *The cardiac catheterization handbook*, 4th ed. Philadelphia, PA: Mosby, 2003: 70, 71.)

Advantages.

- The Duett balloon can be inflated to occlude puncture sites using up to a 12 French sheath, allowing the use of one device for 5–12 French systems
- There is no need for sheath removal so one step is eliminated from the process
- Since no sponge or plug is involved, immediate re-stick in the same area is permissible.

Disadvantages.

- Embolization of procoagulant material distally is a potential hazard

- The current indication is for common femoral artery stick only
- An assistant is required for proper puncture site compression and complete hemostasis.

COMPLICATIONS OF ARTERIAL ACCESS

Hemorrhage

The most common complication from femoral cardiac catheterization is hemorrhage and local hematoma formation, increasing in frequency with the increasing size of the sheath, the amount of anticoagulation, and the degree of obesity of the patient.

Other common complications (in order of decreasing frequency) include: retroperitoneal hematoma pseudoaneurysm, arteriovenous (AV) fistula formation, arterial thrombosis secondary to intimal dissection, stroke, sepsis with or without abscess formation, and cholesterol or air embolization. The frequency of these complications is increased in: high-risk procedures; critically ill elderly patients with extensive atheromatous disease; patients receiving anticoagulation, antiplatelet, and fibrinolytic therapies; and in patients receiving concomitant interventional procedures. Compared to the femoral approach, the brachial (but not radial) approach carries a slightly higher risk of vascular complications.

Infections and Other Rare Events

Infections are more frequent in patients undergoing repeat ipsilateral (same site) femoral punctures or prolonged femoral sheath maintenance (within 1–5 days). Cholesterol embolism, manifesting with abdominal pain or headache (from mesenteric or central nervous system ischemia), skin mottling ("blue toes"), renal insufficiency, or lung hemorrhage, may be a clinical finding in up to 30% of high-risk patients.

Retroperitoneal Hematomas and Pseudoaneurysms

A retroperitoneal hematoma should be suspected in patients with hypotension, tachycardia, pallor, a rapidly falling hematocrit post-catheterization, lower abdominal or back pain, or neurologic changes in the leg with the puncture. This complication is associated with high femoral arterial puncture

and full anticoagulation. Pseudoaneurysm is a complication associated with low femoral arterial puncture (usually below the head of the femur).

In the past, all femoral pseudoaneurysms were routinely repaired by the vascular surgeon to avoid further neurovascular complication or rupture. With ultrasound imaging techniques these false channels can be easily identified and nonsurgical closure can be selected. Manual compression of the expansile growing mass, guided by Doppler ultrasound with or without thrombin or collagen injection, is an acceptable therapy for femoral pseudoaneurysm (Fig. 2-12).

CORONARY ARTERY ACCESS AND GUIDE CATHETERS

Guide Catheters

Compared to diagnostic catheters, the unique handling characteristics of coronary guide catheters may not be

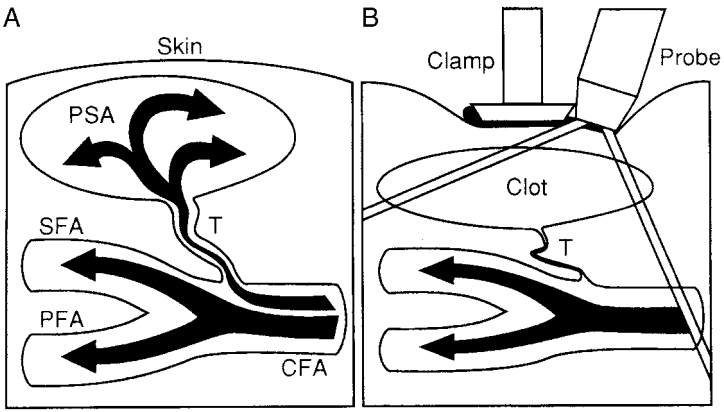


Fig. 2-12 Noninvasive technique for closure of a femoral artery pseudoaneurysm by external compression. Arrows represent the course and direction of blood flow. **A,** Blood is shown flowing from the common femoral artery (CFA) into a large pseudoaneurysm through a large tract (T). **B,** External application of pressure using a vascular clamp guided by Doppler ultrasound color flow probe results in obliteration of the tract and clot formation in the pseudoaneurysm. PFA, profunda femoris artery; SFA, superficial femoral artery. (Redrawn from Agrawal SK, Pinheiro L, Roubin GS, *et al.* Nonsurgical closure of femoral pseudoaneurysms complicating cardiac catheterization and percutaneous transluminal coronary angioplasty. *J Am Coll Cardiol* 1992;20:610–615.)

appreciated by the novice operator. Key points regarding PCI guiding catheters are listed below:

- Catheter advancement and torque should always be gentle and gradual
- If the catheter is not engaged with standard manipulations, a different size or shape should be tried early instead of forcing the catheter into the vessel
- Deep cannulation of the vessel should be avoided
- Guide catheter size should be appropriate for the diameter of the proximal vessel. For ostial disease, consider guide catheters with side holes to permit perfusion when the guide is wedged in the ostium
- Catheter size and shape are selected to be minimally traumatic while providing optimal backup support
- Most procedures can be accomplished with Judkins-type (femoral) guide catheters
- The arteries in which good backup guiding support is most difficult to achieve are, in order of difficulty, the right, circumflex, and left anterior descending coronary arteries. Saphenous vein grafts and the use of the internal mammary artery have unique problems but are less frequent occurrences.

Coronary Guiding Catheter Types

Guide catheters (Fig. 2-13) are available in a wide variety of shapes duplicating diagnostic catheter shapes and several novel curves. Tip shapes are listed in Table 2-2. Guide catheters with various modifications, including short tips, smoother curves, half-sizes, or anterior or posterior tip directions, are available. The great majority of angioplasty procedures are performed using Judkins-type catheters.

Judkins Guide Catheters. The Judkins left coronary catheter has a double curve. The length of the segment between the primary and the secondary curve determines the size of the catheter (i.e., 3.5, 4.0, 5.0, or 6.0 cm). The proper size of the left Judkins catheter depends on the length and width of the ascending aorta. In a small person with a small aorta, a 3.5 cm catheter is appropriate, while in a large person or in one with an enlarged or dilated ascending aorta (e.g., as a result of aortic stenosis, regurgitation, or Marfan syndrome), a 5.0 or 6.0 cm catheter

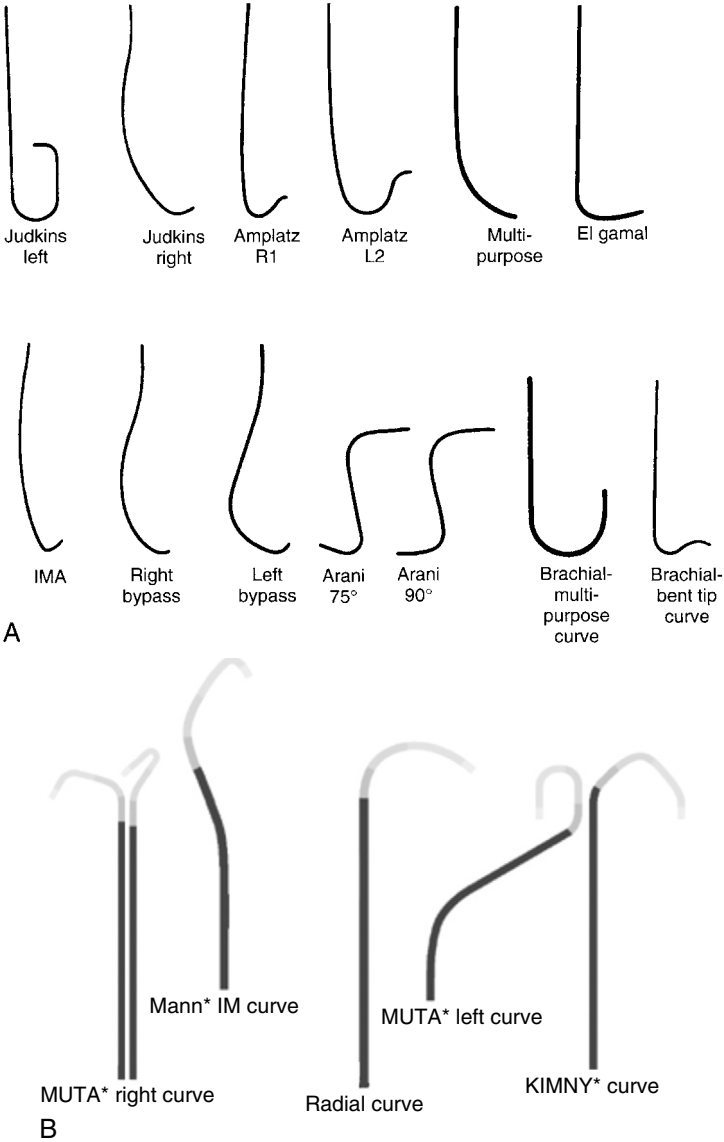


Fig. 2-13 **A, B,** Tip configurations of guiding catheters for coronary angioplasty. (**A** from Kulick DL, Rahimtoola SH, eds. *Techniques and applications in interventional cardiology*. St Louis, MO: Mosby, 1991: 60.)

Table 2-2

Types of Guide Catheter

Guide	Advantages	Disadvantages
For LAD lesions		
JL4	Routine placement	Backs out
AL2	Easy to place	Good backup, but may dissect ostium
For the RCA		
JR4	Easy use	Poor backup
Hockey-stick	Deep seating	Deep seating
Multipurpose	Deep seating	Deep seating
Arani	Excellent backup	Difficult to engage, deep seating
For highly tortuous RCAs		
—left Amplatz	Excellent backup	Difficult to engage, deep seating
For circumflex lesions		
JL4	Routine placement	Backs out
AL2	Easy to place	Good backup, but may dissect ostium
Voda	Easy to seat, excellent backup	Deep engagement
Multipurpose	Good backup	Difficult to seat

Artery	Alternative Catheters
RAC, grafts	Hockey-stick
RCA	Arani
Circumflex	Voda

may be required. The length of the Judkins curve is helpful to selectively direct the PCI wire.

A left 4 cm Judkins catheter fits in most adult patients with the catheter tip aligned with the long axis of the left main coronary trunk. A smaller (3.5 cm) catheter in the same patient will tip upward, favoring subselective left anterior descending artery (LAD) engagement. A larger (5.0 cm) catheter in the same patient will tip downward and favors subselective circumflex cannulation. A slight counterclockwise rotation of the catheter may be necessary to improve alignment of the catheter tip with the left main trunk. When the coronary orifice is not cannulated appropriately, the catheter should be replaced with a better-fitting one rather than manipulated into the coronary artery.

Amplatz Guide Catheters. The left Amplatz-type catheter is a preshaped half-circle with the tip extending perpendicular to the curve. Amplatz catheter sizes (left 1, 2, and 3; and right 1 and 2) indicate the diameter of the tip curve. In most normal-sized adults, AL2 and AR1 right (modified) Amplatz catheters give satisfactory results.

Special attention should be given to using Amplatz catheters. In the LAO projection, the tip is advanced into the left aortic cusp. Further advancement of the catheter causes the tip to move upward into the left main trunk. It is necessary to push and torque the Amplatz catheters slightly to disengage the catheter tip by backing it upward and out of the left main ostium. If the catheter is pulled instead of first being advanced, the tip moves downward and into the left main or circumflex artery. Unwanted deep cannulation of the circumflex might tear this branch or the left main trunk.

Amplatz catheters have a higher incidence of coronary dissection than Judkins-type catheters. However, Amplatz catheters often provide superior backup support.

The right Amplatz (modified) catheter has a smaller but similar hook-shaped curve. The catheter is advanced into the right coronary cusp, as with a Judkins right catheter. The catheter is rotated clockwise 45–90°. The same maneuver is repeated at different levels until the right coronary artery is entered. After coronary injections, the catheter may be pulled, advanced, or rotated out of the coronary artery. Amplatz catheters are often used for the arm approach.

Saphenous Vein and Internal Mammary Artery Graft Catheters. There are right and left graft and internal mammary catheters with shapes similar to their diagnostic counterparts. As in diagnostic angiography, they also may be useful in cannulation of native vessels with unusual origins or proximal courses.

The right coronary vein graft catheter is similar to a right Judkins catheter with a more downward-pointing primary curve, allowing cannulation of a vertically oriented coronary artery vein graft.

The left vein graft catheter is similar to the right Judkins catheter with a smaller and more upward-pointing secondary curve, allowing easy cannulation of saphenous vein grafts supplying the left anterior descending and left circumflex territories.

Such grafts are usually placed higher and more anterior than right coronary grafts and with a relatively horizontal or upward takeoff from the aorta.

The internal mammary artery graft catheter has a short, hook-shaped tip that helps engage the ostium of internal mammary artery grafts. This catheter shape is especially helpful in patients with very vertical origin of the internal mammary artery at the juncture of the subclavian and common carotid arteries.

Guide Catheters for the Arm Approach. Guide catheters from the radial artery approach are the same as those used for the femoral approach, but most operators prefer Judkins, Amplatz, Q-shaped, multipurpose, or special designs. Multipurpose guide catheter shapes are similar to multipurpose diagnostic catheters but are more difficult to manipulate. These catheters give excellent support in special situations such as vertically oriented right coronary artery grafts. The Amplatz catheter can be used effectively from either the right or left arm. This catheter is manipulated in a fashion similar to that described for the Amplatz catheter.

Specially curved catheters have been designed by various interventionalists to provide increased backup support from either the arm or leg approach. These catheters include Arani (75° or 90°) catheters for right coronary arteries (Fig. 2-13), or Voda shape for right or left circumflex arteries. Voda or other Q or G shapes should be used with caution, since deep cannulation of the vessel commonly occurs (Fig. 2-14). The relatively sharp angles of some guide catheter shapes may create difficulty in advancing large interventional equipment or nonballoon catheter devices. For example, a hockey stick catheter has a 90° tip angle, which may be useful in engaging bypass grafts or right coronary arteries but may limit passage of a long stent or rotablator.

Special Features of Guide Catheters.

Small-Bore (≤6F) Guide Catheters. Coronary angioplasty can be performed using large-lumen guide catheters 6 French or smaller in size. Use of diagnostic and guide catheters of 5 French or less has also been reported. Small-bore catheters

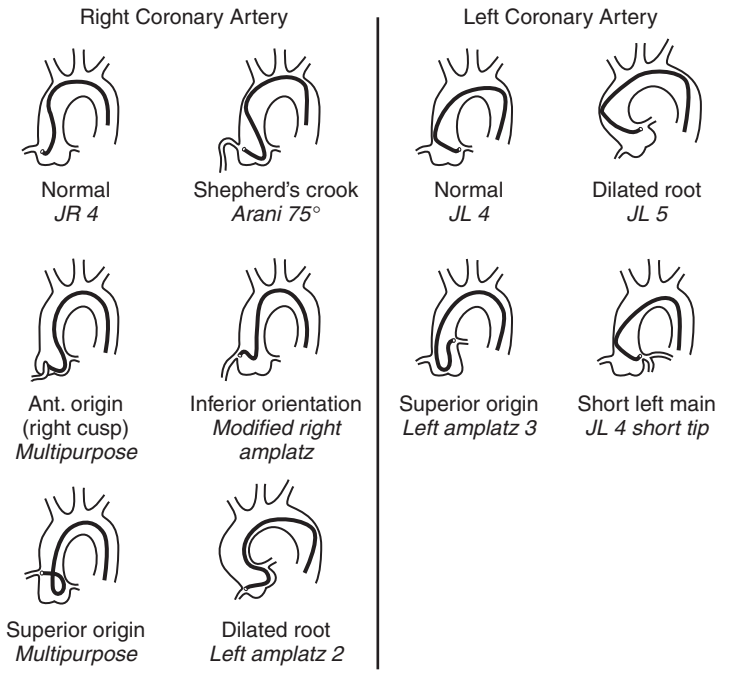


Fig. 2-14 Guide catheter selection based on anatomic variations in aortic root width and coronary artery orientation. Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*, Birmingham, MI: Physicians' Press, 1999.

are associated with less femoral bleeding and allow early patient ambulation. They can be easily used from the radial approach. When extraordinary backup is needed, deep cannulation of the vessel can be accomplished easily, and possibly with less trauma than with larger (>7F) guiding catheters.

Guide Catheters with Side Holes. In general, most guide catheters should be without side holes so that one can detect the pressure damping that suggests ostial disease. Side hole catheters are very useful for PCI in small right coronary or graft vessels when pressure damping cannot be overcome by catheter repositioning. When using side hole guides, measurement of the translesional pressure (see fractional flow reserve, FFR) must be carefully reviewed. Undetected proximal pressure

gradients due to the side holes may occur, complicating distal gradient evaluations.

CANNULATING DIFFICULT CORONARY OR GRAFT OSTIA

Left Coronary Artery

Problems.

Short Left Main, Separate Ostial Left Anterior Descending, and Circumflex Arteries. For the LAD, use a left Judkins catheter that is one size smaller than that usually selected (i.e., 3.5 cm instead of 4.0 cm; Figs. 2-15, 2-16). Selective cannulation of the LAD in patients with a short left main artery may be needed. For the circumflex ostium (Fig. 2-16), withdraw the standard 4 cm left Judkins catheter and rotate it counterclockwise. Alternatively, using a left Judkins catheter that is one size larger is helpful. An Amplatz-type catheter is especially useful for cannulating the circumflex ostium separately, but it must be used with care to avoid dissection.

High Left Coronary Artery Takeoff. An unusually high origin of the left main coronary artery from the aorta usually can be cannulated using an Amplatz-type catheter.

Wide Aortic Root. In patients with a relatively horizontal or wide aortic root with upward takeoff of the left main coronary artery, use a large-curve left Judkins (5 or 6 cm), an Amplatz-type left coronary catheter, or a Voda-shaped catheter.

Posterior Origin of Left Main. Slight counterclockwise rotation and advancement of the left Judkins catheter may bring the tip to the left main. Sometimes it may be necessary to use a posterior out-of-plane tip. Another option is a left Amplatz catheter.

Right Coronary Artery

The origin of the right coronary artery shows more variation than the left coronary artery. Extra backup support is difficult to obtain with standard JR4-type catheters. Directing the catheter tip to the right in the usual fashion using the lateral

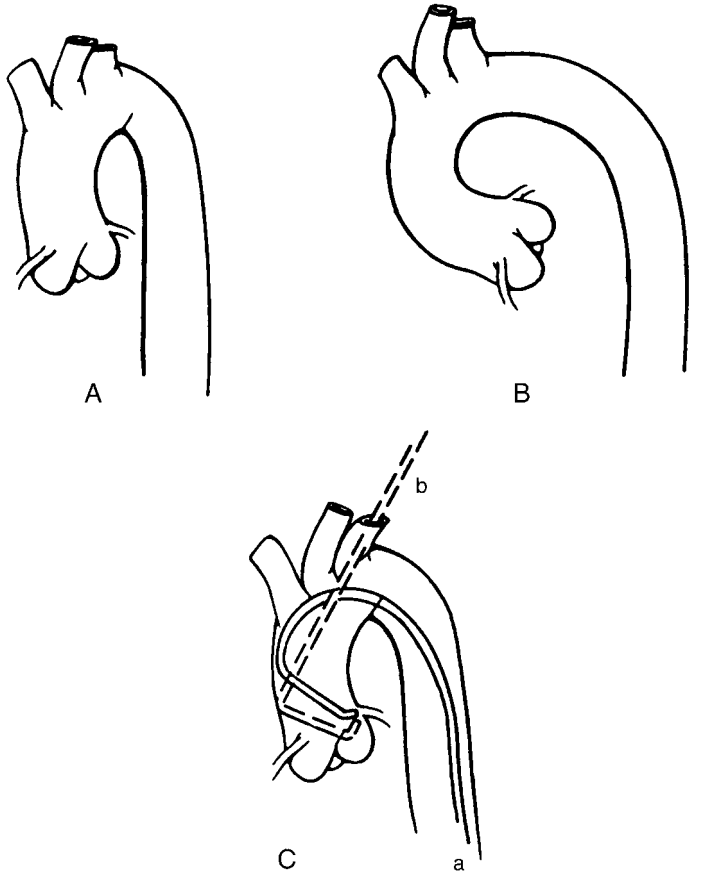


Fig. 2-15 Anatomic variation of aortic arch, root, and valve plane. **A,** Normotensive. **B,** Hypertensive. **C,** Changes in secondary curves of left Judkins catheter when inserted via the femoral approach (a) or from the left radial or brachial approach (b). The right brachial approach can also be used. (From Topol EJ. *Textbook of interventional cardiology*, 2nd ed. Philadelphia, PA: WB Saunders, 1994: 553.)

view permits easy cannulation of the slightly anterior origin of the right coronary artery in the right cusp.

Problems.

High and Upward Takeoff of a Right Coronary Artery. A relatively high origin of the right coronary artery may require a left or right (modified) Amplatz-type catheter (Fig. 2-14).

Fig. 2-16 Left anterior descending artery guide catheter positioning with different secondary curve sizes. (From Jang GD. *Angioplasty*. New York: McGraw-Hill, 1987: 303.)

Wide Aortic Root. In a patient with a horizontal and wide aortic root, cannulation of the right coronary orifice may require an Amplatz or hockey-stick catheter.

"Shepherd's Crook" Right Coronary Artery. In this situation, a right Judkins catheter provides poor support. A left Amplatz (0.75 to 1), hockey-stick, or Arani catheter may provide better support, especially for relatively distal lesions. However, deep cannulation of the vessel is frequent with these catheters, and proximal vessel trauma can occur, especially in a small aortic root.

Anomalous Coronary Artery Origin

The most frequent anomaly is a circumflex origin from a proximal right coronary artery, or a separate orifice just posterior to the right coronary artery orifice. When there is a common trunk, a right Judkins catheter may be sufficient. A separate left circumflex orifice can be entered by rotating the right Judkins more posteriorly. Because of the downward course of the proximal circumflex, better engagement and support may be obtained by a right bypass, Amplatz, or multipurpose guide catheter.

Extreme anterior or left coronary cusp origin of the right coronary artery can be engaged by using a left Amplatz catheter. A more leftward origin of an anomalous right coronary artery (which also tends to be higher) can be entered using a left bypass guide.

Saphenous Vein Bypass Grafts (Figs 2-17, 2-18)

To decrease the manipulation time and select the best catheter shape, the diagnostic angiogram should be reviewed

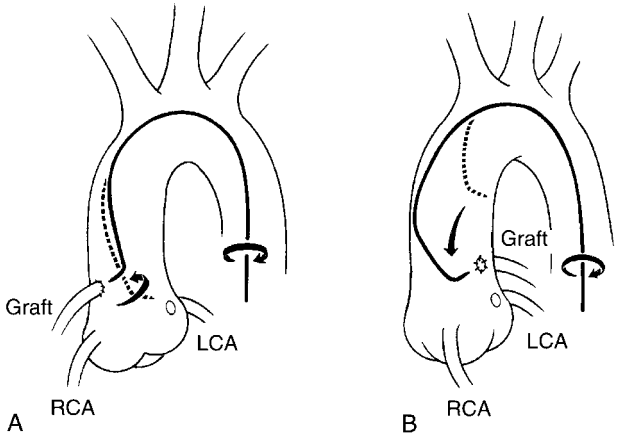
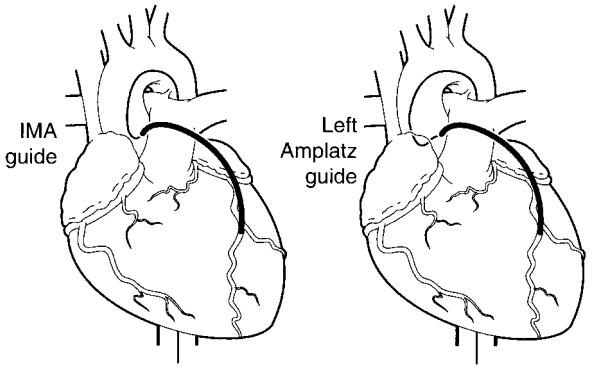


Fig. 2-17 Method of use for Judkins right catheter in cannulating saphenous venous bypass graft conduits. (From Tilkian AG, Daily EK. *Cardiovascular procedures: diagnostic techniques and therapeutic procedures*. St Louis, MO: Mosby, 1986.)

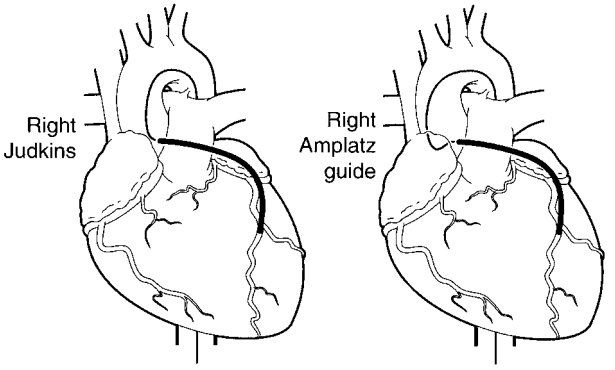
carefully for the location of the aortic anastomosis and proximal course of the vessel. Anatomic landmarks should be noted. Because of the potential risk of embolization, avoid unnecessary manipulation of catheters inside the ostium, especially in old grafts that may contain atherosclerotic material.

Right Coronary Bypass Vein Graft Catheterization (Fig. 2-18). The right coronary vein graft usually can be entered using a 4 cm right Judkins-type catheter. The right coronary catheter is placed in the ascending aorta at a level slightly higher than the expected level of the right coronary vein graft orifice, and the catheter is rotated clockwise from 45° to 90°. This will cause the catheter tip to move along the border of the ascending aortic silhouette in the left anterior oblique position. When the right graft is anastomosed to the far right side of the aorta, a counterclockwise, rather than clockwise, rotation of the catheter may be necessary. In some cases the right Judkins or right bypass catheter may fall short of the ostium, in which case an Amplatz or multipurpose catheter may work. In this situation, the catheter tip is pointed toward

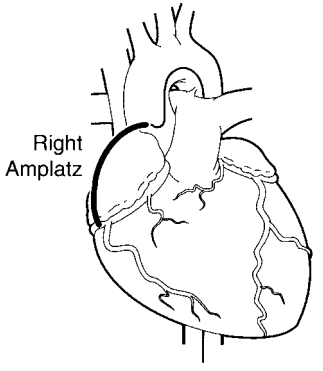
Superior directed vein graft



Transverse directed vein graft



Inferior directed vein graft



the left-hand side of the screen in the AP or RAO position. Advancement or withdrawal while rotating the catheter tip might be necessary for graft engagement. In the case of right vein graft vertical takeoff, the right coronary Judkins catheter tip may be directed upward rather than downward into the lumen, making adequate opacification of the vein graft difficult. In this case, a right coronary bypass vein graft catheter should be used. Because of the downward primary curve, the right vein graft catheter tip usually aligns more parallel to the axis of the graft. Be careful, as the catheter has a tendency to move deeply down into the vein graft. A right modified Amplatz catheter can also be used for horizontal or vertical takeoff vein grafts.

Left Anterior Descending Vein Graft Catheterization. The right Judkins catheter is placed at a level slightly higher than the expected level of the orifice of the anterior descending vein graft, and 30–45° clockwise rotation is applied. The catheter tip will appear foreshortened in the LAO view and will be pointing toward the right-hand side of the ascending aorta silhouette in the right anterior oblique (RAO) view. In some patients, it may be necessary to use a left coronary vein graft catheter or left Amplatz catheter. A slight clockwise rotation of the catheter at the level of the expected aortic anastomosis site will often engage the ostium.

The left anterior descending graft may course horizontally or downward after the origin. In some cases, however, it makes an upward curve before it turns toward the apex. In these cases, the need for stronger backup support may require the use of a left Amplatz catheter or deep cannulation using a hockey stick shaped catheter.

Circumflex Vein Graft Catheterization. Repeating the same maneuvers described for left anterior descending vein graft

Fig. 2-18 Saphenous vein graft ostium orientations. Different guide catheters should be selected based on the angle of graft takeoff, superior, transverse, or inferior orientation. (From Pinkerton CA, Slack JD, Orr CM, Vantassel JW, Smith ML. Percutaneous transluminal coronary angioplasty in patients with prior myocardial revascularization surgery, *Am J Cardiol* 1988;61:15G–22G.)

cannulation using right Judkins or left vein graft catheters will usually produce a successful result.

INTERNAL MAMMARY ARTERY GRAFT CANNULATION

Left Internal Mammary Artery

The left internal mammary artery originates anteriorly from the caudal wall of the subclavian artery and is distal to the vertebral artery origin. There are many variations in the shape of the aortic arch and origin and direction of the subclavian artery (Fig. 2-19). The left subclavian artery can be entered using an internal mammary artery catheter. The catheter is advanced into the aortic arch up to the level of the origin of the left subclavian artery (Fig. 2-20). The guidewire is left in the catheter. Subsequently, the catheter is withdrawn slowly and rotated counterclockwise. The catheter tip is deflected cranially, usually engaging the left subclavian artery at the top of the aortic knob in the anteroposterior projection. The guidewire is advanced into the subclavian artery. The catheter is advanced. The guidewire is withdrawn. More than one attempt is often necessary to engage into the subclavian artery. Once the subclavian artery is engaged, the catheter is advanced slightly over a guidewire beyond the internal mammary orifice. A J-tipped or a Wholey wire is helpful to guide the catheter into the subclavian artery. Once the catheter has been advanced beyond the takeoff of the internal mammary artery, the catheter is withdrawn slowly and small contrast injections are given to visualize the internal mammary artery orifice. The catheter tip should be directed caudally and anteriorly. At the level of the orifice of the internal mammary, a slight counterclockwise rotation and advancement may be necessary to cannulate the artery.

Vigorous manipulation of the catheter and deep intubation of the internal mammary artery should be avoided because of the hazard of dissection. Initially, as with most cannulations, only catheters without side holes should be used. Pressure damping indicates potentially dangerous deep cannulation. During injection of contrast medium, the patient should be reminded to expect discomfort in the shoulder and anterior chest wall.

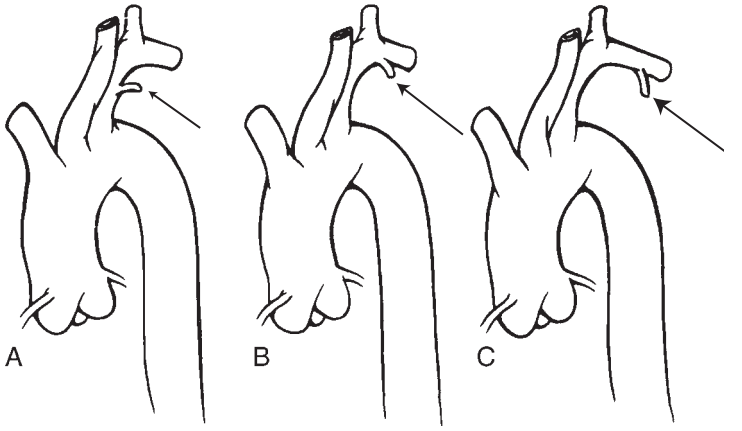


Fig. 2-19 Anatomic variation of the origin of the internal mammary artery from the subclavian artery: **A,** proximal; **B,** mid; **C,** distal. Although the femoral approach is easier for anatomy A, the ipsilateral brachial approach is more difficult than might appear initially. (From Topol EJ. *Textbook of interventional cardiology*, 2nd ed. Philadelphia, PA: WB Saunders, 1994: 560.)

Right Internal Mammary Artery

Right internal mammary artery cannulation is more difficult than left internal mammary artery cannulation. The right brachiocephalic truncus is entered using a right Judkins catheter by rotating the tip with a counterclockwise rotation at the level of the brachiocephalic truncus (Fig. 2-20). The catheter is advanced into the subclavian artery over a guidewire. The rest of the cannulation procedure is similar to that described for left internal mammary artery graft cannulation.

In patients for whom cannulation of the subclavian artery is not possible because of excessive tortuosity or obstructive lesions, an internal mammary artery catheter can be introduced through an arm (ipsilateral) artery and advanced beyond the mammary artery orifice over a guidewire. The catheter is withdrawn slowly by making frequent, small contrast injections, then seated in the usual fashion for PCI.

Table 2-2 shows types of guide and their advantages and disadvantages.

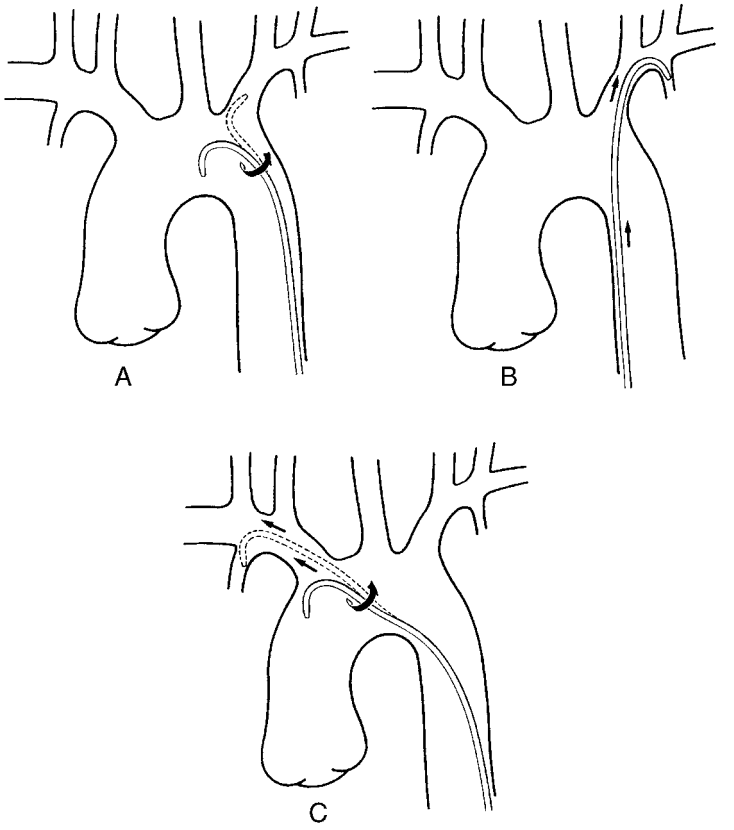


Fig. 2-20 A–C, Technique of catheterization of the internal mammary artery (IMA). Clockwise rotation is employed for both left and right IMA engagement. (From Tilkian AG, Daily EK. *Cardiovascular procedures: diagnostic techniques and therapeutic procedures*. St Louis, MO: Mosby, 1986.)

ANGIOGRAPHIC CLUES FOR GUIDE CATHETER SELECTION

Guide catheters differ from diagnostic catheters in two critical areas: a less open secondary curve tip and a potentially shorter, non-tapered ostial portion of the catheter. Although the majority of left coronary arteries are effectively cannulated with classic standard Judkins left 4 cm curves, guiding catheters may require the shorter Judkins left 3.5 cm or other configurations to engage adequately. A dilated aortic root may thus be

satisfactorily engaged with the Judkins left 4 cm and, on rare occasions, the Judkins left 5 cm guiding catheter.

In the right coronary artery, the upward-angled high-takeoff or shepherd's crook configuration represents a particularly difficult problem for angioplasty guide seating. For engagement of upgoing right coronary artery takeoffs, an Amplatz left coronary artery and Arani or hockey stick have been recommended. Although the internal mammary artery guide has been used, its support against the aortic cusp and posterior aorta is less than that provided by the catheters mentioned above.

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3

ANGIOGRAPHY FOR PERCUTANEOUS CORONARY INTERVENTIONS

Souheil Khoukaz, Steven C. Hermann, and Morton J. Kern

In the years before percutaneous coronary intervention (PCI) was developed, the definition of coronary artery narrowings sufficient for surgical revascularization required only the identification of disease location without detailed characterization of plaque, associated branch points, or lesion morphology. Because of tailored interventional techniques, the precise definition of lesion length and morphology, as well as relationship to side branches, makes angiography even more critical. The interventionist needs to assess several specific aspects of the vessels and lesions under study.

OBJECTIVES FOR PCI ANGIOGRAPHY

- Identify relationship of coronary ostium to aorta for guide catheter selection
- Identify target vessel, pathway, and angle of entry
- Identify lesion length and morphology using angulated views eliminating vessel overlap
- Separate associated side branches and degree of atherosclerosis in branch ostia
- Verify distal distribution of target vessel and collateral supply
- Determine the diameter of the coronary artery at the target site.

Optimal definition of proximal coronary anatomy is critical to guide and balloon catheter selection. Assessment of calcium from angiography is known to be less reliable than intravascular echocardiography, but still serves a useful purpose in assessing risks associated with the procedure. Classical terminology for defining angiographic projections with regard

to left and right anterior oblique, cranial and caudal angulation, and lateral projections remains as defined in previous discussions for diagnostic coronary angiography (see *The Cardiac Catheterization Handbook*, 4th ed, 2003, Chapter 4).

Visualization of vessel bifurcations, origin of side branches, the portion of the vessel proximal to a significant lesion, and previously "unimportant" lesion characteristics (length, eccentricity, calcium, and the like) will differentiate device selection and potential risk. In the case of a total vessel occlusion, the distal vessel should be visualized as clearly as possible by injecting the coronary arteries that supply collaterals and taking cineangiograms with panning long enough to visualize late collateral vessel filling and the length of the occluded segment.

An optimal angiographic image is also critical to a successful intervention. Excellent image quality enhances the accurate interpretation of results. Modification of catheterization technique to reduce motion artifact during imaging, optimal use of beam restrictors (collimation) to reduce scatter, improved image contrast, and conscientious data storage methods can enhance clinical results.

A working knowledge of the principles of radiographic imaging permits the interventionalist to improve the approach to both diagnostic and therapeutic procedures. Continued awareness of the inverse square law of radiation propagation will reduce the exposure to operators and their team. Obtaining quality images should not necessitate increasing the ordinary procedural radiation exposure to either the patient or catheterization personnel.

COMMON ANGIOGRAPHIC VIEWS FOR ANGIOPLASTY

The routine coronary angiographic views described below should include those that best visualize the origin and course of the major vessels and their branches in at least two different projections (preferably orthogonal). Naturally, there is a wide variation in coronary anatomy, and appropriately modified views will need to be individualized.

The nomenclature for angiographic views is described in *The Cardiac Catheterization Handbook* but will be reviewed briefly here, emphasizing the interventionalist's thinking.

Position for Anteroposterior Imaging

The image intensifier is directly over the patient, with the beam perpendicular to the patient lying flat on the x-ray table (Figs 3-1, 3-2). The anteroposterior (AP) view or shallow right anterior oblique (RAO) displays the left main coronary artery in its entire perpendicular length. In this view, the branches of the left anterior descending (LAD) and left circumflex coronary arteries branches overlap. Slight RAO or left anterior oblique (LAO) angulation may be necessary to clear the density of the vertebrae and the catheter shaft in the thoracic descending aorta. In patients with acute coronary syndromes this view will exclude left main stenosis, which can preclude or complicate PCI. The AP cranial view is excellent for visualizing the LAD with septals moving to the left (on screen) and diagonals to the right, helping wire placement.

Position for Right Anterior Oblique Imaging

The image intensifier is to the right side of the patient. The RAO caudal view shows the left main coronary artery bifurcation with the origin and course of the circumflex/obtuse marginals, intermediate branch, and proximal left anterior descending segment well seen. The RAO, caudal view is one of the best two views for visualization of the circumflex artery. The LAD beyond the proximal segment is often obscured by overlapped diagonals.

The RAO or AP cranial view is used to open the diagonals along the mid and distal LAD. Diagonal branch bifurcations are well visualized. The diagonal branches are projected upward. The proximal LAD and circumflex usually are overlapped. Marginals may overlap, and the circumflex is foreshortened.

For the right coronary artery (RCA), the RAO view shows the mid RCA and the length of the posterior descending artery and posterolateral branches. Septals supplying an occluded LAD via collaterals may be clearly identified. The posterolateral branches overlap and may need the addition of the cranial view.

Position for Left Anterior Oblique Imaging

In the LAO position, the image intensifier is to the left side of the patient. The LAO/cranial view also shows the left main

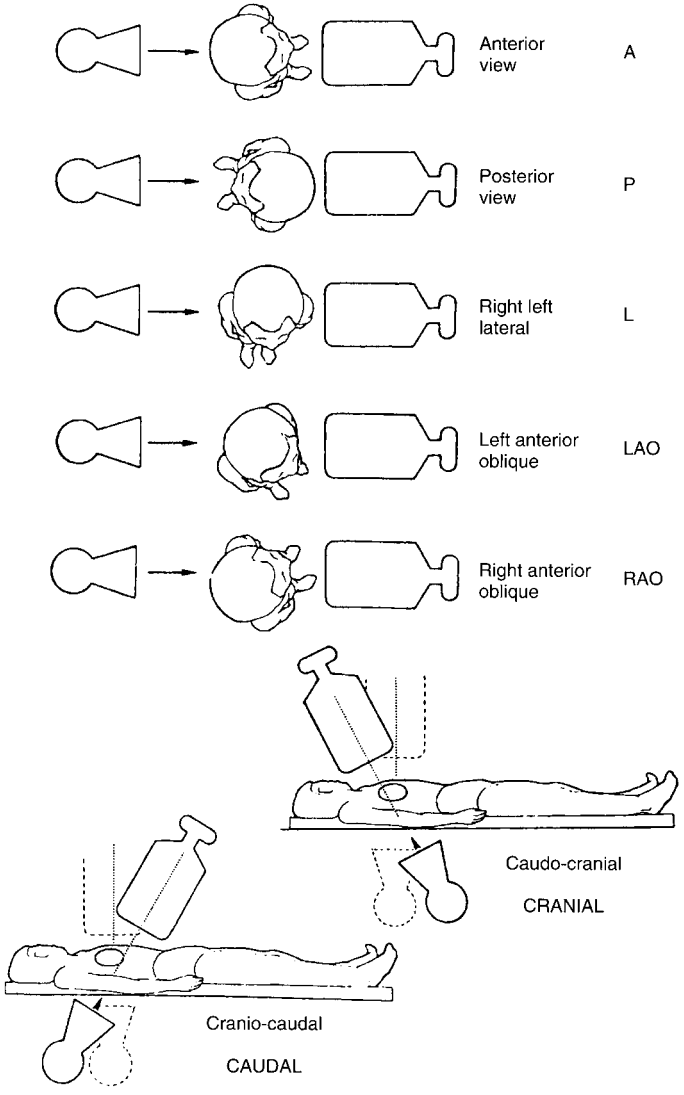

Fig. 3-1 Nomenclature for angiographic views. (Modified from Paulin S. Terminology for radiographic projections in cardiac angiography. *Cathet Cardiovasc Diagn* 1981;7:341.)

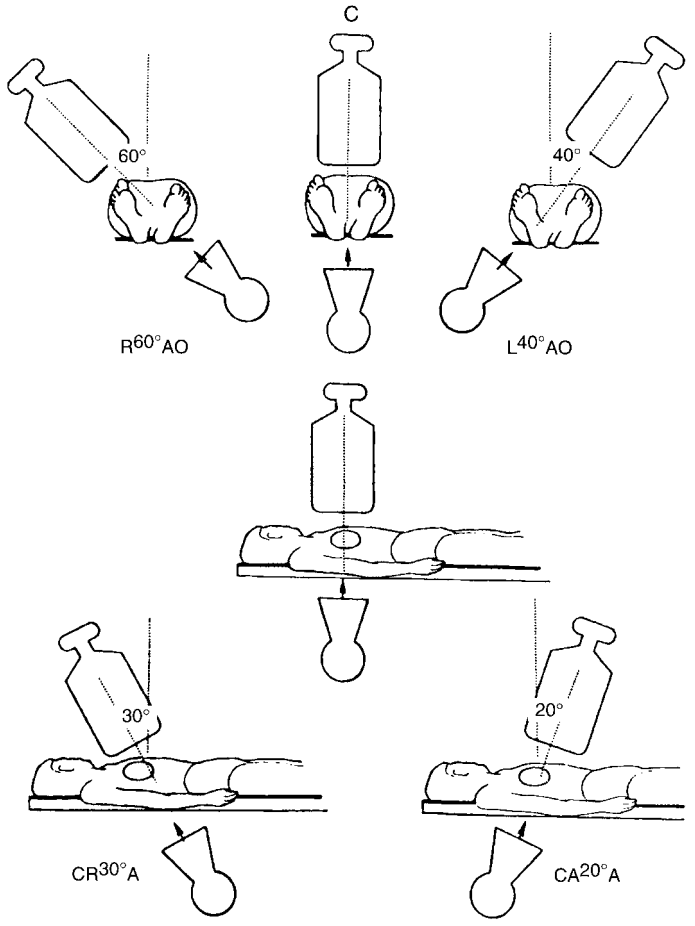

Fig. 3-2 Nomenclature for angiographic views. (Modified from Paulin S. Terminology for radiographic projections in cardiac angiography. *Cathet Cardiovasc Diagn* 1981;7:341.)

coronary artery (slightly foreshortened), LAD, and diagonal branches. Septal and diagonal branches are separated clearly. The circumflex and marginals are foreshortened and overlapped. Deep inspiration will move the density of the diaphragm out of the field. The LAO angle should be set so that the course of the LAD is parallel to the spine and stays in

the "lucent wedge" bordered by the spine and the curve of the diaphragm. Cranial angulation tilts the left main coronary artery down and permits view of the LAD/circumflex bifurcation (Fig. 3-3). Too steep a LAO/cranial angulation or shallow inspiration produces considerable overlapping with the diaphragm and liver, degrading the image.

For the RCA, the LAO/cranial view shows the origin of the artery, its entire length, and the posterior descending artery bifurcation (crux). Cranial angulation tilts the posterior

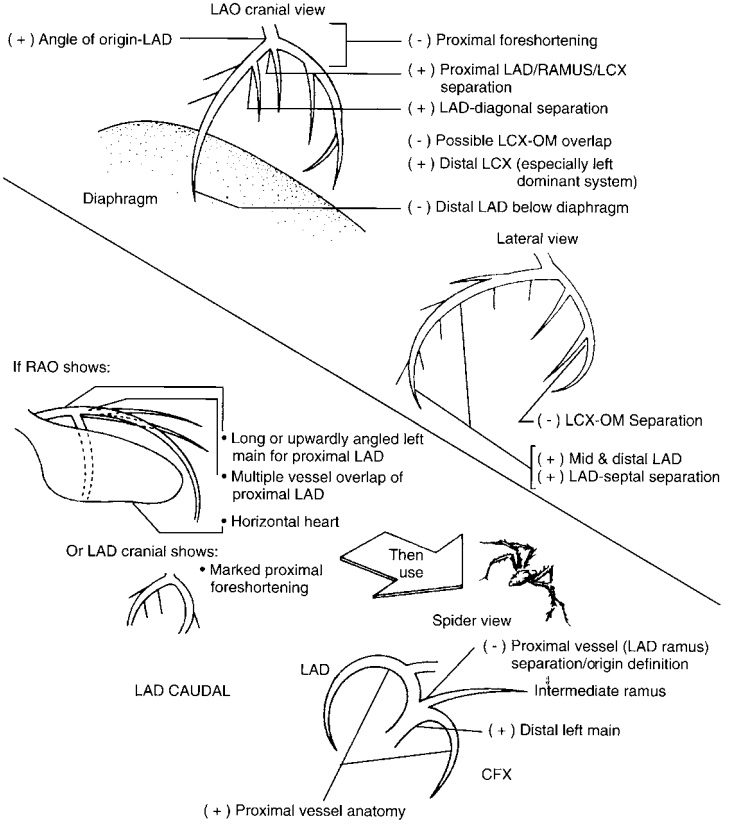

Fig. 3-3 Diagrammatic view of left coronary artery demonstrating special positioning to best observe branch segments. (From Boucher RA, Myler RK, Clark DA, Stertzer SH. Coronary angiography and angioplasty. *Cathet Cardiovasc Diagn* 1988;14:269–285.)

descending artery down to show vessel contour and reduces foreshortening. Deep inspiration clears the diaphragm. The posterior descending artery and posterolateral branches are foreshortened.

The LAO/caudal view ("spider" view; Fig. 3-3) shows a foreshortened left main coronary artery and the bifurcation of the circumflex and LAD. Proximal and midportions of the circumflex and the origins of obtuse marginal branches are usually seen excellently. Poor image quality may be due to overlapping of diaphragm and spine. The LAD is considerably foreshortened in this view.

A left lateral view shows the mid and distal LAD best. The LAD and circumflex are well separated. Diagonals usually overlap. The course of the (ramus) intermediate branch is well visualized. This view is best to see coronary artery bypass graft (CABG) conduit anastomosis to the LAD.

For the RCA, the lateral view also shows the origin (especially in those with more anteriorly oriented orifices) and the mid RCA well. The posterior descending artery and posterolateral branches are foreshortened.

Angulation for Saphenous Bypass Grafts

Coronary artery saphenous vein grafts are visualized in at least two views (LAO and RAO). It is important to show the aortic anastomosis, the body of the graft, and the distal anastomosis. The distal runoff and continued flow or collateral channels are also critical. The graft vessel anastomosis is best seen in the view that depicts the native vessel best. A general strategy for graft angiography is to perform the standard views while assessing the vessel key views for specific coronary artery segments (Table 3-1) to determine the need for contingency views or an alteration/addition of special views. Therefore, the graft views can be summarized as follows:

- **RCA graft:** LAO cranial/RAO, and lateral
- **LAD graft (or internal mammary artery):** lateral, RAO cranial, LAO cranial, and AP (the lateral view is especially useful to visualize the anastomosis to the LAD)
- **Circumflex (and obtuse marginals) grafts:** LAO and RAO caudal.

Table 3-1

Recommended "key" angiographic view for specific coronary artery segments

Coronary Segment	Origin/Bifurcation	Course/Body
Left main	AP	AP
	LAO cranial	LAO cranial
	LAO caudal*	
Proximal LAD	LAO cranial	LAO cranial
	RAO caudal	RAO caudal
Mid LAD	LAD cranial	
	RAO cranial	
	Lateral	
Distal LAD	AP	
	RAO cranial	
	Lateral	
Diagonal	LAO cranial	RAO cranial, caudal, or straight
	RAO cranial	
Proximal circumflex	RAO caudal	LAO caudal
	LAO caudal	
Intermediate	RAO caudal	RAO caudal
	LAO caudal	Lateral
Obtuse marginal	RAO caudal	RAO caudal
	LAO caudal	
	RAO cranial	
	(distal marginals)	
Proximal RCA	LAO	
	Lateral	
Mid RCA	LAO	LAO
	Lateral	Lateral
	RAO	RAO
Distal RCA	LAO cranial	LAO cranial
	Lateral	Lateral
PDA	LAO cranial	RAO
Posterolateral	LAO cranial	RAO cranial
	RAO cranial	RAO cranial

* Horizontal hearts.

AP, Anteroposterior; LAD, left anterior descending artery; LAO left anterior oblique; PDA, posterior descending artery (from RCA); RAO, right anterior oblique; RCA, right coronary artery. From Kern MJ, ed. *The cardiac catheterization handbook*, St Louis, MO, Mosby, 1995: 286.

TECHNIQUES FOR CORONARY ARTERIOGRAPHY

Imaging During Respiration

Since deep inspiration may change the proximal course of the artery and the spatial relation of the lesion to anatomic landmarks, guide angiograms should be taken in such a way that frequent inspiratory effort leading to patient fatigue during manipulation is not necessary. Select a view requiring minimal inspiratory hold while providing optimum presentation of the lesion.

Power Injection Versus Hand Injection for Coronary Arteriography

Power injection of the coronary arteries has been used in thousands of cases in many laboratories and is equal in safety to hand injection. A power injector at a fixed setting may require several injections to find the optimal contrast delivery flow rate. Power injectors now incorporate hand controls, permitting precise operator touch-sensitive variable volume injection (Acist, Bracco Diagnostics) as well as a computer touch screen for precise contrast delivery settings. Typical settings for power injections are:

- Right coronary artery: 6 mL at 2–3 mL/sec; maximum pressure 450 psi
- Left coronary artery: 10 mL at 4–6 10 mL/sec; maximum pressure 450 psi.

Panning Techniques. Many laboratories use x-ray image mode sizes of <7 inch diameter, which precludes having the entire coronary artery course visualized without panning over the heart to include late filling of the distal arterial or collateralized segments. In addition, in most views some degree of panning will be necessary to identify regions that are not seen from the initial setup positioning. Some branches may unexpectedly appear later from collateral filling or other unusual anatomic sources.

Angiographic TIMI Classification of Blood Flow

Thrombolysis in myocardial infarction (TIMI) flow grading has been used to assess, in a qualitative fashion, the degree of restored perfusion achieved after thrombolysis or angioplasty

in patients with acute myocardial infarction. Table 3-2 provides descriptions used to assign TIMI flow grades.

Classification of Distal Angiographic Contrast Runoff. The distal runoff is classified into four stages (also known as TIMI grade):

- Normal distal runoff (TIMI 3)
- Good distal runoff (TIMI 2)
- Poor distal runoff (TIMI 1)
- Absence of distal runoff (TIMI 0).

Contrast run off is now performed quantitatively by using cine frame counts from the first frame of the filled catheter tip to the frame where contrast is seen filling a predetermined distal arterial end point.

TIMI Frame Count. Myocardial blood flow has been assessed angiographically using the TIMI score for qualitative grading of coronary flow. TIMI flow grades 0–3 have become a standard

Table 3-2

description of coronary blood flow in clinical trials. TIMI grade 3 flows have been associated with improved clinical outcomes.

The method uses cineangiography with 6 French catheters and filming at 30 frames per second. The number of cine frames from the introduction of dye in the coronary artery to a predetermined distal landmark is counted. The TIMI frame count for each major vessel is thus standardized according to specific distal landmarks. The first frame used for TIMI frame counting is that in which the dye fully opacifies the origin of the artery and in which the dye extends across the width of the artery touching both borders with antegrade motion of the dye. The last frame counted is when dye enters the first distal landmark branch. Full opacification of the distal branch segment is not required. Distal landmarks used commonly in analysis are:

- For the LAD, the distal bifurcation of the LAD artery
- For the circumflex system, the distal bifurcation of the branch segments with the longest total distance
- For the RCA, the first branch of the posterolateral artery.

Typically a normal contrast frame count reflecting normal flow is 24 ± 10 frames.

The TIMI frame count (TFC) can further be corrected for the length of the LAD. The TFC in the LAD requires normalization or correction for comparison to the two other major arteries. This is called corrected TIMI frame count (CTFC). The average LAD is 14.7 cm long, the right 9.8 cm, and the circumflex 9.3 cm, according to Gibson *et al.* CTFC accounts for the distance the dye has to travel in the LAD relative to the other arteries. CTFC divides the absolute frame count in the LAD by 1.7 to standardize the distance of dye travel in all three arteries. Normal TIMI frame count for the LAD is 36 ± 3, and CTFC 21 ± 2; for the circumflex artery TFC = 22 ± 4; for the RCA TFC = 20 ± 3. TIMI flow grades do not correspond to measured Doppler flow velocity or CTFC. High TFC may be associated with microvascular dysfunction despite an open artery. A CTFC of less than 20 frames was associated with low risk for adverse events in patients following myocardial infarction. A contrast injection rate increase of more than 1 mL/sec by hand injection can decrease the TFC by two frames. The TFC method provides valuable information relative to clinical response after coronary intervention.

Angiographic Classification of Collateral Flow

Collateral flow can be seen and classified angiographically. The late opacification of a totally or subtotally (99%) occluded vessel through antegrade or retrograde channels will assist in correct guidewire placement, lesion localization, and a successful procedure. The collateral circulation is graded angiographically as follows:

- Grade 0: No collateral branches seen
- Grade 1: Very weak (ghostlike) opacification
- Grade 2: Opacified segment is less dense than the source vessel and filling slowly
- Grade 3: Opacified segment is as dense as the source vessel and filling rapidly.

Collateral visualization will help establish the size of the recipient vessel for the purposes of selecting an appropriately sized balloon. Determining whether the collateral circulation is ipsilateral (e.g., proximal RCA to distal RCA collateral supply) or contralateral (e.g., circumflex to distal RCA collateral supply) and exactly which region will be affected should collateral supply be disrupted is important in order to be able to gauge procedural risk. The evaluation of collaterals must be included when making decisions on which vessels should be protected or lost during coronary angioplasty.

Assessment of Coronary Stenoses

The evaluation of the degree of a stenosis relates to the percentage reduction in the diameter of the vessel. This is calculated in the projection where the greatest narrowing can be observed. Exact evaluation is almost impossible and, in fact, the lesions are roughly classified. Six categories can be distinguished in this way:

- 0 = normal coronary artery
- 1 = irregularities of the vessel
- 2 = narrowing of less than 50%
- 3 = stenosis between 50% and 75%
- 4 = stenosis between 75% and 95%
- 5 = total occlusion

Quantitative Coronary Angiography. The degree of coronary stenosis is quantitated from the cineangiogram and, in clinical

practice, is usually a visual estimation of the percentage of diameter narrowing using the presumed proximal normal arterial segment and the ratio of the normal diameter to the stenosis diameter. This technique is widely applicable in clinical practice but is inadequate for the quantitative methodology done in most research studies. The intraobserver variability may range between 40% and 80% and there is frequently as wide as a 20% range on interobserver differences. Quantitative methodology uses digital calipers or automated or manual edge detection systems. Densitometric analysis with digital angiography also provides quantitative lesion measurements.

Coronary Lesion Descriptions for Angioplasty. In 1988, the American College of Cardiology and the American Heart Association Task Force characterized coronary lesions by specific characteristics, classifying lesions as type A, B, or C (Table 3-3).

General characteristics of the artery proximal to the lesion dilated are as follows:

- **Tortuosity**: None/mild = straight proximal segment or only one bend of 60° or more. Moderate = two bends of 60° or more proximal to the lesion. Severe = three or more bends of 60° or more proximal to the lesion
- **Arterial calcification**: Light = proximal artery wall calcification (not necessarily the lesion) seen as thin line(s). Heavy = easily seen calcification

Angiographic characteristics of the dilated target lesion are as follows:

- **Arrangement of the lesion(s).** Tandem = two lesions located within one balloon length (i.e., both lesions can be covered during a single balloon inflation). Sequential = two lesions located at a distance longer than the balloon
- **Length.** Discrete = ≤5 mm in length. Tubular = 5–10 mm in length. Diffuse = >10 mm in length
- **Eccentricity:** Concentric = lumen axis is located along the long axis of the artery or on either side of it, but by no more than 25% of the normal arterial diameter
- **Ostial.** Lesion is located at the aorto-ostial or bifurcation points
- **Side branch.** Bypassable side branch (1.5 mm or larger)
- **Contour:** Smooth, irregular, or ulcerated

Table 3-3

Lesion-specific Characteristics of Types A, B, and C Lesions

Type A Lesions (high success, >90%; low risk)	Type B Lesions (moderate risk)	Type C Lesions (high risk)
Discrete (<10 mm in length)	Tubular (10–20 mm in length)	Diffuse (>2 cm in length)
Concentric	Eccentric	
Readily accessible	Moderate tortuosity of proximal segment	Excessive tortuosity of proximal segment
Nonangulated segment, <45°	Moderately angulated segment, 45–90°	Extremely angulated segment >90°
Smooth contour	Irregular contour	
Little or no calcification	Moderate to heavy calcification	
Less than totally occlusive	Total occlusions <3 months old	Total occlusion >3 months old
Not ostial in location	Ostial in location	
No major branch involvement	Bifurcation lesions requiring double guidewires	Inability to protect major side branches
Absence of thrombus	Some thrombus present	Degenerated vein grafts with friable lesions

Modified from Ryan TJ, Faxon DP, Gunnar, *et al.* Guidelines for percutaneous transluminal coronary angioplasty. *J Am Coll Cardiol* 1988;12:529–545.

- **Thrombus:** Definite = intraluminal, round filling defect, visible in two views, largely separated from the vessel wall and/or documentation of embolization of this material. Possible = other filling defects not associated with calcification, lesion haziness, irregularity with ill-defined borders, intraluminal staining at the total occlusion site
- **Stenosis calcification.** Calcification at the actual lesion site
- **Angulation:** None/mild = lesion located on a straight segment or a bend of less than 45°. Moderate = 45–90° bend. Severe = bend of more than 90°. Bend should be evaluated in end-diastolic frame.

PROBLEMS AND SOLUTIONS IN THE INTERPRETATION OF CORONARY ANGIOGRAMS

Vessel Overlap

Because coronary angioplasty requires a clear view of the target vessel, which may be overlapped, multiple angles are required to reveal the locations of lesions not previously considered important.

Poor Vessel Opacification

Poor contrast opacification of the vessel may lead to a false impression of an angiographically significant lesion or lucency that could be considered a clot. Inadequate mixing of contrast and blood presents as a luminal irregularity. A satisfactory bolus injection of contrast must be delivered if adequate opacification is to be achieved and the angiogram is to be interpreted correctly. Enhanced contrast delivery can be best achieved by obtaining better coaxial engagement of the guiding catheter or using a larger catheter, injecting during Valsalva maneuver phase III, or using a power injector.

Total Vessel Occlusion

Total occlusion of a vessel may erroneously be suspected if a catheter is subselective in its location or an anomalous origin and course of a vessel are not recognized. A short left main coronary artery may lead to opacification of only the LAD, and a presumption of a circumflex occlusion or anomalous position may result. If this is thought to be the case, an aortic cusp "flush" injection of contrast may reveal the second vessel. Subselective injections into each vessel separately may be necessary if the left main artery is too short to opacify both vessels simultaneously. This problem may also occur for subselective injection into a large RCA conus branch that does not visualize the main RCA adequately.

Vasospasm

Catheter-induced spasm may appear as a fixed stenotic lesion. This has been observed in both right and left (and left main) coronary arteries and must be considered when an organic lesion is the only suspected anomaly. Nitroglycerin will reverse coronary spasm and should be administered in cases where

possible catheter-induced spasm is suspected. Catheter-induced spasm may occur not only at the tip of the catheter touching the artery but also more distally. Repositioning of the catheter and administration of nitroglycerin (100–200 μg through the catheter) may clarify if the presumed lesion is structural and not spastic. Often a change to a smaller-diameter (6 or 5 French) catheter, or catheters that do not seat deeply, may help.

Special Problems

Defining Left Main Coronary Anatomy. Since the engagement of the guide catheter into the left coronary ostium is critical to the successful performance of angioplasty, any disease within the left main coronary artery should be identified. Important left main coronary artery disease should be considered a contraindication to elective PCI for unprotected dilatation of proximal left coronary vessels. Optimal views to identify the left main coronary artery remain the same as for those during diagnostic studies, with a shallow RAO with cranial or caudal angulation often providing an excellent view. In addition, complementary LAO caudal view (spider view) will display the left main artery in an orthogonal projection.

Coronary angiography of patients who have left main coronary artery stenosis remains one of the few critical situations in which the operator and the team may affect the life and death of the patient directly. In general, interventions are not performed through left main stenosis of 40% of the diameter or more. However, some situations warrant stenting despite some degree of left main stenosis. These interventions are high-risk and should not be contemplated except by the most experienced interventionalist under exceptional circumstances. Left main stenoses of questionable severity can be accurately assessed by fractional flow reserve measurement (FFR) or intravascular ultrasound before recommending revascularization (see Chapter 10).

Left Circumflex Coronary Artery Takeoff. The angle of departure of the circumflex artery represents the most difficult aspect of angioplasty in this vessel. Since the circumflex may be upgoing and then immediately downgoing, a right-angled bend of the circumflex is best appreciated from the LAO caudal view. Horizontal takeoffs in the LAO cranial of the circumflex artery

permit easy passage of the system in the proximal portion of the coronary artery. A transfer to the RAO caudal projection will facilitate movement down the coronary artery. Guide catheter selection for the circumflex artery often requires longer guides (i.e., 4.0 JL4 guides, left Amplatz, or Voda) with special tips.

Anomalous Origin of Coronary Arteries. The most common coronary anomaly is the circumflex artery arising from the proximal RCA. This feature is often suggested during left coronary angiography by seeing a very long left main coronary artery segment with a very small or trivial circumflex branch. The circumflex may also be thought to be occluded. When the circumflex artery arises from the right coronary cusp or the proximal RCA it invariably follows a retroaortic course with the circumflex artery passing posteriorly around the aortic root to its normal position. During RAO ventriculography, aortography, or coronary angiography, the circumflex artery will be seen on end, appearing as a radio-opaque dot posterior to the aorta. This variant is benign. The left coronary anomalous pathways are described in Table 3-4 and shown in Fig. 3-4. The most dangerous is the left main segment passing anteriorly between the pulmonary artery and aorta, characterized by an anterior dot in front of the aorta.

Diagonal Branch Origin. Visualization of diagonal branches from the LAD artery is often best achieved using RAO with cranial projections or AP with cranial projections adequately identifying the takeoff from the path of the LAD. Lateral projections are often excellent but fail to reveal diagonal origins unless significant cranial angulation can be established. The mid and distal LAD artery segments are best seen with the lateral projection.

ANGIOGRAPHIC AND VIDEO IMAGING SYSTEMS

Video Systems and Fluoroscopy

In modern interventional practice, video systems and fluoroscopy have an increasingly important role in safe and effective procedures. Video and fluoroscopy systems have evolved more rapidly than any other component in the x-ray

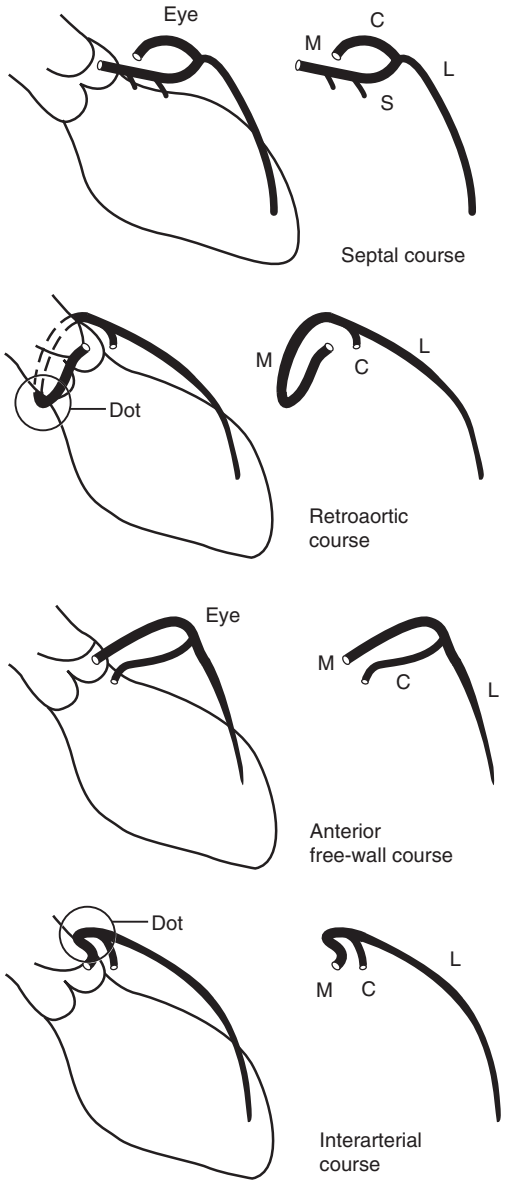

Fig. 3-4 Origin and radiographic appearance of left main coronary artery arising from right sinus or left sinus of Valsalva. (From Serota H, Barth CW III, Seuc CA, *et al.* Rapid identification of the course of anomalous coronary arteries in adults: the "dot and eye" method. *Am J Cardiol* 1990;65:891–898.)

Table 3-4

Origin and Radiographic Appearance of Left Main Coronary Artery Arising from Right Sinus or Valsalva

Left Main Artery Course	RAO Ventriculography		LAD Length	Septal From Left Main
	Dot	*Eye*		
Septal	–	+ (upper circumflex)	Short	Yes
Anterior	–	+ (upper left main)	Short	No
Retroaortic	+ (posterior)	–	Normal	No
Interarterial	+ (anterior)	–	Normal	No

LAD, left anterior descending coronary artery; RAO, right anterior oblique.
Adapted from Serota H, Barth CW III, Seuc CA, *et al.* Rapid identification of the course of anomalous coronary arteries in adults: the "dot and eye" method. *Am J Cardiol* 1990;65:891–898.

system. A typical video camera used for fluoroscopy has a lens that focuses light from the output phosphor of the image intensifier. The incident light strikes a target in the video camera composed of small globules of photoconductive material. Each small globule of material is insulated from surrounding areas. In general, the larger the target area, the better the image quality.

Video Signal Generation

The light-sensitive material emits electrons in quantities proportional to the intensity of the incident light. The free electrons are attracted to an anode in the video camera and each globule of photoconductive material thus becomes positively charged, acting as a small capacitor. An electron beam subsequently scans the photoconductive material, progressively discharging each tiny globule of material. The resulting current flows through a conductive signal plate and, following appropriate amplification, constitutes the video signal emerging from the camera. The voltage of the video output signal is proportional to the intensity of light that struck each point in the target.

A television monitor works in an opposite but analogous fashion. An electron beam scans a fluorescent screen that emits light in proportion to the number of electrons striking the phosphor. A control grid modulates the current of the scanning

beam based on the voltage of the incoming video signal. The resulting television image varies in brightness at each point in the image proportionally to the amount of light that originally illuminated the camera. The accuracy of video reproduction is determined by the precision with which the video camera converts light into an electrical signal and the accuracy with which the monitor converts the video signal into a fluorescent image.

Video Recording Systems

In modern laboratories, video tape recorders have assumed a secondary role, because digital angiographic systems have replaced video recorders as the principal means of reviewing images during interventional procedures. Cassette decks, including ¾ inch and ½ inch VHS, rarely offer more than 300 lines of resolution. A few laboratories have adopted the improved ½ inch super-VHS format to store images in a "cine-less" laboratory. At the moment, this practice results in suboptimal image quality and poor archival properties, and cannot be recommended.

Digital Archiving: Cine Replacement

Digital angiography is attractive as a long-term storage medium for archiving cardiac catheterization studies. Digital information allows production of multiple copies with no image degradation. Digital data can be transmitted electronically for remote examination and consultation. The economic cost of storing patient studies may be reduced relative to current film technology with a cost of approximately $100.00 per patient for film and development. Digital studies may cost less than $10.00 per patient.

At the time of publication, a committee from the American College of Cardiology and the National Electrical Manufacturers Association (NEMA) has been formed to address the issue of standardization. Until the development of a standard is accepted, most laboratories should defer conversion to digital archiving.

MEDICATIONS USED IN CORONARY ANGIOGRAPHY

Recording medications on cine during angiography will help the assessment of events during procedural review. If drugs are

given during the course of the catheterization that may affect the angiograms in any way, the film can be marked with a cineangiographic exposure of a radio-opaque drug marker to identify the drug and indicate that there may be a change in the subsequent arteriograms. Box 3-1 lists medications commonly used during cardiac catheterization.

Radiographic Contrast Media

The contrast material is selected from commercially available liquid solutions appropriate for the specific examination to be conducted. All contrast materials are x-ray-dense (as a result of iodine), as compared to anatomical structures, which are x-ray-lucent and absorb x-rays to produce different gray shades in x-ray images. The quantity and concentration of contrast materials used are specific to the patient's left ventricular and renal function, age, size, general health, and allergies.

All contrast agents contain iodine, an effective absorber of x-rays. Although all agents are derivatives of benzoic acid, the number of iodine molecules and ionic and osmolar composition will vary. Osmolarity, viscosity, sodium content, and other additives and properties are different among these agents. Ionic contrast media produce hypotension by peripheral arterial vasodilation, transient myocardial dysfunction, and decreasing circulating volume and blood pressure after osmotic diuresis (initially contrast media increased circulating fluid volume by osmotically shifting fluid into vascular space). For these reasons, nonionic or low-osmolar contrast agents are now routinely used. Selection of a nonionic or low osmolar contrast agent for the particular interventional procedure is, to a large extent, a matter of personal preference.

Major differences among the contrast agents include cost and potential effect on renal and left ventricular function. Thousands of studies have been performed safely with conventional high-osmolar/ionic agents and these pose no major risks. However, considerable data exist to suggest that the low-osmolar/nonionic agents are safer and provide satisfactory diagnostic quality, especially for high-risk patients. Low-osmolar/non-ionic contrast agents are favored for PCI, especially in patients with unstable ischemic syndromes, congestive heart failure, diabetes, renal insufficiency, hypotension, severe bradycardia, history of contrast allergy, severe

Box 3-1

Medications Used in the Cardiac Catheterization Laboratory*

Inotropics

- Digitalis, 0.125–0.25 mg IV >4 h apart
- Dobutamine, 2–10 mg/kg/min IV drip
- Dopamine, 2–10 mg/kg/min IV drip
- Epinephrine, 1:10,000 IV

Antiarrhythmics, anticholinergics, beta blockers, calcium blockers

- Adenosine, 5–12 mg IV bolus
- Amiodarone, 150–300 mg IV bolus
- Atropine, 0.5–1.2 mg IV
- Diltiazem, 10 mg IV
- Esmolol, 4–24 mg/kg IV drip (beta blocker)
- Lidocaine, 50–100 mg IV bolus; 2–4 mg/min IV drip
- Procainamide, 50–100 mg IV
- Propranolol, 1 mg bolus; 0.1 mg/kg in three divided doses (beta blocker)
- Verapamil, 2–5 mg IV, may repeat dose to 10 mg (calcium channel blocker)

Analgesics, sedatives

- Diazepam, 2–5 mg IV
- Diphenhydramine, 25–50 mg IV
- Fentanyl, 25 μg IV
- Meperidine, 12.5–50 mg IV
- Morphine sulfate, 2.5 mg IV
- Naloxone, 0.5 mg IV

Anticoagulants

- Heparin 40–70 u/kg bolus units IV

Vasodilators

- Nitroglycerin, 1/150 sublingual; 100–300 mg IV or IC
- Nitroprusside, 5–50 mg/kg/min IV

Vasoconstrictors

- Aramine, 10 mg in 100 mL saline, 1 mL IV
- Ergonovine, 0.4 mg IV in divided doses
- Norepinephrine, 1:10 000 IV; 1 mL dose IV
- Phenylephrine, 10 mg IV

Diuretic

- Furosemide, 20–100 mg IV

Metabolic buffers

- Calcium chloride and/or gluconate, 10 mEq
- Sodium bicarbonate, 50 mEq

Miscellaneous

- Protamine, 15–50 mg IV
- Succinylcholine, 1–4 mg IV

* The list is meant to be neither all-inclusive nor exclusive of emergency life-support techniques or standards.

valvular heart disease, and internal mammary artery and peripheral vascular contrast injections.

Coronary Vasodilators

Nitroglycerin. Nitroglycerin is the drug most commonly used during coronary arteriography and ventriculography. It dilates peripheral arteries, venous beds, and coronary arteries. Nitroglycerin is a very safe and short-acting drug. It can be given through the sublingual, intravenous, intracoronary, or intraventricular route. Sublingual (or oral spray) nitroglycerin (0.4 mg) is almost always given before coronary arteriography. Exceptions include patients in whom coronary spasm is suspected and those with hypotension (systolic pressure <90 mm Hg). In patients with documented coronary spasm, sublingual or intracoronary nitroglycerin is given to eliminate coronary spasm. In patients with unstable angina, intravenous infusion of nitroglycerin of up to 250 μg/min is permissible with a systolic blood pressure of 90 mm Hg. In patients with elevated left ventricular end-diastolic pressure in the catheterization laboratory from ischemia or from congestive heart failure, intraventricular or intravenous boluses of 200 μg of nitroglycerin will reduce left ventricular end-diastolic pressure and is appropriate if not required before or after ventriculography. Nitroglycerin increases coronary blood flow without a marked reduction in pressure in (intracoronary) doses of 50, 100, and 200 μg. In doses of more than 250 μg, hypotension without further increases in coronary blood flow may be evident.

Calcium Channel Blockers. Calcium channel blockers dilate vascular smooth muscle and reduce heart muscle contractility, and some block atrioventricular nodal conduction. Calcium channel blockers are used to reduce peripheral vascular resistance, reduce blood pressure, block coronary spasm, and increase coronary blood flow. Acute use in the cardiac catheterization is limited to treating arrhythmias and "no-reflow" of coronary interventions or to treat radial artery spasm when performing transradial approach.

Doses for calcium channel blockers are:

- Diltiazem; 30–60 mg PO, 10 mg IV
- Verapamil; 120 mg PO, 2.5–5 mg IV (for coronary "no-reflow," intracoronary bolus Verapamil 100–200 μg to be repeated for 2–4 doses if needed).

Adenosine. Intravenous adenosine is used for breaking supraventricular tachycardia and is the drug of choice for intracoronary induction of maximal hyperemia for coronary vasodilator reserve. For the RCA, the intracoronary adenosine dose is 24–30 μg and for the left coronary artery (LCA) 36–50 μg produces optimal results. Adenosine infusions, 140 μg/kg/min intravenously, produce sustained hyperemia. Adenosine hyperemia lasts less than 60 seconds after drug administration is ended.

Papaverine. Papaverine is a potent arterial vasodilator used in the investigation of coronary vasodilatory reserve. Intracoronary papaverine causes a marked increase in blood flow in the RCA in doses from 4 to 8 mg and in the LCA in doses from 8 to 12 mg. Doses exceeding these recommended levels do not appear to provide an increase over the maximal blood flow. Papaverine causes QT prolongation. Rare cases of papaverine-induced *torsade de pointes* have been reported, and antiarrhythmic preparations for this unusual event should be in place before administration of intracoronary papaverine.

Acetylcholine. Acetylcholine dilates normal coronary arteries and constricts diseased vessels. In Japan, intracoronary doses of 20, 50, and 100 μg have been used to induce coronary spasm in patients. The drug is very short acting and rapidly inactivated, making it suitable for catheterization laboratory use. Marked bradycardia, heart block, and vasospasm are common with acetylcholine. Temporary pacing is required during its administration. Continuous infusions of 0.02–2.2 μg (10^{-8}, 10^{-7}, 10^{-6} mol/L) have been used to identify normal endothelial function of coronary vessels (vasodilation, not vasoconstriction).

Nitroprusside. Nitroprusside is a potent, short-acting intravenous arterial vasodilator used in the treatment of aortic insufficiency, mitral regurgitation, hypertensive crisis, and congestive heart failure. Doses administered range from 10 to 100 μg/min and must be monitored by direct arterial pressure measurement. For coronary "no-reflow," 25–100 μg bolus can be used and repeated as needed.

Anticholinergics for Vagal Reactions

Atropine. Atropine is used to block vagally induced slowing of the heart rate and hypotension. Doses of 0.6–1.2 mg intravenously given immediately will reverse bradycardia and hypotension within 2 minutes. It is important to remember that, in elderly patients, heart rate may not slow during vagal episodes in which the only manifestation is low blood pressure. This low blood pressure can be alleviated by the administration of intravenous atropine and normal saline. In the rare patient in whom intravenous access is not immediately available, intra-arterial atropine (into the aorta) can be administered.

Antiarrhythmic Drugs

Amiodarone. Amiodarone is indicated for recurrent ventricular fibrillation or recurrent hemodynamically unstable ventricular tachycardia not responsive to adequate doses of other antiarrhythmics or when alternative agents cannot be tolerated.

The loading dose is 150 mg IV over 10 minutes (15 mg/min); then 360 mg IV over the next 6 hours (1 mg/min), followed by 540 mg IV over the next 18 hours (0.5 mg/min). After the first 24 hours, continue with maintenance intravenous infusion of 720 mg/24 hours (0.5 mg/min).

In the catheterization lab, amiodarone has been associated with bradycardia, hypotension, arrhythmias, heart failure, heart block, sinus arrest, and edema.

Amiodarone may reduce hepatic or renal clearance of certain antiarrhythmics (especially flecainide, procainamide, and quinidine). The use of amiodarone with other antiarrhythmics (especially mexiletine, propafenone, quinidine, disopyramide, and procainamide) may induce *torsade de pointes*. Use together cautiously with antihypertensives, beta blockers, and calcium channel blockers, because of increased cardiac depressant effects and slowing of SA node and atriventricular conduction.

Amiodarone may potentiate an anticoagulant response with the potential for serious or fatal bleeding. Decrease warfarin dosage by 33–50% when amiodarone is initiated.

Amiodarone is contraindicated in cardiogenic shock, second- or third-degree atriventricular block, and severe SA

node disease resulting in pre-existing bradycardia, unless a pacemaker is present.

Lidocaine. Lidocaine is an antiarrhythmic drug used to block or reduce the number of ventricular extrasystoles. Lidocaine can be administered as a bolus of 50–100 mg intravenously before ventriculography if a stable and quiet catheter position within the left ventricle cannot be obtained. In patients in whom myocardial ischemia is developing during cardiac catheterization or angioplasty, lidocaine for frequent ventricular ectopy is indicated. A bolus of 50–100 mg intravenously followed by a 1–2 mg/min infusion is usually satisfactory.

Cardiac Agonists

Dopamine. Dopamine is a potent vasoconstrictor. In low doses, it causes renal vasodilatation. In high doses, it causes peripheral vasoconstriction, elevating the blood pressure and increasing myocardial contractility. Dopamine 2–15 μg/min will cause vasoconstriction, elevating the blood pressure.

Dobutamine. Dobutamine is a potent inotropic agent with no peripheral vasoconstrictor effects. It increases cardiac contractility (inotropy) and is especially useful in patients with congestive heart failure. It may be used in conjunction with a potent vasodilator such as nitroprusside in those patients with markedly elevated left ventricular filling pressures and poor cardiac output.

Epinephrine. Epinephrine (1:10,000) is a naturally occurring catecholamine that stimulates cardiac function. It is administered only during cardiac emergencies. This medicine will increase heart rate and blood pressure immediately, sometimes to very high levels. Epinephrine should be reserved for cases needing cardiac resuscitation, or in which refractory hypotension is present and not responding to peripheral vasoconstrictors, or in the treatment of anaphylactic reactions. Transthoracic administration of epinephrine through a long needle is no longer performed. Intravenous or intra-arterial administration of 1 mL of 1:10,000 dilution can increase systemic pressure transiently during hypotension to a safe level

until intravenous vasopressors have been prepared. This dose of epinephrine has a duration of action between 5 and 10 minutes.

RADIATION EXPOSURE DURING PCI

Cardiac angiography with combined fluoroscopy and cineradiography carries the highest patient x-ray doses in diagnostic radiology. Coronary angioplasty will deliver greater x-ray exposure because of the more complicated and time-consuming nature of the procedure. Previous studies have demonstrated that operator exposure is 93% greater for angioplasty than for routine diagnostic coronary angiography. This increase is due to longer fluoroscopy times in angioplasty without corresponding longer cineradiography times. Because of the angled projections used in coronary angioplasty, increased x-ray exposure may be present. The scattered x-ray dose has been reported to be four times higher with angioplasty than with diagnostic cardiac catheterization (Fig. 3-5).

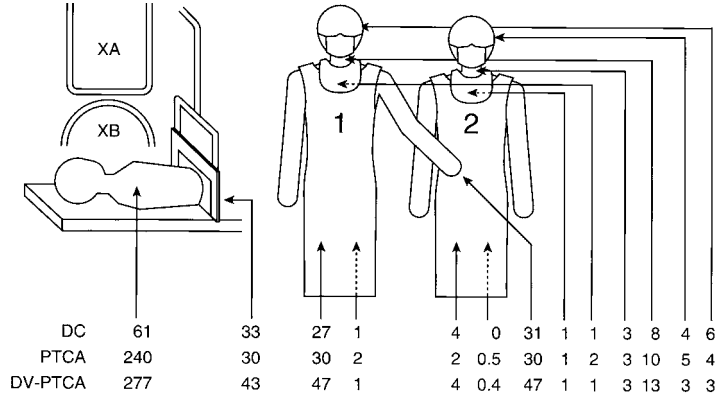

Fig. 3-5 Radiation exposure rates for two operators during coronary angioplasty. DC, Diagnostic catheterization; V-PTCA, double-vessel percutaneous transluminal coronary angioplasty; XA, x-ray amplifier in plane A; XB, x-ray amplifier in plane B. (Modified from Finci L, Meier B, Steffenino G, *et al*. Radiation exposure during diagnostic catheterization and single- and double-vessel percutaneous transluminal coronary angioplasty. *Am J Cardiol* 1987;60:1401–1403.)

Fluoroscopy Times

A study by Pattee *et al.* (1993) of radiation risk to patients from coronary angioplasty indicated that radiation doses varied considerably during the procedure because of large differences in exposure times. Fluoroscopy time per angioplasty case averaged 19 minutes, but in some procedures exceeded 60 minutes. Average patient skin entrance exposure per angioplasty procedure was 32 μCi/kg (124 rad), of which 70% was from cineradiography. Cancer mortality risk per angioplasty procedure was 8×10^{-4}. The study indicated that skin exposures estimated for angioplasty are, on average, higher than for other x-ray procedures, and that the cancer mortality risk does not exceed the mortality risk of bypass surgery (Table 3-5). Good professional practice requires maximal benefit-to-risk ratio for angioplasty procedures employing high-dose fluoroscopy or cineradiography.

New Device Procedure Times

New intracoronary interventional devices increase radiation exposure (Table 3-6). Federman *et al.* of the Mayo Clinic

Table 3-5

Organ Doses and Risks of Cancer Mortality for an Average Coronary Angioplasty Procedure

Organ	Organ Dose (cGy)*	Cancer Mortality Risk ($\times 10^{-6}$)
Red bone marrow	2.29	92
Bone (surfaces)	2.29	9.2
Lung	9.35	636
Thyroid	0.99	5.9
Breast (women)	4.89	157
Total risk		
Men		743
Women		899

* 1 Gy = 1 J/kg = 1 rad.

From Pattee PL, Johns PC, Chambers RJ. Radiation risk to patients from percutaneous transluminal coronary angioplasty. *J Am Coll Cardiol* 1993;22:1044–1051.

Table 3-6

Estimated Radiation Entrance Exposure of Patients Using Phantom Model Data

Procedure	Fluoroscopy (R)	Cine (R)
Isolated balloon angioplasty	43	25
Isolated directional coronary atherectomy	32	23
Directional coronary atherectomy + balloon angioplasty	66	29
Isolated laser coronary angioplasty	45	18
Laser coronary angioplasty + balloon angioplasty	57	27
Elective stenting	52	27
Emergency stenting	96	41

From Federman J, Bell MR, Wondrow MA, *et al.* Does the use of new intracoronary interventional devices prolong radiation exposure in the cardiac catheterization laboratory? *J Am Coll Cardiol* 1994;23:347–357.

(1994) found that, in 900 patients of whom two-thirds were undergoing balloon angioplasty and one-third were undergoing directional atherectomy or other new procedures, including 37 with intracoronary stent placement, the duration of fluoroscopy for angioplasty was 24 ± 18 minutes, which was greater than for directional atherectomy (18 ± 8 min). Fluoroscopy times were similar, at 25 and 29 minutes. When atherectomy or laser angioplasty was performed with balloon angioplasty, or if emergency intracoronary stent placement was performed, the duration of fluoroscopy was significantly prolonged compared to angioplasty alone. Increased radiation exposure should be expected when emergency procedures are required.

Angulated View and Radiation Exposure

Left anterior oblique views produce 2.6–6.1 times the dose of radiation for the operator of equivalently angled RAO views (Table 3-7). Steeper LAO views also increased operator dose. LAO 90° produces 8 times the dose of LAO 60° and 3 times the dose of LAO 30°.

Fluoroscopy produced more radiation than cine during angioplasty, by a factor of 6:1. Reducing the steepness of angulation reduces operator radiation dosage.

Table 3-7

Radiation Dose and Angulation

View	Dose (relative increase)
Image intensifier position	
RAO 30–60°	1
LAO 30–60°	2.6–6.1
Increasing angulation	
LAO 30°	1
LAO 60°	3
LAO 90°	9

LAO, left anterior oblique; RAO, right anterior oblique.

PERIPHERAL VASCULAR ANGIOGRAPHY (see also Chapter 11)

Cineangiography provides satisfactory information if the filming time, frame rates, and contrast dosages are properly established.

Renal Arteriography

Selective renal arteriography or arteriography obtained from aortic flush is used to evaluate the renal artery origins and vasculature. *Remember,* for renal artery identification during aortography, the origins of the arteries usually arise at the L1 vertebra (just below the T12 ribs). Selective renal arterial injections provide the most detail. The LAO projection often provides the best view of the renal artery ostia in a majority of patients. Acute angled takeoffs of the renal artery may require specially shaped catheters or a brachial arterial approach from above. Atherosclerotic disease of the renal artery usually involves the proximal one-third of the renal artery and is seldom present without abdominal atherosclerotic plaques. A renal artery stenosis artery is rarely the sole determinant for surgery or angioplasty. Refractory hypertension and determination of the renin–angiotensin levels are usually the indicators for an interventional (angioplasty or stent) procedure. Renal artery fibromuscular dysplasia may occur and appear as

atherosclerotic disease. This finding is often present in middle-aged women in whom other vessels are involved, most commonly cerebral or visceral arteries. In contrast to atherosclerotic narrowing, the proximal one-third of the main renal artery is usually free of disease.

Aortography—Thoracic and Abdominal Aorta

Aortography is indicated for suspected aneurysms or dissections by clinical, historical, or procedural signs. Injection techniques are the same as for ascending aortography. Evaluation of peripheral lower extremity disease requires identification of iliac bifurcation and common femoral artery patency before subselective injections. (Fig. 3-6).

Indications for abdominal aortography include:

- Non selective evaluation of renal arteries and mesenteric vessels
- Abdominal aneurysm or dissection

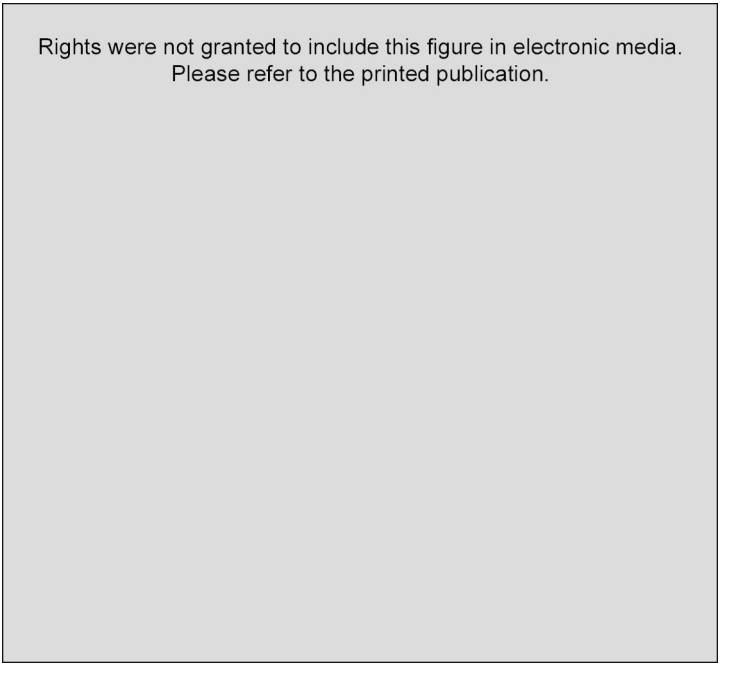

Fig. 3-6 Pelvic and proximal femoral arterial branches. (From Johnsrude IS, *et al. A practical approach to angiography*, 2nd ed. Boston: Little, Brown & Co., 1987.)

- Abdominal aortic atherosclerotic disease
- Vascular assessment prior to intra-aortic balloon pump insertion
- Initial evaluation of claudication
- Evaluation of the difficulty of catheter movement during coronary angiography.

The contraindications to abdominal aortography are the same as thoracic aortography.

Angiography of Lower Extremities

Based on clinical signs and symptoms of arterial insufficiency to the legs, suspected obstructions of vessel are often screened with noninvasive studies (i.e., ankle brachial index) before angiography is performed. Small-diameter (5F) catheters are satisfactory. Reduced volumes of contrast (10–20 mL over 1–2 sec) are injected during filming with panning down the artery, following the course to the most distal locations. Angulated views may be necessary to open bifurcations and overlying vessels that obscure the vessel origin. When possible, angiographic filming should extend at least to the ankle. Long cut-films that cover the entire lower extremity on a moving table are available in radiologic suites. In cardiac catheterization laboratories, cineangiographic filming with prolonged filming and panning down to the ankle must be tested before obtaining final views. Digital subtraction techniques are commonly available in modern laboratories. Nonionic contrast agents are less painful than ionic media for peripheral angiography.

One major challenge encountered with femoral–iliac angiography is the contralateral (opposite leg) approach, crossing over the aortic bifurcation of the iliac vessels, especially in patients with high bifurcation or prior aorto-bifurcation graft. To enter the opposite iliac artery, a right Judkins or internal mammary artery graft catheter or other special catheters (crossover, Simmons catheter, etc.) is advanced with a guidewire over the bifurcation and down into the opposite femoral artery. The wire is passed down into the selected artery. The catheter may be advanced and exchanged (over a long 300 cm wire) for an appropriate angiographic or balloon dilatation catheter, as required.

The area most frequently involved in peripheral atherosclerotic disease is the distal superficial femoral artery at the abductor canal (Fig. 3-7). The calf (tibial), and knee (popliteal) arteries are the next most commonly involved vessels after the superficial femoral artery. Disease in the deep femoral artery (femoral profunda) is rare. Pathways of collateralization are often rich and varied in patients with chronic distal femoral artery disease, especially in total occlusion of the superficial femoral artery that reconstitutes at or below the knee, close to the branching trifurcation of the tibial and deep peroneal arteries. Determining the level of reconstitution of collateralized vessels and distal runoff is crucial in determining the feasibility of revascularization. Magnified images focusing on the area of interest are frequently needed.

Diagrams and nomenclature for additional angiographic studies are shown in Figures 3-8–3-10; see also Chapter 11.

PACEMAKERS DURING PCI

Cardiac pacing, a low-risk means of providing emergency cardiac rhythm, is used in cases of symptomatic bradycardia or asystole. Since the introduction of low-osmolar or nonionic contrast media, significant bradycardia and asystole during PCI is rare, obviating the need for prophylactic pacing in this setting. Cardiac pacemakers may be used prophylactically during PCI to reduce the hemodynamic compromise of heart block and have been used to rescue patients after the development of conduction abnormalities associated with hypotension.

The routine use of pacemakers for PCI of RCA lesions is not required. External pacing patches are useful for emergency pacing when a temporary pacing wire cannot be immediately positioned. When using pacing patches, sedate the patient, since each electrical stimulation cause contraction of chest muscles as well as heart muscle and may be painful.

Indications

- Previously demonstrated high-degree conduction block
- Symptomatic bradycardia (after contrast or angiography of RCA)
- Acute myocardial infarction with trifascicular block

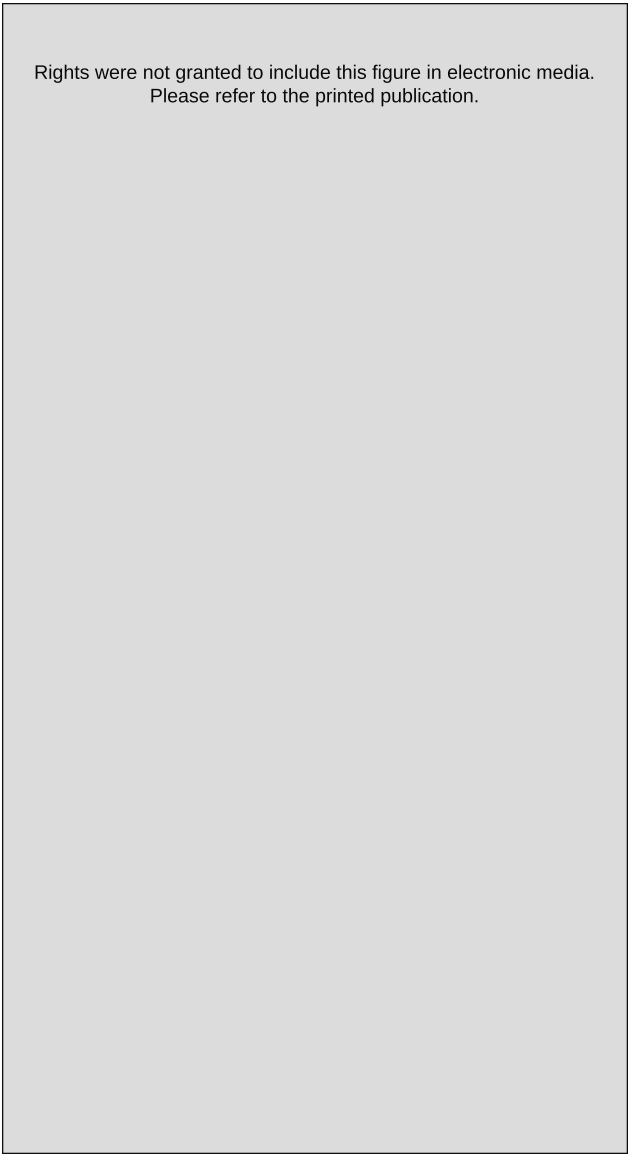

Fig. 3-7 Lower extremity vascular anatomy. (From Medical Learning Incorporated, with permission.)

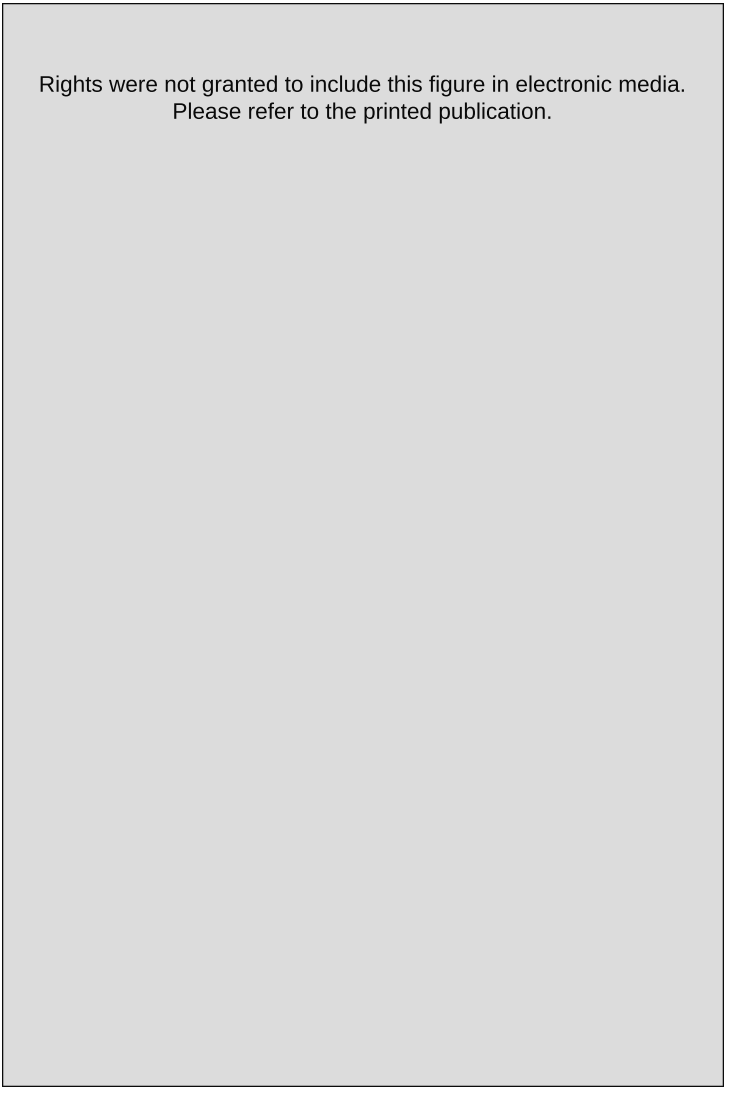

Fig. 3-8 Ascending aorta and head and neck vessels. (From Medical Learning Incorporated, with permission.)

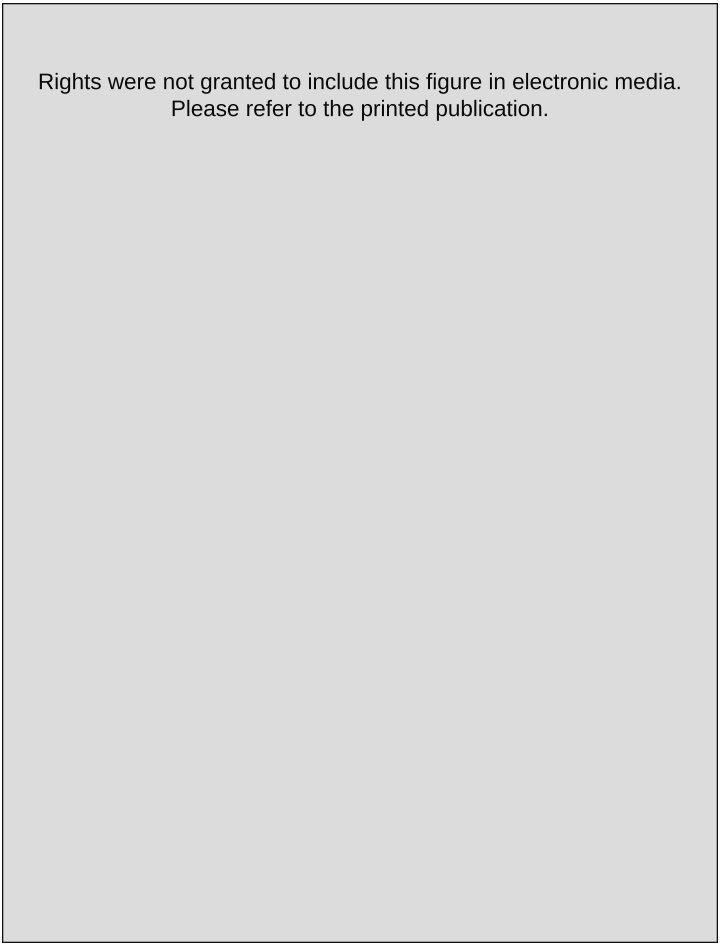

Fig. 3-9 Commonly accessed arteries of the abdomen. (From Medical Learning Incorporated, with permission.)

- Prophylactic use for rotational atherectomy and thrombectomy procedures, especially involving the RCA
- Transluminal alcohol septal artery ablation in hypertrophic obstructive cardiomyopathy (HOCM) patients.

Atropine may be used to prevent bradycardia but a pacemaker should be on standby for patients who experience severe bradycardia during coronary injections.

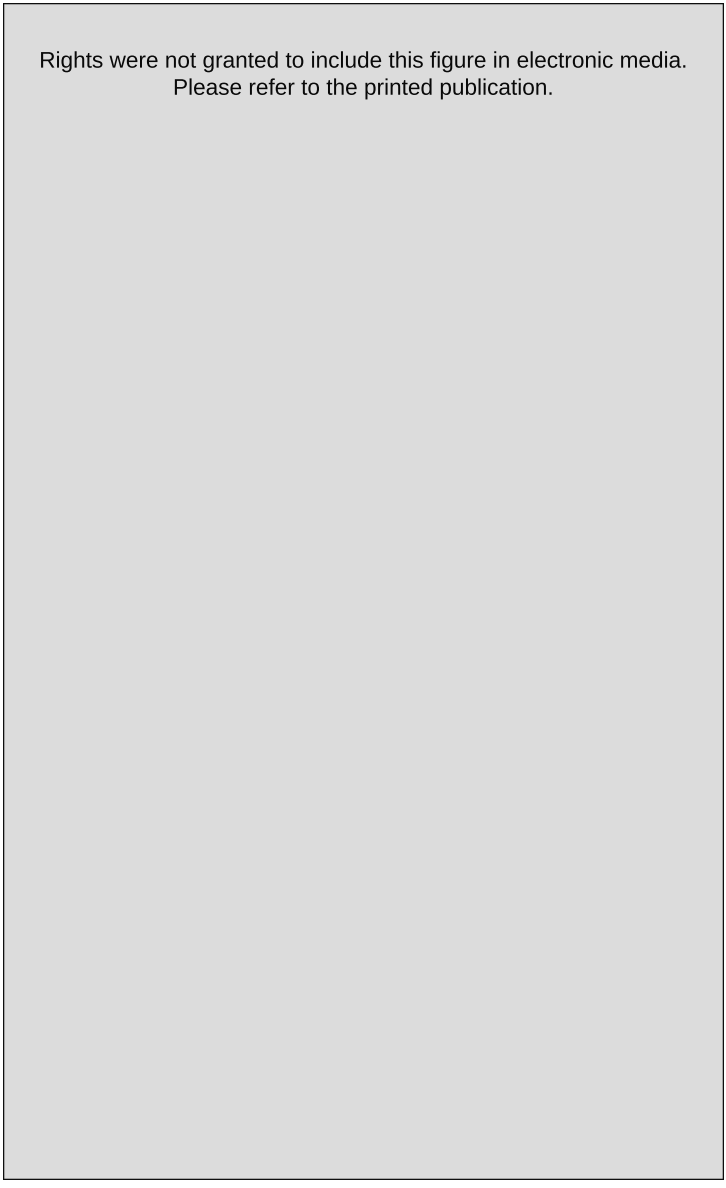

Fig. 3-10 Vascular anatomy of the upper extremity. (From Medical Learning Incorporated, with permission.)

Transvenous Technique

Temporary transvenous pacemaker placement can be achieved through the internal jugular, subclavian, brachial, or femoral vein route. The easiest access is usually the vein next to the arterial entry site. Right ventricular pacing is best accomplished with a 5 French balloon-tipped pacing catheter because there is a reduced incidence of perforation of the thin free wall or apex of the right ventricle when the balloon is inflated.

Cutaneous Patch Pacemakers

Cutaneous patch pacemakers are also effective until secured pacing routes can be established. Muscle contractions induced by the cutaneous pacing patches are uncomfortable so the patient should be well sedated.

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4

COMPLICATIONS OF PERCUTANEOUS CORONARY INTERVENTIONS

Glenn N. Levine and Morton J. Kern

The most important complications of percutaneous coronary interventions (PCI) relate to abrupt vessel closure, which may result in myocardial infarction or death. Minor complications predominantly involve hemostasis and vascular access problems (Box 4-1). Unusual complications, such as coronary ostial trauma (e.g., left main coronary artery dissection) and coronary perforation, are more frequent with devices requiring large-diameter guiding catheters or techniques of atherectomy or laser angioplasty.

The incidence of complications is related to both patient and lesion characteristics, as well as operator skill and procedural factors. A higher risk of adverse angioplasty outcome has been associated with a clinical presentation of unstable ischemia, probably due to the presence of intracoronary thrombus, impaired left ventricular function, and certain anatomic characteristics indicating increasing lesion complexity (Boxes 4-2, 4-3). Procedural factors, such as failure to administer aspirin and adequate doses of heparin, balloon-to-artery ratio of more than 1.3:1, and severe angiographically apparent intimal dissection, also confer an increase in the risk of vessel closure.

MYOCARDIAL INFARCTION DURING PCI

The incidence of myocardial infarction varies depending on the interventional technique employed, as well as the manner in which myocardial infarction is defined. Rates of myocardial

Box 4-1

Potential Complications Following Percutaneous Transluminal Coronary Angioplasty

Acute vessel closure (dissection, thrombus, spasm)
- Acute myocardial infarction
- Emergent coronary bypass surgery
- Death

Coronary artery emboli or perforation
Right ventricular perforation (with pacing catheter)
Coronary ostial dissection with guiding catheter
Fracture of guidewire within coronary circulation
Ventricular tachyarrhythmias
Severe angina
Transient hypotension
Transient bradyarrhythmias
Small coronary artery side-branch occlusion
Allergic reaction to radiographic contrast medium
Contrast nephropathy
Local vascular access-site complications: bleeding, arterial damage, thrombosis

From Kulick DL, Kawaniski DT. Percutaneous transluminal coronary angioplasty. In: Kulick DL, Rahimtoola SH, eds. *Techniques and applications in interventional cardiology*. St Louis, MO: Mosby, 1991: 76.

infarction reported for the following devices are estimated below.
- Balloon angioplasty, elective: 1–3%
- Coronary stenting, elective: 0.5–2% subacute thrombosis causing myocardial infarction
- Rotational atherectomy (rotablator)
  —ST elevation myocardial infarction: 2–3%
  —Non-ST elevation myocardial infarction: 5–6%
  (Predisposing factors: lesion length >4 mm; right coronary stenosis; bend >60°; female gender)
- Directional coronary atherectomy
  —5% for native vessels
  —10% in saphenous vein grafts.

ABRUPT VESSEL CLOSURE AFTER PCI

Ischemia due to impaired blood flow through the target vessel may cause arrhythmia, hypotension, infarction, and death if

Box 4-2

Characteristics Associated with Increased Mortality from Cardiac Catheterization

Age
Infants (<1 year old) and the elderly (>65 years old). Elderly women appear to be at higher risk than elderly men

Functional Class
Mortality in class IV patients is more than 10 times greater than in class I and II patients

Severity of Coronary Obstruction
Mortality for patients with left main disease is more than 10 times greater than for patients with one- or two-vessel disease

Valvular Heart Disease
Especially when combined with coronary disease, this condition is associated with a higher risk of death at cardiac catheterization than coronary artery disease alone

Left Ventricular Dysfunction
Mortality for patients with left ventricular ejection fraction <30% is more than 10 times greater than in patients with ejection fraction >50%

Severe Noncardiac Disease
Patients with:
- Renal insufficiency
- Insulin-requiring diabetes
- Advanced cerebrovascular and/or peripheral vascular disease
- Severe pulmonary insufficiency

Modified from Grossman W. Complications of cardiac catheterization: incidence, causes and prevention. In: Grossman W, ed. *Cardiac catheterization and angiography*, 3rd ed. Philadelphia, PA: Lea & Febiger, 1986.

the ischemia is not relieved in a timely manner. Acute ischemia is manifested by any one or combination of the following findings: chest pain, ST-T wave changes, arrhythmias, or hypotension. A hypertensive response may sometimes be seen early in response to pain. Hypotension may be associated with mental status changes (e.g., confusion, or loss of consciousness).

Box 4-3

Factors Associated with Abrupt Vessel Closure During Elective Coronary Angioplasty

Angiographic Factors
- Intraluminal thrombus
- Type B and C lesions
- Multivessel disease
- Ostial right coronary artery disease
- Saphenous vein grafts
- Subtotal coronary occlusions

Clinical Conditions Predisposing to Acute Vessel Closure
- Unstable angina
- Diabetes
- Female gender
- Advanced age (>80 years)

Conditions Associated with Increased Mortality after Major Complication of Coronary Angioplasty
- Unstable angina
- Left ventricular ejection fraction <30%
- Congestive heart failure
- Multivessel disease
- Proximal right coronary artery stenosis
- Unstable angina
- Age >65 years
- Female gender

Management of abrupt vessel closure must address the three most common mechanisms: dissection, thrombus, and spasm, alone or in combination.

Preventive Measures

Preventive measures should be used beforehand to limit the incidence of acute vessel closure.

- All patients should receive antiplatelet therapy (aspirin and clopidogrel when possible). Aspirin-allergic patients should be treated with clopidogrel (at least 300 mg at least 6 hours prior to the procedure when possible).

- Patients who do not receive concurrent glycoprotein IIb/IIIa inhibition should be treated with a weight-adjusted bolus of 70–100 u/kg of heparin, with a target activated clotting time (ACT) of 300–350 sec (Hemochron) or 250–300 sec (HemoTec). For patients who are treated with a glycoprotein IIb/IIIa inhibitor, a weight-adjusted bolus of 50–70 u/kg heparin should be administered, with a target ACT of 200–300 sec (Hemochron or HemoTec). A continuous intravenous heparin infusion after bolus administration is no longer recommended, although in longer procedures the ACT should be periodically checked.
- Systemic arterial blood pressure should be maintained with fluids, intravenous vasopressors (dopamine) or intra-aortic balloon pumping when necessary (e.g., systolic blood pressure <85 mm Hg). Temporary transvenous pacemaking should be instituted for heart block.
- Intra-aortic balloon pumping augments coronary blood flow and may be helpful in some patients.

A suggested management algorithm of acute or threatened coronary occlusion during PCI is shown in Figure 4-1.

Treat Coronary Dissections

Although characteristic angiographic imaging is diagnostic, at times, a dissection cannot be differentiated from thrombus,

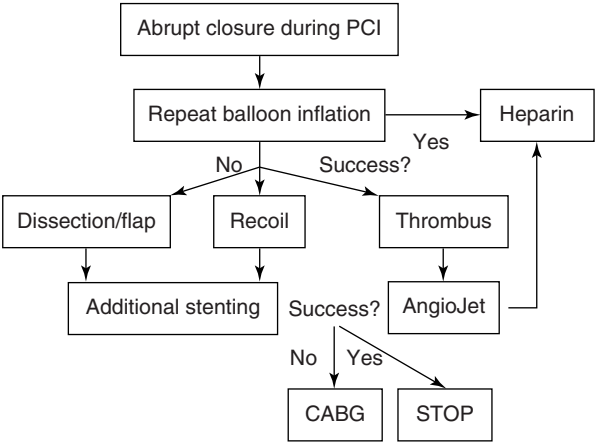


Fig. 4-1 Algorithm for treatment strategy for abrupt vessel closure. CABG, coronary artery bypass grafting.

with lucent and linear streaking of contrast in and around the site of angioplasty. Coronary dissection is treated with stent placement covering the extent of dissection. Figure 4-2 provides the NHLBI classification of coronary artery dissections for each. Risk of myocardial infarction and complexity of treatment increase with advancement of dissection types A–F.

Factors that limit effective stent placement in dissections include thrombus, small vessel size (small vessels are harder to treat) tortuosity, extensive calcification, tolerance for anticoagulation, and the length of the dissection. Box 4-4 lists conditions that complicate or may preclude stent placement. Figure 4-3 is an example of coronary dissection treated with a perfusion balloon and stenting.

Intracoronary Thrombus

Intracoronary thrombus has characteristic angiographic features, which include filling defect in contrast column, persistent contrast stain, meniscus sign, and long, worm-like

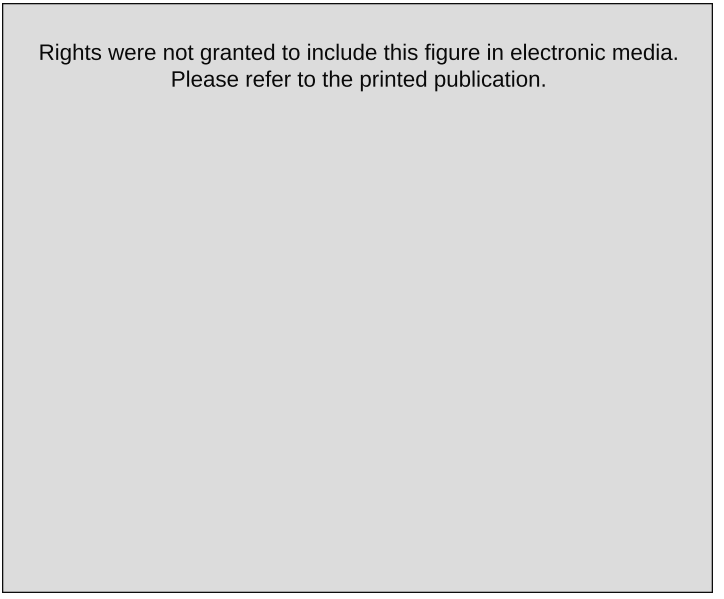


Fig. 4-2 Types of coronary artery dissection: NHLBI classification system. (Modified from Safian R, Freed M, eds. *The Manual of Interventional Cardiology*, 3rd edition. Birmingham, Michigan: Physicians' Press, 2001, p. 389.)

Box 4-4

Indications That May Preclude Bailout Stent Implantation

Anatomy
- Very small vessels (<2.0 mm)
- Severe angulation point
- Severe tortuosity
- Very long dissection
- Very proximal left anterior descending or circumflex dissection necessitating stent implantation in left main stem
- Thrombosis is the main component of acute occlusion

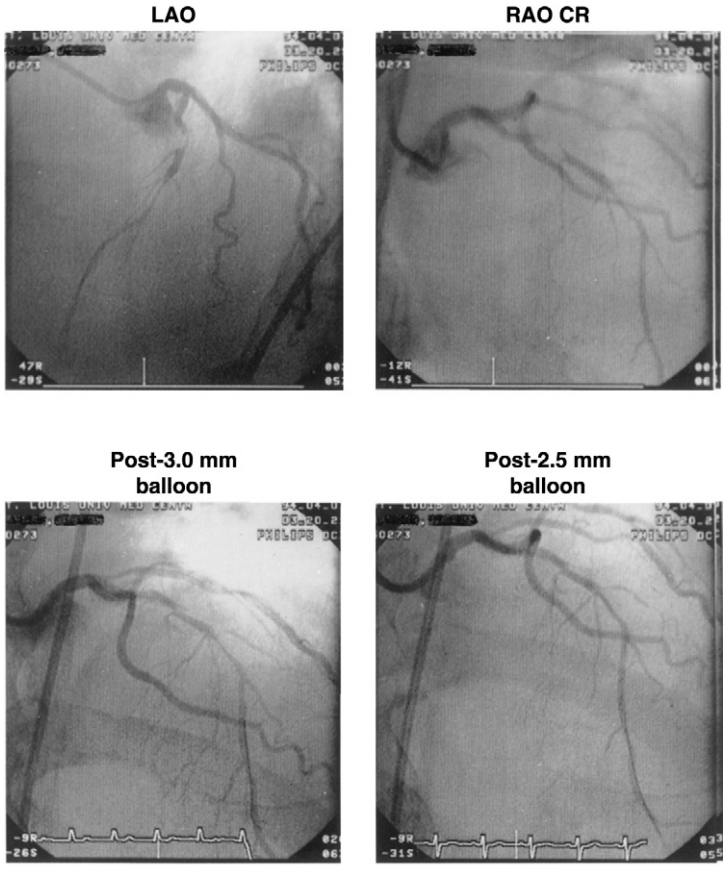
Clinical Situation
- Refractory unstable angina (Braunwald class IIIB and IIIC)
- Acute myocardial infarction
- Contraindication for intensive anticoagulant or antiplatelet treatment

From de Feyter PJ, de Jaegere PPT, Serruys PW. Incidence, predictors, and management of acute coronary occlusion after coronary angioplasty. *Am Heart J* 1994;127:643–651.

lucency in artery (Fig. 4-4). Presumed intracoronary thrombus is treated with increased heparin, serial balloon inflations or a thrombus aspiration system (e.g., Possis AngioJet), and glycoprotein IIb/IIIa inhibitor administration. Routine use of intracoronary thrombolytics has not reduced, and may potentially increase, the risk of ischemic events. Additional intravenous heparin is used to reduce thrombus propagation. Prolonged low-pressure balloon inflations using a slightly oversized balloon may compress thrombus. Anecdotally, many operators have noted that thrombus that is likely to be due to coronary artery dissection resolves after stent placement to treat the mechanical complication of arterial dissection.

Coronary Arterial or Thrombotic Embolism

Occasionally, coronary air embolism will occur in the performance of catheter exchange, balloon extraction, and reinsertion. Air embolism may occur under the following conditions:
- Incomplete aspiration of guiding catheter upon introduction into the circulation
- Balloon leakage or rupture



A

Fig. 4-3 Case example of coronary dissection during percutaneous transluminal coronary angioplasty (PTCA). **A,** Proximal left anterior descending artery stenosis (95%) is eccentric and precedes diffuse distal disease. PTCA with 2.5 mm and 3.0 mm PTCA balloons results in a dissection (type C).

Continued

- Prolonged negative suction of self-venting balloon catheters when exposed to room air
- Introduction of balloon catheters into the guide
- Removal of balloon catheters from deeply seated guiding catheters
- Structural failure of catheter

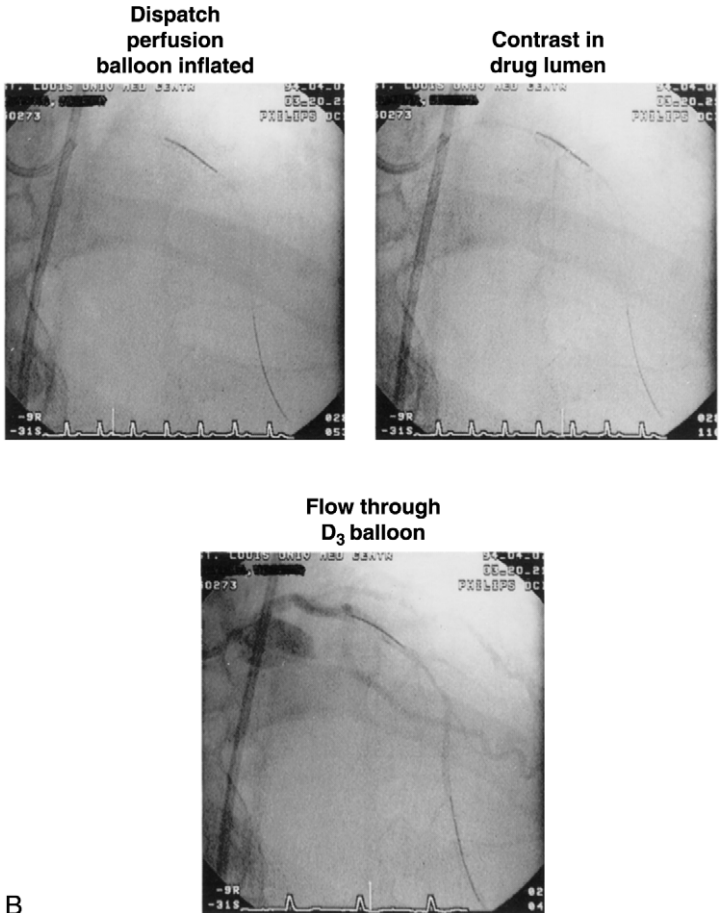
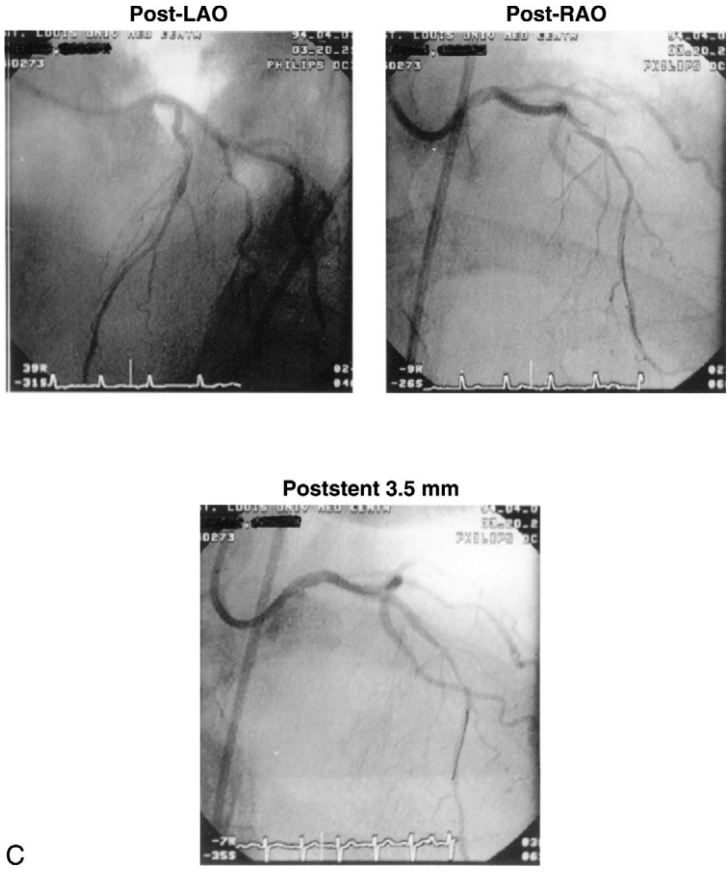


Fig. 4-3, cont'd **B,** A coiled perfusion balloon is placed in the dissection. Flow is maintained and ischemia reduced.

Continued

- Injection of air due to bubble in contrast injection line and/or syringe.

Management of coronary air embolus includes stabilization of hemodynamics and ischemia, analgesics, treatment of arrhythmias, circulatory support with pressors, intra-aortic balloon pumping as needed, and inhalation of 100% oxygen (F_IO_2) by mask. Air embolism is usually a self-limiting condition that generally results in no serious compromise;



C

Fig. 4-3, cont'd **C,** Dissection persisted despite prolonged balloon inflations. The vessel was stabilized after placement of 3.5 mm Palmaz–Schatz stent.

however, acute myocardial infarction has been attributed to air embolus despite successful coronary angioplasty.

Clot (thrombus) embolism may occur during guidewire passage or balloon inflation in a lesion containing thrombus or during intracoronary thrombolytic administration. On rare occasions, some thrombus may be transported proximally via the deflated balloon catheter and may embolize in an uninvolved territory. Limitation of the balloon catheter movement is recommended until a stable lumen is achieved.

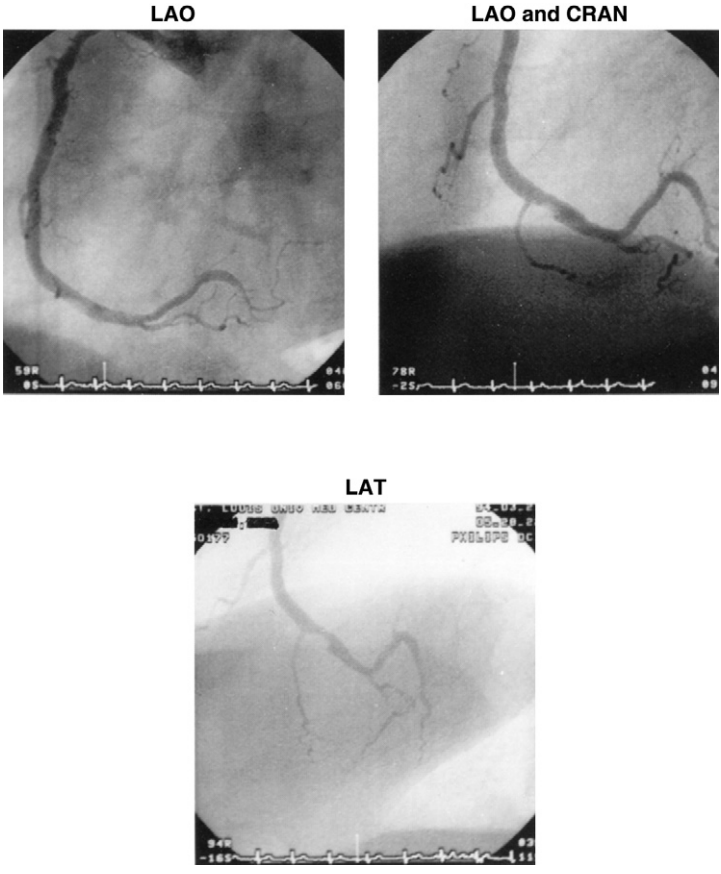


Fig. 4-4 Angiography of right coronary artery with thrombus before percutaneous transluminal coronary angioplasty. Note the difficulty in visualizing the outline of thrombus in the left anterior oblique (LAO) view.

Embolus in a relatively large segment or branch of the artery may be eliminated by balloon catheter passage and/or low-pressure balloon inflation. Thrombolytic infusions are not useful.

The No-reflow Phenomenon

The interventional cardiologist identifies the no-reflow phenomenon in the presence of an acute reduction in coronary flow despite a widely patent epicardial vessel during PCI. The

pathophysiologic mechanisms are thought to be: microvascular spasm, distal thromboembolism, and microembolization of atherosclerotic debris. No-reflow is an uncommon (0.6–2.0%) complication of PCI but occurs frequently following reperfusion for acute myocardial infarction or unstable angina and is most common during rotational atherectomy and PCI in saphenous vein grafts.

During PCI procedures, no-reflow usually manifests as acute ischemia with ECG changes and chest pain. Transient or permanent conduction disturbances, including atrioventricular block and bundle branch blocks, also occur. There is a 32% incidence of myocardial infarction when no-reflow is observed after PCI, and a 5–15% incidence of death. Hypotension and cardiogenic shock may develop, especially when baseline left ventricular function is diminished. However, most cases of no-reflow are clinically silent without sequelae reversing spontaneously. Box 4-5 describes methods to prevent no-reflow. Intra-aortic balloon pumping might be considered early in the course. Box 4-6 indicates methods of evaluation and treatment of no-reflow. Coronary bypass surgery does not correct no-reflow problems.

Box 4-5

Prevention of No-Reflow

- Distal protection devices when treating diffuse disease or bulky saphenous vein graft lesions, especially in older grafts
- When using rotational atherectomy, limit runs to less than 30 sec, avoid drops of more than 5000 rpm, begin with small burr size (1.25–1.5 mm), increase burr size no more than 0.5 mm, consider use of nitroglycerin, verapamil, and heparin combination in the flush solution
- Consider pretreatment with IIb/IIIa inhibitors during PCI in patients with unstable coronary syndromes
- Minimize balloon inflations—consider stent deployment without predilation and self-expanding stent designs that do not require high pressure inflation in vessels with bulky atheroma or in saphenous vein grafts
- Consider pretreatment with intracoronary verapamil, adenosine, nitroprusside
- Adenosine (10–20 μg bolus)
- Verapamil (100–200 μg boluses or 100 μg/min up to 1000 μg total dose with temporary pacer or standby)
- Nitroprusside (50–200 μg bolus, up to 1000 μg total dose)

Box 4-6

Evaluation and Treatment of No-Reflow

- Exclude dissection, thrombus, spasm at lesion site (intravenous ultrasound, distal contrast injections and/or translesion pressure gradient may be useful)
- Consider whether the ACT is adequate: 250–300 sec with unfractionated heparin if a IIb/IIIa inhibitor has been given, >300 sec if one has not been given, and 325–375 sec with direct thrombin inhibitors
- Maintain adequate perfusion pressure with intravenous fluids, vasopressors, inotropes, and intra-aortic balloon pump if necessary
- Administer intracoronary nitroglycerin (100–200 μg up to four doses) to exclude epicardial spasm
- Consider administering a glycoprotein IIb/IIIa receptor inhibitor
- Administer pharmacologic agents through an infusion catheter or the central lumen of a balloon catheter to ensure drug delivery to the distal bed.

Coronary Spasm

Coronary vasospasm occurs frequently during PCI. Intracoronary nitroglycerin (100–400 μg) can readily reverse vasospastic tendencies. Some patients may require continuous intravenous nitroglycerin to remedy spasm. Coronary vasospasm should be suspected in every case of reduced flow and excluded by the administration of intracoronary nitroglycerin. In rare cases, administration of a second vasodilating agent, such as intracoronary verapamil (100–200 μg), may be necessary.

Out-of-Laboratory Abrupt Closure After PCI

Abrupt vessel closure rarely occurs after stenting but can occur within the first 24 hours after balloon angioplasty. The mechanism of abrupt closure usually involves untreated dissection and/or thrombus formation with or without concomitant coronary vasospasm. Symptoms with ischemic electrocardiographic (ECG) findings demand repeat angiography and PCI if needed. Chest pain without new ECG findings suggests a noncoronary origin of the pain. New ECG findings with symptoms require repeat angiography.

Stent Thrombosis

Stent thrombosis occurs in the days to weeks after intracoronary stent placement. Factors associated with an increased risk

of stent thrombosis include inadequate expansion and/or apposition of the stent in the coronary artery, and inadequate oral antiplatelet therapy. Appropriate balloon/stent sizing and at least moderately high balloon inflammation (≥12 atm) should be used during stent placement. It should be verified that the balloon on which the stent is mounted is completely expanded at the time of stent deployment; no "waist" should be present. The presence of a "step-up" and "step-down" at the site of stent placement should be evaluated. Oral anticoagulation post-stent deployment should consist of both aspirin and clopidogrel. For the prevention of stent thrombosis, clopidogrel should be prescribed for 4 weeks in patients treated with bare-metal stents and for 3–6 months in patients treated with drug-eluting stents. All efforts should be made to avoid stent placement in patients who are undergoing brachytherapy.

Bypass Surgery for Acute Vessel Closure

The need for emergency coronary bypass surgery after acute or threatened artery closure is dramatically reduced with stenting. Mortality rates for emergency bypass after failed angioplasty range from 1% to 4%, with Q-wave myocardial infarction rates of 28–43%. Emergency bypass surgery limits the likelihood that surgeons will utilize an internal mammary artery conduit. Box 4-7 summarizes indications for *not* performing percutaneous reintervention.

PCI without on-site surgical backup should be limited to special situations. The ACC–AHA Task Force reporting guidelines for PCI in 2001 stipulate that "an experienced cardiovascular surgical team should be available within 60 minutes." Given the commonly available surgical facilities in the USA and the proximity of major medical centers to the population at large, it does not appear to be warranted that elective PCI should be performed in centers without on-site surgical backup, or at least surgical backup within easy reach by standby ambulance transport. Although several European countries report that PCI can be performed in large numbers without surgical coverage, these centers have highly experienced operators with on-site air ambulance backup. There appears to be no medical necessity to perform elective PCI without surgical backup at this time.

Box 4-7

Various Indications for Not Performing Percutaneous Reintervention

Expected problems in recrossing the lesion
- Tortuosity/calcification/angulation
- Unstable guiding catheter

Expected unfavorable redilation outcome
- Diffuse disease
- Long, spiral dissection
- Small caliber vessel
- Severe angulation

High risk of damage to left main artery
Severe hemodynamic instability
Additional significant (nondilatable) lesions

From de Feyter PJ, de Jaegere PPT, Serruys PW. Incidence, predictors, and management of acute coronary occlusion after coronary angioplasty. *Am Heart J* 1994;127:643–651.

HYPOTENSION

Hypovolemia

Hypotension during PCI, unrelated to myocardial ischemia, may be due to vasovagal stimulation, hypovolemia (e.g., retroperitoneal bleeding), or dehydration. These conditions are easily treated with fluid administration before or during the event.

Ischemia

Transient hypotension during the procedure due to ischemic myocardial dysfunction may start a downward spiral of more ischemia leading to more hypotension and shock. Urgent stabilization of blood pressure can be achieved with small intravenous doses of epinephrine or metaraminol. The epinephrine is prepared as a 1/10,000 dilution in a 10 ml syringe. 1 ml of this dilution can be used to maintain pressure. 1 mg of intravenous metaraminol will also raise blood pressure within 2 minutes and lasts for 5–10 minutes.

Cardiac Tamponade

Cardiac tamponade may occur after temporary pacemaker placement with an unsuspected perforation of the right

ventricle in an anticoagulated patient, or after coronary perforation from guidewire, balloon catheter, stent, rotoblation, or laser angioplasty. Tamponade must be excluded as an unsuspected cause of hypotension.

Management of tamponade after PCI is as follows:

- Reversal of heparin and pericardial drainage using standard subxyphoid techniques should proceed rapidly depending on the degree of hemodynamic compromise. Heparin reversal complicates an otherwise successful angioplasty and must be carefully considered. Early recognition and monitoring of pericardial effusion will permit selection of an aggressive or conservative approach
- Inflation of a balloon at the site of coronary perforation will control pericardial leakage from a ruptured or perforated artery
- Coronary guidewire perforation, although rare, is not always associated with significant complications but it should be monitored closely and steps taken to ensure that tamponade and/or continued bleeding are controlled. Depending on the clinical situation, management of guidewire perforation has included nonoperative management with prolonged coronary balloon compression to tamponade the puncture site, or emergency pericardiocentesis with urgent surgical repair and coronary artery bypass surgery. A covered stent has been approved by the Food and Drug Administration for the emergency treatment of arterial perforations.

ARRHYTHMIAS

Severe Bradyarrhythmias

During PCI, especially that involving the right coronary artery, symptomatic bradycardia not responding to atropine (0.5–1.0 mg intravenously every 3–5 min up to a maximum of 3 mg) requires immediate temporary pacing. Transcutaneous pacing may restore rhythm until transvenous pacing is established. When performing pacemaker insertion, especially under urgent circumstances, avoid maneuvers that may cause perforation of the right ventricle and resultant tamponade. The method of transcoronary cardiac pacing using an angioplasty guidewire connected to the pacemaker with a cable clamp is a backup technique. Transcoronary pacing can be performed successfully until a transvenous pacemaker can be inserted.

Ventricular Arrhythmias

During PCI, ventricular arrhythmias may result from transient myocardial ischemia, contrast injection, or catheter-induced ventricular stimulation (temporary pacemakers, Swan–Ganz catheter, deep guidewire manipulation). Ventricular arrhythmias can be managed with mechanical means (catheter repositioning), antiarrhythmics (lidocaine or amiodarone), or electrical cardioversion. Maintenance of left ventricular pressure with fluids, reduction of ischemia, and electrolyte replacement may be needed to eliminate ventricular irritability.

PERIPHERAL VASCULAR COMPLICATIONS

Vascular Access Site Complications

PCI vascular access site complications range from 6% to 29%, occurring more frequently following stenting (14%) and extractional atherectomy (12.5%) than with conventional balloon angioplasty (3.2%). These results were reported with 8 French guide catheters. The current incidence with improved anticoagulation regimen and smaller guide catheters (<7 F) is considerably lower.

Vascular access-site complications occur more frequently among females, older patients (≥65 years old), and those patients with peripheral vascular disease and the obese patients. Higher rates of postprocedural access-site hemorrhagic complications occur with concomitant use of fibrinolytic agents and with the duration and extent of heparin anticoagulation. In addition, multivariate analysis indicated that age above 70 years, stenting, multiple procedures, large sheath size, and low platelet count were also independent predictors of major vascular complications. These factors should be kept in mind when expressing the risk of vascular complications to patients undergoing PCI. Procedures with the highest rate of surgical repair after interventional procedures include balloon valvuloplasty, intra-aortic balloon pumping, and procedures involving sheaths of 10 French or larger.

Vascular access-site complications require early identification and diagnosis. Sanguinous oozing at the access site usually requires a dressing change, to observe the "ooze" or active bleeding, temporary manual compression if a sheath is in place, an "upsizing" of the vascular sheath (e.g., from 6 F to 8 F).

A large or continually expanding femoral or brachial vascular access-site hematoma necessitates the differentiation of active vessel bleeding, arteriovenous (AV) fistula, or pseudoaneurysm by use of vascular duplex color flow ultrasonography.

Retroperitoneal Hematoma

In current clinical practice, the incidence of major access site bleeding complications is low (0.7–1.7%). Vascular sheath insertion may produce groin or retroperitoneal hematomas. Retroperitoneal hematomas are rare (0.15–0.44%) but should be suspected in patients with unexplained hypotension and/or marked decrease in hematocrit. Patients with retroperitoneal bleeding may experience flank, abdominal or back pain, lower abdominal pain, urinary or bowel urgency, or peritoneal irritation. The absence of these symptoms does not exclude this condition. The diagnosis is made by abdominopelvic computed tomography (CT) scan. Most retroperitoneal hematomas can be treated conservatively with discontinuation and/or reversal of anticoagulation and antiplatelet therapy, and blood transfusion when necessary. About 16% of patients require surgery for persistent hypotension with a falling hematocrit despite transfusion, or femoral nerve compression.

Bleeding into the retroperitoneal space may be caused by a high femoral arterial puncture, a situation aggravated by the required anticoagulation. A high index of suspicion should prompt emergency CT scan for the diagnosis. Clinical signs of hypovolemia with or without a major drop in hemoglobin should alert one to the possibility of retroperitoneal hemorrhage. Treatment consists of discontinuation of anticoagulation, intravenous volume replacement, transfusions, and vascular surgery consultation.

Important steps that can limit vascular access-site hemorrhagic complications are listed in Box 4-8. They include:

- Preprocedural identification of variables associated with higher risk for bleeding complications
- Meticulous care in obtaining arterial and venous vascular access: single-wall, first-attempt puncture is best
- Avoidance of venous sheaths unless strongly indicated
- Weight-adjusted heparin dosing as described previously (based on AHA-ACC guidelines)
- Consider a vascular closure device

Box 4-8

Management Strategies to Limit Bleeding Complications Following PCI

Preprocedural Risk Assessment

Clinical variables

- Age
- Gender
- Weight

Angioplasty procedure

- Heparin dosing (weight-adjusted 40–70 u/kg, ACT >200 sec)
- Accurate arterial puncture site (midline femoral head)
- Vascular closure device

Postprocedural

- Sheath removal (target ACT <150–180 sec, bed rest)
- Compression device
- Meticulous clinical care and observation
- Vascular closure device
- Blood-product transfusion protocol

ACT = activated clotting time.

- Discontinuation of heparin infusions immediately postprocedure.

Femoral Pseudoaneurysm

After PCI and sheath removal, a femoral pseudoaneurysm, a communication between the femoral artery and the overlying fibromuscular tissue resulting in a blood-filled cavity, can form. Pseudoaneurysm occurs in 0.5–6.3% of patients. The condition is suggested by groin tenderness, a palpable pulsatile mass, and/or bruit in the groin area and can be confirmed by Doppler flow imaging. Small pseudoaneurysms are followed clinically; large pseudoaneurysms are treated with either ultrasound-guided compression, ultrasound-guided thrombin injection, surgical repair, or percutaneous polytetrafluoroethylene (PTFE)-covered stent-graft deployment.

Pseudoaneurysms are often due to inappropriately low or lateral femoral artery puncture site, inadequate arterial

compression, or excessive anticoagulation at the time of sheath removal. In general, pseudoaneurysms less than 2 cm in diameter are less likely to rupture than larger aneurysms. Infrequently, hemorrhage may occur in the anticoagulated patient during the postintervention period. Superficial femoral pseudoaneurysms may be a risk factor for rupture.

Duplex Doppler-guided pseudoaneurysm compression is a technique that is an effective nonsurgical management of this condition. Key points are listed below.

- Once the pseudoaneurysm cavity and communicating track are located, the surface of the Doppler imaging probe is pressed downward over the imaged track using continuous color flow Doppler monitoring
- The compression angle should be sufficient to obliterate the arterial inflow into the pseudoaneurysm cavity
- A registered vascular technologist or skilled technician may apply the compression
- The positioning of the probe should avoid compression of the femoral nerve and artery
- Manual compression is then maintained for 10 min intervals, after each of which pressure is slowly released and inflow into the pseudoaneurysm reassessed
- Compression can be continued for 45 min
- If compression longer than 2 hours is performed with nonclosure of the communicating track, surgical intervention may be required. Duplex Doppler ultrasound-guided compression of the femoral artery pseudoaneurysm is successful in more than 80% of patients in whom the technique is applied.

Arteriovenous Fistula

An AV fistula resulting from sheath-mediated communication between the femoral artery and femoral vein is associated with a systolic and diastolic bruit and confirmed by Doppler examination. The reported incidence ranges from 0.2% to 2.1%. AV fistulas can be observed or treated with ultrasound-guided compression, surgical repair, or implantation of covered stents.

Cholesterol Emboli

Cholesterol embolism is an extremely rare and devastating complication of any invasive vascular procedure. Cholesterol

crystals from disrupted atheromatous plaques occlude small arteries and arterioles. Patients may present days to weeks after PCI with blue (cyanotic) toes and foot pain, livedo reticularis, gangrene, renal failure, neurologic or ocular deficits, or bowel ischemia. Cholesterol emboli have been associated with a triad of foot pain, livedo reticularis, and intact peripheral pulses. Only anecdotally reported cases have been documented after PCI.

There is no proven treatment for cholesterol embolism syndrome. Heparin does not help and may exacerbate the condition.

Complications of Vascular Access Closure Devices

Many arterial access sites will now be closed in the laboratory with percutaneous vascular closure devices. Hemostasis success rates are less than 100%. Each device has failure modes particular to its mechanism of action. Suture fractures or failure to deliver a knot (Prostar, Closer), collagen introduction or emboli into the vessel (Angio-Seal, VasoSeal, Duett), or failure to seal the puncture site cause femoral or retroperitoneal bleeding. All vascular complications, including pseudoaneurysm, bleeding and hematoma, infection, arterial stenosis or occlusion, and venous thrombosis, can occur, with an incidence of approximately 1–5%.

Compared to manual compression, percutaneous closure device complications tend to have a greater incidence of pseudoaneurysms not amenable to ultrasound compression therapy, a greater loss of blood and need for transfusions, a greater incidence of arterial stenosis or occlusion, the need for more extensive surgical repair, and a greater incidence of groin infections. Thus, patients treated with vascular closure devices merit as much, if not more, attention to vascular complications than those treated with manual compression.

Limb Ischemia

Management of limb ischemia depends on the probable cause, which includes the following.

Acute Thromboembolism. For thromboembolism, thrombectomy or thrombus aspiration via catheter can be performed. Intra-arterial nitroglycerin for vasospasm may be helpful.

Vascular surgical consultation should be obtained, with Doppler assessment of pulses.

Sheath-Induced Ischemia. For sheath-induced ischemia, remove the sheath. Consult vascular surgery for pulse loss or persistent bleeding.

Venous Occlusion

After prolonged arterial pressure-device application, femoral venous thrombosis may develop, with swelling, pain, and cyanosis in the extremity. Resume heparinization. Consult vascular surgery.

Blood Product Transfusions

Procedures that increase the incidence of local vascular complications increase the requirement for blood transfusion. Prototypic guidelines for blood product transfusions adapted from Welch *et al.* are listed in Box 4-9. Asymptomatic patients

Box 4-9

Transfusion Guidelines

Packed Red Blood Cell Transfusions

- Correct hypovolemia (crystalloid infusion)
- Avoid transfusion "thresholds"
- Transfuse only if:
  - —Signs/symptoms occur
  - —Hematocrit falls below 21%
  - —"Unit-by-unit" basis: relief of symptoms

Other Transfusions

- Severe/acute bleeding/emergency surgery
- Administer random donor platelets for:
  - —Emergency measure
  - —Severe asymptomatic thrombocytopenia (i.e., <50,000/mm^3)
  - —Thrombocytopenia (<100,000/mm^3) associated with bleeding
  - —Bleeding time >9 min
- Reserve cryoprecipitate and fresh frozen plasma for true coagulation abnormalities

From Welch HG, Meehan KR, Goodnough LT. Prudent strategies for elective red blood cell transfusion. *Ann Intern Med* 1992;116:393–402.

with evidence of clinical bleeding should receive crystalloid infusion to correct hypovolemia. Transfusion "thresholds" should be put in clinical context. For example, many clinicians use a hemoglobin concentration of less than 10 g/dl as the threshold for initiation of transfusion therapy. Normovolemic anemia (i.e., hemoglobin concentration of 7–10 g/dl) is often clinically well tolerated. Packed red blood cell transfusion should be administered only if signs or symptoms of anemia or acute blood loss occur. In addition, hemoglobin concentrations of less than 7 g/dl or a hematocrit of less than 21% are usually not well tolerated, often necessitating blood-product transfusion. Administration of packed red blood cells should be performed on a "unit-by-unit" basis with the goal of obtaining symptom relief. The guidelines for other blood-product transfusions, including platelets, are also listed in Box 4-9.

COMPLICATIONS RELATED TO RADIOGRAPHIC CONTRAST MEDIA

Anaphylactoid Reactions

Contrast-mediated anaphylactoid reactions to contrast often occur upon first exposure to contrast media and recur with the same severity on subsequent exposures. Direct complement activation is postulated to be the mechanism. Clinical manifestations of anaphylactoid reaction include urticaria in approximately 1% of patients, angioedema or bronchospasm in 0.03%, and circulatory shock in 0.01%. Patients with a history of anaphylactoid reactions are at high risk of recurrence, ranging from 15% to 35%. Recurrence is reduced to 5–10% for minor reactions and less than 1% for severe reactions upon pretreatment with prednisone and diphenhydramine. Table 4-1 summarizes treatment regimens for anaphylactoid reactions during PCI.

Contrast Nephropathy

Contrast nephropathy may occur in patients with compromised renal function who receive doses of radiographic contrast media larger than 100 ml. The incidence for a detectable change in renal function of more than 1 mg/dl rise in creatinine is less than 1% for hospitalized patients. In patients with baseline creatinine above >1.5 mg/dl, the risk

Table 4-1

Recommended Regimens for Treatment of Anaphylactoid Reactions in Patients Undergoing PCI

Clinical Setting	Pretreatment/Treatment
Pretreatment	
Known history of prior contrast anaphylactoid reaction	Prednisone 50 mg PO administered 13, 7 and 1 h pre-procedure and diphenhydramine 50 mg PO 1 h pre-procedure *Or* Prednisone 60 mg PO administered the night before and on the morning of the procedure and diphenhydramine 50 mg PO on the morning of the procedure
Treatment	
Urticaria/pruritus	No treatment or diphenhydramine 25–50 mg IV If no response to therapy: epinephrine (0.3 ml of 1:1000 solution SC) q15 min up to 1 ml ± cimetidine (300 mg) or ranitidine (50 mg) in 20 ml normal saline IV over 15 min
Bronchospasm	1. Oxygen by mask and oxymetry monitoring 2. Depending on condition: Mild: Albuterol inhaler Moderate: Epinephrine 0.3 ml of 1:1000 solution SC q15 min up to 1 ml Severe: Epinephrine 10 μg boluses IV q1 min then infusion of 1–4 μg/min 3. Diphenhydramine 50 mg IV 4. Hydrocortisone 200–400 mg IV 5. Optional: H_2 blocker
Facial and laryngeal edema	1. Airway protection/intubation + supplemental oxygen 2. Epinephrine 0.3 ml of 1:1000 solution SC q15 min up to 1 ml, or epinephrine 10 μg boluses IV q1 min then infusion of 1–4 μg/min
Hypotension/shock	1. Epinephrine 10 μg boluses IV q1 min then infusion of 1–4 μg/min 2. Supplemental oxygen/intubation 3. Diphenhydramine 50–100 mg IV 4. Hydrocortisone 400 mg IV 5. If unresponsive to therapy: dopamine, H_2 blocker, and advanced cardiac life support as indicated

Most recommendations adapted from Goss *et al.,* 1995.

Table 4-2

Incidence, Prevention, and Management of Adverse Events

Adverse Event	Incidence	Comments
Preprocedure		
Allergic reactions	Rarely in general population 17–35% in patients with prior reaction (if not premedicated)	Pretreat patients with history of allergic reaction to contrast with steroid and H_1 blocker
Contrast nephropathy	14–38% incidence of any detectable effect on kidney function Incidence of actual renal failure <1% in general population	Risks higher in diabetics and patients with pre-existing renal insufficiency Hydrate patients pre- and post-procedure Consider *N*-acetyl cysteine
Lactic acidosis	Occurs rarely and almost exclusively in patients on metformin with dye-induced renal failure	Hold metformin the day of procedure and for 2 days afterwards Reassess kidney function before restarting
Periprocedure		
Myocardial infarction	Enzymatically defined myocardial infarction, 5–30%	Patients with significant elevations in CK-MB may warrant additional monitoring and care, and more cautious resumption of daily activities Intracoronary nitroglycerin (rule out spasm) Consider aspiration, embolectomy devices Emergency aortocoronary bypass
Emergency CABG	<1%	Decreased incidence probably due to stents
Death	<1%	Decreased incidence probably due to pharmacotherapy and technology

Major bleeding	0.7–1.7%	Discontinue heparin therapy post-procedure
Acute pulmonary edema	Uncommon	Oxygen, morphine (2–5 mg IV), nitrates (100–200 μg IV), furosemide (20–200 mg IV) Nitroprusside for afterload reduction with dopamine Intra-aortic balloon pump Endocardial intubation, sedation, monitoring PCWP Treat pre-existing CHF optimally
		Limit contrast medium, avoid LV angiography Use nonionic or low-osmolar contrast medium Avoid hypotension Check oxygen saturation
Cardiogenic shock	Usually associated with acute myocardial infarction	If shock caused by coronary occlusion, treat with emergency PCI or CABG Manage as high risk with hemodynamic support Prophylactic IABP
		Rule out tamponade with RA/RV pressures and urgent echocardiogram
Ventricular tachycardia or fibrillation	0.6%	Use nonionic contrast agents Do not inject when catheter tip pressure is damped Cough to maintain blood pressure Remove catheter from coronary ostium Cardiopulmonary resuscitation followed by prompt defibrillation Defibrillation (200, 300 then 360 J) Epinephrine 1 mg IV or vasopression 40 u IV Lidocaine 1.0–1.5 mg/g IV bolus or amiodarone 300 mg IV (rapid infusion after dilution in 20–30 ml fluids)

Continued

Table 4-2

Incidence, Prevention, and Management of Adverse Events

Adverse Event	Incidence	Comments
Cerebrovascular accident	0.1%	Observation Stabilization Neurologic consultation Systemic heparinization, aspirate/flush catheters frequently Remove air bubbles in tubing or injection syringes, change bottles of contrast medium
Retroperitoneal hematoma	0.15–0.44%	Suspect in patients with unexplained hypotension or fall in hematocrit, severe back or flank pain, or tachycardia (if not receiving beta blockers) Diagnosis confirmed by computed tomography Reverse anticoagulants Volume replacement Transfusion if hematocrit <25 Surgical consultation Avoid high femoral artery puncture
Large coronary dissection	0.1%	Do not manipulate catheter in coronary ostium, monitoring pressure of catheter tip Do not inject with damped pressure Limit coronary injections If ischemia produced, and cannot stent, emergency aortocoronary bypass If dissection associated with thrombus but no ischemia, use stent and aspiration device
Cardiac tamponade	Rare	Reverse anticoagulation, urgent echo Prompt pericardiocentesis with catheter drainage Cardiovascular surgery consultation

		Surgical exploration and closure for persistent bleeding Avoid stiff catheters in RA or RV; pacing catheters handled gently Avoid posterior LA wall during trans-septal catheterization
Pseudoaneurysm	Approximately 1%	Presence suggested by groin tenderness, a palpable pulsatile mass, and/or bruit Confirmed by ultrasound examination
Arteriovenous fistula	0.2–2.1%	Presence suggested by continuous bruit over groin area Confirmed by Doppler ultrasound examination
Air embolism	Rare	Meticulous flush technique Same as cerebrovascular accident
Cholesterol embolism	Extremely rare	Suspect if severe toe or foot pain with intact pulse, livedo reticularis, renal failure, or multiorgan pathology Diagnosis further suggested by eosinophilia and confirmed by biopsy
Post-procedure		
Stent thrombosis	Approximately 1%	Clopidogrel for 4 weeks post-PCI (in addition to chronic aspirin therapy) for bare-metal stents; clopidogrel 6–9 months (in additon to chronic aspirin therapy) for drug-eluting stents Patients with severe anginal chest pains days–weeks after stent implantation should be admitted
Radiodermatitis	Rare, only anecdotally reported	Myriad of dermatological pathology can occur hours–years after exposure(s) Lesions most commonly occur in axilla, scapula, or midback regions

CABG, coronary artery bypass grafting; CHF, congestive heart failure; CPR, cardiopulmonary resuscitation; IABP, intra-aortic balloon pump; LA, left atrium; LV, left ventricle; PCI, percutaneous coronary angioplasty; PCWP, pulmonary capillary wedge pressure; RA, right atrium; RV, right ventricle.

increases by two to three times. In diabetic renal insufficiency, the incidence may be 30–50%. Kidney transplant recipients are also at high risk of contrast-induced nephropathy when the volume exceeds 125 mg per exposure. Mechanisms to reduce contrast nephropathy include limiting the amount and frequency of contrast administration and preprocedural hydration. Administration of various pharmacologic agents during the periprocedural period, including furosemide, mannitol, dopamine, aminophylline, and atrial natriuretic peptide, have not been shown to be of benefit in patients undergoing diagnostic cardiac catheterization or PCI and in some cases had a detrimental impact on renal function. Several studies have now demonstrated a modest protective effect of acetylcysteine administration in the pre- and periprocedural period (600 mg PO bid on the day prior to and day of PCI).

An overview and summary of complications and their management during PCI are provided in Table 4-2.

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5

ANTITHROMBOTIC AND ANTIPLATELET THERAPY FOR PERCUTANOUS CORONARY INTERVENTIONS

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Familiarity with commonly used antithrombotic and antiplatelet agents is required in the management of interventional cardiology patients. This chapter outlines recommendations and uses of various antithrombotic agents. Antithrombotic and antiplatelet agents for PCI are summarized in Box 5-1 and recommendations from the AHA/ACC/SCAI are summarized in Table 5-1.

ANTICOAGULANTS

Heparin

Mode of Action.

- Heparin is a mixture of glycosaminoglycans (mucopolysaccharides) that combine with a plasma protein called antithrombin III (AT III) to make the AT III a highly effective inhibitor of thrombin and several other clotting factors
- Heparin requires the presence of AT III to be effective
- Unfractionated heparin is a heterogeneous polysaccharide that binds to antithrombin to inhibit thrombin and factor Xa.

Absorption and Clearance. Elimination via a rapid, saturable phase secondary to hepatic uptake and a slower phase from renal clearance.

Box 5-1

Antithrombotic and Antiplatelet Agents for Percutaneous Coronary Intervention

Antithrombotic Therapy
- Heparin (unfractionated)
- Low-molecular-weight heparin
- Direct thrombin inhibitor
  —Polypeptide inhibitors (hirudin [Lepirudin], bivalirudin [Hirulog])
  —Low-molecular-weight inhibitors (argatroban [Acova])

Antiplatelet Therapy
- Aspirin
- Clopidogrel
- Platelet GP IIb/IIIa antagonists
  —Abciximab
  —Eptifibatide
  —Tirofiban

Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*, Birmingham, MI: Physicians' Press, 1999.

- Plasma half-life of 60–90 min when given intravenously
- Slower absorption and clearance when given subcutaneously.

Route of Administration. Intravenous or subcutaneous, *not intramuscular.*

Duration of Action. Minutes to hours when given intravenously, several hours when given subcutaneously.

Frequency of Dosing.
- Single bolus
- Constant infusion intravenously
- Every 8–12 hours subcutaneously.

Monitoring. Heparin therapy is usually monitored by the activated clotting time (ACT), which is the time for whole blood to form a firm clot. When blood levels of heparin have been measured directly, antithrombotic efficacy occurs at between 0.2 and 0.4 u/ml. It is common practice to maintain the Hemochron ACT at 300–350 sec and the HemoTec ACT at

Table 5-1

Recommendations for Pharmacologic Management of Patients Undergoing Percutaneous Coronary Intervention

	Clinical Status				
			Transmural MI		
Drug	Class I Angina	Class II–IV Angina, Unstable Angina NSTEM	Acute Phase MI	After Thrombo-lysis	Hospital Manage-ment Phase
Aspirin	I	I	I	I	I
Ticlopidine, clopidogrel*	I†	I	I	I	I‡
Warfarin§	III§	III	III	II	I
GP blockers¶: Abciximab Tirofiban Eptifibatide	II	I	II	I	III
Unfractionated heparin	I	I	I	I	III¶

Roman numerals indicate American College of Cardiology/American Heart Association class indication I, II, III.

* In conjunction with stenting. † To be given 24–48 h before planned stenting, if possible; ‡ 2–4 weeks following stent placement. § Patients without atrial fibrillation or other pre-existing clinical indications: patients with anterior myocardial wall motion abnormalities or left ventricular thrombus. ¶ Every indication to replace unfractionated heparin. ¶ Other noncoronary thrombotic complications (e.g., thrombophlebitis).

LV, left ventricular; MI, myocardial infarction; NSTEM, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention.

(Reproduced with permission from Smith SC Jr, Dove JT, Jacobs AK, *et al.* ACC/AHA guidelines of percutaneous coronary interventions (revision of the 1993 PTCA guidelines)—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (committee to revise the 1993 guidelines for percutaneous transluminal coronary angioplasty). *J Am Coll Cardiol* 2001;37:2215–2239.)

250–300 sec, although some interventionalists may prefer higher values.

For use with abciximab, the recommended heparin bolus is reduced to 40–70 u/kg to achieve a target ACT of 200–250 sec. Although the current FDA-approved labeling of eptifibatide and tirofiban includes a recommendation for a heparin dose of 100 u/kg to achieve an ACT of 300–350 sec, a weight-adjusted heparin bolus is recommended. In the ESPRIT trial, in which

patients were treated with a double bolus followed by an infusion of eptifibatide, the heparin bolus was 60 u/kg to achieve a target ACT of 200–300 sec.

Indications.

- Angina for PCI, unstable angina, acute myocardial infarction
- Following anterior wall myocardial infarction, 7–10 days until coumadin increases international normalized ratio (INR) to therapeutic levels
- Prevention and treatment of deep venous thrombosis
- Treatment of pulmonary embolism.

Precautions.

Bleeding.

- Keep ACT <300–350 sec
- Use weight-adjusted doses.

Thrombocytopenia.

- Heparin can cause an immune-mediated thrombocytopenia that can lead to a platelet thrombus with stroke, loss of limb, or other ischemic events (heparin-induced thrombocytopenia with thrombosis, HITT)
- During acute treatment, monitor platelet count daily. If platelet falls below 100,000 μl or otherwise falls precipitously or in a sustained fashion, *discontinue heparin*
- There are two types of HITT (Table 5-2). Type I HITT (HITT-1) is due to direct (non-immune-mediated) platelet activation, with mild thrombocytopenia and a benign clinical course. Type II HITT (HITT-2) is due to immune-mediated platelet activation, with moderate or severe thrombocytopenia and serious thromboembolic complications. Platelet transfusions should not be used to treat HITT due to increased thrombotic complications. Anticoagulation in HITT-2 patients has also been achieved with viper venoms. Low-molecular-weight heparin may reduce but not eliminate the risk of HITT-1, but is contraindicated in patients with prior HITT-2. The direct acting thrombin antagonists lepirudin and argatroban are alternatives to using heparin.

Heparin as a Bridge to Warfarin Therapy.

- Heparin 40–70 u/kg IV bolus followed by 1000–1700 u/h IV infusion
- Obtain ACT at 4–6 h and keep ACT between 200 and 300 sec

Table 5-2

Heparin-Induced Thrombocytopenia

	Type I Heparin-Induced Thrombocytopenia	Type II Heparin-Induced Thrombocytopenia
Incidence	10%	Rare
Mechanism	Direct platelet aggregating effect of heparin	Autoantibody (IgG or IgM) directed against platelet factor IV–heparin complex
Onset	Early (1–5 days)	Later (>5 days); may occur sooner if prior heparin exposure
Platelet count	50,000–150,000/mm^3	<50,000/mm^3
Duration	Transient; often improves even if heparin is continued	Requires discontinuation of *all* heparin; gradual recovery in platelet count over 1–5 days in most patients
Clinical course	Benign	Recalcitrant venous and arterial thromboses and thromboembolism; may be fatal
Heparin	Unfractionated or low-molecular-weight heparin may be continued	Argatroban and lepirudin are FDA-approved

Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*. Birmingham, MI: Physicians' Press, 1999.

- Start warfarin on day 1 at no more than 10 mg a day × 3
- Obtain INR and platelet count
- Give heparin and warfarin jointly with heparin until INR is 2.0–3.0, then stop heparin
- Continue warfarin at an INR of 2.0–3.0.

Managing Bleeding in Patients Receiving Heparin.

Minor Bleeding (Puncture Site, gums, etc.).

- Discontinue heparin
- Monitor vital signs, activated partial thromboplastin time (aPTT), hemoglobin, hematocrit, platelet count, ACT

Major Bleeding (Retroperitoneal, Gastrointestinal).

- After bolus only
  —Give protamine sulfate (1% solution) at 1 mg/100 u heparin or approximately 25 mg slow IV infusion over 10 min

—Monitor vital signs, aPTT, hemoglobin, hematocrit, platelet count, ACT
—Give blood transfusions as necessary according to transfusion guideline (see Chapter 4)

- For patients receiving intravenous heparin infusion:
  —Discontinue infusion
  —Give protamine sulfate (1% solution) at 1 mg/100 u heparin or approximately 25 mg slow IV infusion over 10 min
  —Repeat ACT in 20 min.
- For patients receiving large doses (>5000 u) of subcutaneous heparin:
  —Give protamine sulfate (1%) solution (1 mg/200 u heparin) slow IV infusion over 10 min
  —Repeat aPTT/ACT in 20 min and 1 h
  —It may be necessary to repeat the protamine sulfate infusion after 1 h because of the slow absorption of subcutaneous heparin
  —Observe for protamine reaction.
  (Protamine sulfate can cause severe anaphylactoid-like reactions with back pain, hypotension, and chills. Use protamine only when severe bleeding warrants it. Have resuscitation equipment available. Morphine (2–4 mg IV) is helpful for chills.)

Low-Molecular-Weight Heparin

Low-molecular-weight heparins (LMWHs) are fractionated to have molecular weights between 3000 and 7000, in contrast to standard heparin (3000–30,000). LMWHs have features distinct from unfractionated heparin (UFH), including:

- More predictable anticoagulation effect
- Lack of inhibition by platelet factor 4
- Lack of need for monitoring
- Lower risk of HITT.

Table 5-3 compares LMWHs with UFH.

Absorption and Clearance.

- Peak plasma anti-Xa levels achieved 3–4 hours after subcutaneous dosing and detectable for up to 12 hours
- Eliminated via the kidneys and caution should be used for patients with creatinine clearance <30 ml/min.

Table 5-3

Low-Molecular-Weight Heparin Versus Unfractionated Heparin

Characteristic	Unfractionated Heparin	Low-Molecular-Weight Heparin
Composition	Heterogeneous mix of polysaccharides; molecular weight 3000–30,000	Homogeneous glycosaminoglycans; molecular weight 4000–6000
Mechanisms	Activates antithrombin III*; equivalent activity against factor Xa and thrombin; releases TFPI from endothelium; unable to inactivate clot-bound thrombin or FDP; inactivates fluid phase thrombin	Less activation of antithrombin III; greater activity against factor Xa than thrombin; releases TFPI for endothelium; unable to inactivate clot-bound thrombin or FDP; weaker inactivation of fluid-phase thrombin
Pharmacokinetics	Variable binding to plasma proteins, endothelial cells, and macrophages leads to unpredictable anticoagulant effects (less available to interact with antithrombin III); half-life is short	Minimal binding to plasma proteins, endothelial cells, and macrophages leads to predictable anticoagulation; longer half-life
Laboratory monitoring	Unpredictable anticoagulant effects; use aPTT or ACT	Unable to use aPTT or ACT except in renal failure to body weight <50 kg or >80 kg; use anti-factor-Xa levels
Clinical uses	Venous thrombosis; unstable angina, acute myocardial infarction, ischemic stroke, percutaneous coronary interventions	Venous thrombosis in surgery and trauma patients, unstable angina, ischemic stroke. No advantage during percutaneous intervention
Reversal	Protamine neutralizes antithrombin activity	Protamine neutralizes antithrombin activity but only partially verses anti-factor-Xa activity
HIT-2 heparin-immune thrombocytopenia	Should not be used in patients with a history of HIT-2	Should not be used in patients with a history of HIT-2
Cost	Inexpensive	10–20 times more expensive than unfractionated heparin

* Antithrombin III is now commonly referred to as antithrombin.

ACT, activated clotting time; aPTT, activated partial thromboplastin time; HIT, heparin-induced thrombocytopenia; TFPI, tissue factor pathway inhibitor; FDP, fibrin degradation products.

Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*. Birmingham, MI: Physicians' Press, 1999.

Route of Administration. Intravenous or subcutaneous, not intramuscular.

Duration of Action. Half-life 2–4 hours longer than UFH.

Frequency of Dosing.
- 1 mg/kg subcutaneously every 12 hours
- May be preceded by a 30 mg intravenous bolus.

Monitoring.
- Routine monitoring not currently indicated in most cases
- Anti Xa levels are the gold-standard for drug activity.
- Low-molecular-weight heparins have a very predictable antithrombotic effect, which makes monitoring and dose adjustment unnecessary. When given subcutaneously, LMWH has a longer plasma half-life than standard heparin. Currently, studies have shown a range of clinical results, with some showing strong efficacy and others using a different agent showing more modest effects. This has made it difficult to extrapolate between specific LMWHs and it is unclear whether all LMWHs are equivalent.
- Low-molecular-weight heparins have been shown to be more effective than UFH in the prevention of recurrent myocardial infarction and recurrent angina when used in the treatment of acute coronary syndromes. LMWHs are reasonable alternatives to UFH for PCI but they have not been shown to be superior to UFH during PCI for preventing ischemic complications (Table 5-4). The most commonly used LMWH, enoxaparin, is safe and effective, and may be a reasonable alternative to UFH for PCI anticoagulation. The NICE-3 study demonstrated that patients with acute coronary syndromes receiving a glycoprotein IIb/IIIa antagonist and subcutaneous enoxaparin could safely undergo PCI without UFH. The major issues with respect to increased use of LMWHs with PCI appear to be the uncertainty of plasma activity of the drug, whereas the ACT has been a long-time hallmark of UFH activity. Furthermore, protamine administration is currently thought to reverse about 60% of the drug's anti-Xa level. Clinical studies are ongoing to further define the role of LMWH within the catheterization lab.

Table 5-4

Low-Molecular Weight Heparin Preparations

Preparation	Indications
Dalteparin (Fragmin)	Venous thrombosis, unstable angina (120 u/kg SC bd; max 10,000 u)
Enoxaparin (Lovenox)	Venous thrombosis, unstable angina (1 mg/kg SC bd); PTCA (1 mg/kg IV)
Tinzaparin (Innohep)	Venous thrombosis (175 anti-Xa units/kg SC qd)
Ardeparin (Normiflo)	Venous thrombosis (50 u/kg SC bd)
Danaparoid (Orgaran)*	Venous thrombosis (750 anti-Xa units SC bd)

Different preparations are not interchangeable; each is classified as a distinct drug by the Food and Drug Administration.
* Low-molecular-weight heparinoid.
Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*. Birmingham, MI: Physicians' Press, 1999.

Warfarin

Mode of Action.

- Racemic sodium warfarin is a coumarin derivative
- The agent acts by inhibiting the gamma carboxylation of glutamic acid residues in the clotting proteins II (prothrombin), VII, IX, X.

Absorption and Clearance.

- Absorption is rapid and nearly complete
- Warfarin is cleared from the blood and taken up by the liver over several hours.

Route of Administration.

- Oral, daily.

Duration of Action.

- Daily warfarin takes 4–7 days to produce a therapeutic INR
- Large loading doses do not markedly shorten the time to achieve a full therapeutic effect.

General Recommendations for Warfarin Use.

- Initiate therapy with either the estimated daily maintenance dose (2–5 mg) or, if a larger initial dose is chosen, start with no more than 10 mg
- Elderly or debilitated patients often require low daily doses of warfarin (2–3 mg)

- 4–5 days is required after any dose change or any new diet or drug interaction to reach the new antithrombotic steady state.

Indications.

- Long-term secondary prevention for stroke after myocardial infarction (lifetime in enteric-coated aspirin failures)
- Atrial fibrillation (lifetime)
- Mechanical heart valves (lifetime)
- Tissue heart valves (4 to 6 weeks, then start enteric-coated aspirin)
- Long-term treatment of pulmonary embolus/deep venous thrombosis (3–6 months).

Contraindications and precautions.

Pregnancy.

- Warfarin is contraindicated during any stage of pregnancy because of its teratogenic and fetopathic effects
- Obtain a pregnancy test before starting women of childbearing potential on warfarin.

Purpura.

- This rare skin and subcutaneous necrosis has been seen in a few individuals during the first few weeks of therapy with warfarin
- The condition seems to be linked to protein C deficiency.

Dietary and Drug Interactions with Warfarin.

Patients Taking Warfarin Should Eat a Diet that is Constant in Vitamin K.

- Minimize changes in intake of green leafy vegetables (spinach, greens, and broccoli), green peas, and oriental green tea. Conditions that interfere with vitamin K uptake or interfere with liver function will increase the warfarin effect
- Expect a longer prothrombin time in patients with congestive heart failure, jaundice, hepatitis, liver failure, diarrhea, or extensive cancer or connective tissue disease
- Expect a longer prothrombin time when patients receiving warfarin are hospitalized for any reason.

Metabolic Alterations Can Affect the Prothrombin Time.

- Expect a longer prothrombin time in patients with hyperthyroidism or high fever
- Expect a shorter prothrombin time in patients with hypothyroidism.

Direct Thrombin Inhibitors

Direct thrombin inhibitors are polypeptide or low-molecular weight-inhibitors (Box 5-2). Polypeptide inhibitors such as hirudin and bivalirudin inactivate circulating thrombin at the active binding site and clot-bound thrombin at exosite-1. Low-molecular-weight inhibitors such as argatroban inactivate circulating thrombin at the active binding site but do not inactivate clot-bound thrombin.

Unlike heparin, hirudin and bivalirudin do not require antithrombin for anticoagulant effect, form highly stable noncovalent complexes with circulating and clot-bound thrombin, and are not inhibited by platelet factor 4.

Fewer ischemic and bleeding complications have been reported with bivalirudin in high-risk patients with postinfarction angina.

Box 5-2

Direct Thrombin Inhibitors

Polypeptide Inhibitors
- Hirudin (lepirudin)*,†
- Bivalirudin

Low-Molecular-Weight Inhibitors
Noncovalent
- Argatroban†
- Napsagatran
- Inogatran
- Melogatran

Reversible-covalent
- Efegatran
- Boro-arginine derivatives

*Derived from medicinal leech saliva and available by recombinant DNA technology as lepirudin. †FDA-approved for patients with HIT-2 who require anticoagulation.

A higher incidence of intracranial hemorrhage has been reported in three trials combining hirudin and thrombolytic therapy (GUSTO IIa, TIMI 9A, HIT-III).

In the USA, lepirudin and argatroban are approved for use in heparin-induced thrombocytopenia (HIT). In such patients, lepirudin is administered as an initial bolus of 0.4 mg/kg (maximum 44 mg) over 15–20 seconds, followed by a continuous infusion of 0.15 mg/kg/h (maximum rate 16.5 mg/h). Monitoring is accomplished using the same aPTT/ACT guidelines as for UFH. Bivalirudin is approved for procedural anticoagulation in unstable angina. Table 5-5 compares UFH with direct thrombin inhibitors.

Table 5-5

Comparison of Unfractionated Heparin and Direct Thrombin Inhibitors

	Unfractionated Heparin	Direct Thrombin Inhibitors (Hirudin, Bivalirudin)
Effect on clot-bound thrombin, FDP	None	Inactivation
Effect on antithrombin	High-affinity interaction; inhibits thrombin and factor Xa	High affinity interaction
Effect on factor Xa bound to platelets	None	Inactivation
Binding to endothelium and plasma proteins	High; results in less heparin availability to activate antithrombin	None
Binding to PF-4	High affinity	None
Anticoagulant effects	Highly variable	Predictable
Laboratory monitoring	Essential	May be unnecessary with bivalirudin

Summary: Direct thrombin inhibitors have biologic pharmacokinetic advantages compared to heparin. The biologic advantage reflects their ability to inactivate clot-bound thrombin via exosite 1 (polypeptide inhibitors), whereas the pharmacologic advantages produce more predictable anticoagulant effects without the need for intensive laboratory monitoring (especially bivalirudin), by less binding to endothelial and plasma proteins. Bivalirudin may block procoagulant activity associated with eptifibatide and tirofiban.

FDP, fibrin degradation products; PF-4, platelet factor-4.

Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*. Birmingham, MI: Physicians' Press, 1999.

Antiplatelet Agents

Aspirin.

Mode of Action. Acetylates and inactivates platelet cyclooxygenase, inhibiting production of thromboxanes, which are potent inducers of platelet aggregation and vasoconstrictors.

Route of Administration. Oral.

Absorption and Clearance. Rapid absorption, peak plasma levels in 20 min, rapid clearance.

Duration of Action. Days (for the lifetime of the platelet).

Frequency of Dosing
Daily or every other day (Table 5-6).

Monitoring. None is routinely used. The template bleeding time can be used to gauge aspirin's effect on platelet function.

Indications.
- Stable angina
- Unstable angina
- Acute myocardial infarction
- Coronary angioplasty
- Primary and secondary prevention of myocardial infarction
- Carotid or primary cerebrovascular disease (stroke prevention)
- Peripheral vascular disease
- Atrial fibrillation (not as effective as warfarin; use when warfarin is contraindicated)
- Prosthetic heart valves (adjunctive therapy with warfarin).

Table 5-6

Effective Doses of Aspirin

Before and after coronary angioplasty	325 mg qd
Acute myocardial infarction	160 mg qd (chewed)
Unstable angina	75 mg qd
Stable angina	325 mg qd
Primary prevention of myocardial infarction	325 mg qd
Secondary prevention of myocardial infarction	160 mg qd
Peripheral vascular disease	325 mg qd
Stroke prevention (after transient ischemic attack)	30 mg qd
Mechanical heart valves (adjunctive to warfarin)	100 mg qd

Precautions.
- Aspirin allergies (asthma)
- Active peptic ulcer disease or other bleeding predispositions.

Clopidogrel.

Route of Administration. Oral.

Absorption and Clearance. Rapid absorption, peak plasma level in 2 hours, plasma half-life of 12 hours, steady-state drug levels in 14–21 days.

Mechanism of Action. Unidentified metabolite interferes with platelet membrane function by inhibiting adenosine diphosphate (ADP)-induced platelet-fibrinogen binding and platelet-to-platelet interactions.

Duration of Action. Days (for the lifetime of the platelet).

Dose and Interval. Clopidogrel 300 mg PO load then 75 mg PO daily.

Monitoring.
- No direct monitoring
- Because of neutropenia and/or TTP with ticlopidine and, rarely, clopidogrel, obtain complete blood count and white blood count differential at 2 weeks then at 3 months of therapy.

Indications. Clopidogrel is indicated for prevention of stent thrombosis and in patients who cannot take aspirin or fail aspirin therapy for stroke prevention.
- Stroke prevention in patients with risk factors for cerebrovascular accident
- Secondary prevention after completed stroke.

Precautions.
- Neutropenia—occurs in 1–3% of patients in the first 3 months of therapy. Monitor complete blood count and white blood count differential every 2 weeks for the first 3 months
- Not recommended in patients with severe liver disease
- Dosage reduction may be necessary in patients with renal insufficiency. Monitor template bleeding time.

Platelet Glycoprotein IIb/IIIa Receptor Blockers. Tables 5-7 and 5-8 summarize the use of glycoprotein receptor blockers for PCI.

Table 5-7

Platelet Glycoprotein IIb/IIIa Antagonists for Coronary Intervention

	Abciximab	Eptifibatide	Tirofiban
Dose for PCI	0.25 mg/kg IV bolus plus 0.125 μg/kg/min (maximum 10 μg/min) IV infusion for 12 h. Low-dose heparin and early sheath removal to minimize bleeding. For patients with unstable angina planning to undergo PCI within 24 h, bolus plus infusion of abciximab (PCI dose) can be started up to 24 h prior to PCI and continued at the same rate until 1 h after the procedure	*Acute coronary syndromes (PURSUIT dose)*: 180 μg/kg IV bolus plus 2.0 μg/kg/min IV infusion. If arrive in cath lab >4 h after initiating therapy, no additional bolus is required. *Percutaneous intervention (ESPRIT dose):* 2 × 180 μg/kg/min IV bolus 10 min apart, plus 2.0 μg/kg/min IV infusion for 18–24 h	10 μg/kg IV bolus (over 3 min) immediately prior to PCI followed by an infusion of 0.15 μg/kg/min for 18–24 h. Patients with creatinine clearances <30 ml/min should receive half the usual infusion rate
Heparin (unfractionated)	Maintain ACT at 200–250 sec to minimize bleeding. Initial IV heparin dose based on ACT: ACT (sec) Heparin (bolus) <150 70 u/kg 150–199 50 u/kg > 200 No additional Discontinue heparin immediately after PCI	100 u/kg bolus, titrate to ACT 300–350 sec. May also consider lower doses, as recommended for abciximab. In ESPRIT, the recommended initial heparin dose was 60 u/kg to achieve a target ACT of 200–300 sec	100 u/kg bolus, titrate to ACT 300–350 sec. May also consider lower doses, as recommended for abciximab
Aspirin	325 mg started at least 1 day prior to PCI and continued indefinitely; four chewable baby aspirin (325 mg total) for urgent intervention. For stents, add clopidogrel 300 mg oral load, then 75 mg PO daily for 2–4 weeks	See abciximab	See abciximab

ACT, activated clotting time; PCI, percutaneous coronary intervention.

Modified from Safian R, Grines C, Freed M. *Manual of interventional cardiology*, Birmingham, MI: Physicians' Press, 1999.

THROMBOLYTIC AGENTS

Mechanisms of Action and Pharmacologic Properties

Recognition that acute coronary thrombosis is the primary cause of acute myocardial infarction led to the use of plasminogen activators to achieve rapid thrombolysis. Thrombolytic agents are proteins that convert a plasma proenzyme, plasminogen, to the active enzyme plasmin. Plasmin then solubilizes fibrin and degrades a number of other plasma proteins, most notably fibrinogen. All the currently available thrombolytic (fibrinolytic) agents are plasminogen activators. They all work enzymatically, directly or indirectly, to convert the single-chain plasminogen molecule to the double-chain plasmin (which has potent intrinsic fibrinolytic activity).

Clinical Trials

Thrombolytic therapy provides a survival benefit for patients with acute myocardial infarction, based on large, well-controlled clinical trials. Benefit has been shown individually for therapy with streptokinase, anistreplase, and alteplase. In an overview of nine controlled randomized trials involving more than 1000 patients, a highly significant ($p < 0.00001$) 18% proportional reduction in mortality was observed, corresponding to the avoidance of 18 deaths per 1000 patients treated. The largest of these studies (ISIS-2, more than 17,000 patients), showed that, when aspirin was combined with streptokinase and treatment was given within 4 hours of the onset of symptoms, an odds reduction in mortality of 53% was achieved (control, 13.1%; streptokinase plus aspirin, 6.4%; $p < 0.0001$).

After GUSTO-I, several clinical applications of the risk: benefit ratio have attempted to compare the cost:benefit ratio, risk of cerebral bleeding, and mortality benefits in subgroups of patients. Alteplase (tPA) appears to have the greatest benefit in patients with large infarctions and appears to pose a low risk of ICH in younger patients who present early.

Common Agents

Tissue Plasminogen Activator (t-PA). Derived by recombinant genetics from human DNA. Fibrin specific. Activates plasminogen associated with fibrin directly by enzymatic action. Plasma half-life 5 minutes.

Table 5-8

Multicenter Randomized Studies Involving Glycoprotein Blockers for Short-Term Events After Percutaneous Coronary Intervention

Study	Study Design	Definition of MI	Followup period	Incidence of MI (%)
CAPTURE	Placebo vs abciximab in patients with refractory unstable angina undergoing PCI 18–24 h after randomization	↑ CK or CK-MB ≥ 3 × ULN in two separate blood samples and increased 50% over the previous value, or new pathological Q waves	30 days*	Placebo = 8.2 Abciximab = 4.1
EPIC	Placebo vs c7E3 Fab bolus vs c7E3 Fab bolus and infusion	↑ CK-MB ≥ 3 × ULN or new pathological Q waves (different enzymatic criteria existed for those with baseline elevated CK-MB)	30 days	Placebo = 8.6 C7E3 Fab bolus = 6.2 C7E3 Fab bolus and infusion = 5.7
EPILOG	Abciximab vs Placebo in balloon angioplasty	↑ CK or CK-MB ≥ 3 × ULN; or new Q Waves	30 days	Placebo = 8.7 Abciximab + low-dose heparin = 3.7 Abciximab + high-dose heparin = 3.8

EPISTENT	Stent + placebo vs balloon angioplasty + abciximab vs stent + abciximab	↑ CK-MB ≥ 3 × ULN	30 days	Stent + placebo = 9.6 Balloon + abciximab = 5.3 Stent + abciximab = 4.5
ESPRIT	Placebo vs eptifibatide during coronary stenting	↑ CK-MB ≥ 3 × in two separate blood draws	48 hours	Placebo = 9.0 Eptifibatide = 5.4
IMPACT-II	Placebo vs eptifibatide in patients undergoing balloon angioplasty	↑ CK-MB ≥ 3 × ULN or new pathological Q waves	30 days	
RESTORE	Placebo vs tirofiban during balloon angioplasty or directional coronary atherectomy	↑CK or CK-MB ≥ 3 × ULN†	30 days	Placebo = 5.7 Tirofiban = 4.2
TARGET	Tirofiban vs abciximab during coronary stenting	CK-MB ≥ 3 × ULN in two separate blood samples, or pathological Q waves	30 days	Tirofiban = 6.9 Abciximab = 5.4

Re-analyzed; final results from the Bivalirudin Angioplasty Study.
* Includes MIs in the 18–24 hours pre-PCI. † Multiple additional definitions of MI were also used.
MI, myocardial infarction; PCI, percutaneous coronary intervention; ULN, upper limit of normal.

Anisoylated Plasminogen-SK Activator Complex. Derived by anisoylating human plasminogen to standard SK. Weakly fibrin-specific. Complex activates adjacent plasminogen. Plasma half-life of 90 minutes.

TNK.

Doses of Thrombolytics

r-PA. This is a nonglycosylated deletion mutation of t-PA (contains 355–527 of the amino acids that t-PA contains). Dose: 10 u + 10 u double bolus injection. Each bolus is given intravenously over 2 minutes. The second bolus is given 30 minutes after the first (supplied as a kit of two single-use vials).

APSAC (Anisoylated Plasminogen Streptokinase Activator Complex). Dose: 30 u IV over 5 min.

TNKase (TNK-tPA). Dose: Based on weight, <60 kg, 30 mg IV over 5 min, maximum 50 mg. Weight 60–69 kg, 35 mg IV, maximum 50 mg. Weight >70 kg, 40 mg IV, maximum 50 mg.

Precautions and Contraindications (Box 5-3)

- Bleeding is the major complication of thrombolytic therapy. Consequently, absolute contraindications include dissecting aortic aneurysm, pericarditis, stroke, or neurosurgical procedures within 6 months of known intracranial neoplasm
- Relative contraindications include major surgery or bleeding within 6 weeks, known bleeding diathesis, and severe uncontrolled hypertension
- Allergic reactions—anisoylated plasminogen streptokinase activator complex is potentially allergenic. Patients are usually pretreated with 100 mg of intravenous hydrocortisone
- t-PA induces antibody production, which makes treatment with either of these agents less effective.

MONITORING COAGULATION

International Normalized Ratio (INR) Considerations

Definition. The INR is the prothrombin time ratio that would have been obtained had the World Health Organization (WHO) international reference thromboplastin standard been used. The INR is important for chronically anticoagulated

Box 5-3

Contraindications and Cautions for Thrombolytic Use in Myocardial Infarction

Contraindications

- Previous hemorrhagic stroke at any time, other strokes or cerebrovascular events within 1 year
- Known intracranial neoplasm
- Active internal bleeding (does not include menses)
- Suspected aortic dissection

Caution/relative contraindications

- Severe uncontrolled hypertension on presentation (blood pressure >180/110 mm Hg)*
- History of prior cerebrovascular accident or known intracerebral pathology not covered in contraindications
- Current use of anticoagulants in therapeutic doses (INR 2–3); known bleeding diathesis
- Recent trauma (within 2–4 weeks) including head trauma or traumatic or prolonged (>10 min) CPR or major surgery (3 weeks)
- Noncompressible vascular punctures
- Recent (within 2–4 weeks) internal bleeding
- For streptokinase/anistreplase: prior exposure (especially within 5 days to 2 years) or prior allergic reaction
- Pregnancy
- Active peptic ulcer
- History of chronic severe hypertension

These contraindications and cautions are viewed as advisory for clinical decision making and may not be all-inclusive or definitive.

* Could be an absolute contraindication in low-risk patients with myocardial infarction.

INR, international normalized ratio; CPR, cardiopulmonary resuscitation.

Reproduced with permission from Ryan TJ, Antman EM, Brooks NH, *et al.* ACC/AHA guidelines for the management of patients with acute myocardial infarction: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Management of Acute Myocardial Infarction). *J Am Coll Cardiol* 1999;34:890–911.

patients because commercially available thromboplastin reagents give quite different prothrombin times when a patient is taking warfarin. The international sensitivity index (ISI) of a thromboplastin relates it to an international reference thromboplastin. The use of a thromboplastin reagent with an

ISI as close to 1.0 as possible, as recommended by the WHO, will further reduce the variability of the INR results. The INR is the most reliable way to compare prothrombin time measurements performed in different laboratories:

$$\text{INR} = \frac{(\text{patient's prothrombin time in seconds})^{\text{ISI}}}{\text{mean normal prothrombin time in seconds}}$$

The INR is the most reliable way to measure the anticoagulant effect of warfarin in stable patients on long-term therapy. The INR was not designed to interpret prothrombin times that are used for evaluation of liver function or a bleeding abnormality.

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6

NON-BALLOON PERCUTANEOUS CORONARY INTERVENTIONAL DEVICES: ROTATIONAL ABLATION CATHETER, DIRECTIONAL ATHERECTOMY, THROMBUS ASPIRATION SYSTEMS, DISTAL PROTECTION DEVICES, AND THE CUTTING BALLOON

Souheil Khoukaz and Morton J. Kern

Balloon angioplasty has given way to stenting as the primary modality of percutaneous cardiac intervention (PCI). However, because the amount of atherosclerotic plaque in the artery may influence outcomes, physical removal of the plaque from inside the artery, atherectomy (*athero*, "plaque"; *ectomy*, "cut"), was thought to improve the results of PCI. Although this promise was not fulfilled, two devices developed for the purpose of plaque removal remain in current practice: the high-speed rotational ablation catheter (Rotablator) and the directional atherectomy catheter (DCA).

None of the devices directed at plaque modification and removal can remove thrombus very well. Thus, additional non-balloon devices include thrombus aspiration catheters designed to treat arteries with significant amounts of thrombus complicating PCI and/or acute myocardial infarction. The most practical and often used device is the Possis AngioJet®

Thrombus Aspiration system. Each device has unique mechanisms and specific indications.

ROTATIONAL ATHERECTOMY (ROTABLATOR)

The Rotablator is made of an olive-shaped steel burr (1.25–2.5 mm in diameter) that is embedded with microscopic diamond particles on the front half and is rotated with a torque wire at ≤200,000 rpm by an external air turbine (Fig. 6-1). The device is inserted through 6–10 French guide catheters over a special 0.009 inch steel guidewire. A continuous pressurized heparin, saline, and emulsifier mixture infusion is flushed around the device drive shaft to aid lubrication and heat dissipation.

Indications

Nearly all percutaneous transluminal coronary rotational atherectomy (PTCRA) procedures are combined with balloon and stenting in suitable-sized vessels. PTCRA as a sole therapy or with adjunctive balloon angioplasty is rarely used. PTCRA is indicated in patients with coronary artery disease, which may include:

- Single-vessel atherosclerotic coronary artery disease with a calcified stenosis that can be passed with a guidewire
- Low-risk, multiple-vessel coronary artery disease
- Native vessel atherosclerotic coronary artery disease less than 25 mm in length
- Complementary or adjunctive balloon therapy is used 85% or more of the time; larger vessels (>2.5 mm) may require the use of complementary or adjunctive balloon dilation more frequently than smaller vessels, since the largest Rotablator burrs are 2.50 mm in diameter

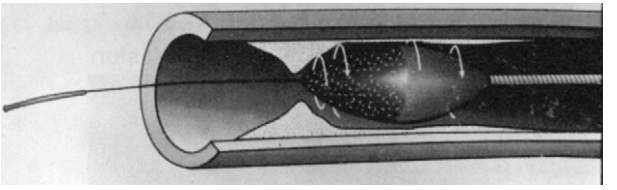

Fig. 6-1 Diagram of the rotational ablation catheter. (From the Rotablator manual of operations, courtesy of Boston Scientific Corporation, Boston, MA.)

- Undilatable lesions, when it is impossible to cross and to dilate the lesions with a balloon
- Ostial lesions, especially aorto-ostial lesions.

High-Risk Rotablator

Operators should be aware of the higher-risk conditions for PTCRA and the current lack of scientific evidence for these applications. High-risk conditions and patients include:

- Patients who are not candidates for coronary artery bypass surgery
- Patients with severe, diffuse, three-vessel disease (multiple diseased vessels should be treated in separate sessions)
- Patients with unprotected left main coronary artery disease
- Patients with ejection fraction less than 30%
- Lesions more than 25 mm in length
- Angulated (≥45°) lesions.

Contraindications

Contraindications to the use of the Rotablator include:

- Occlusions through which a guidewire will not pass
- Last remaining vessel with compromised left ventricular function
- Saphenous vein grafts
- Angiographic evidence of thrombus
- Angiographic evidence of significant dissection at the treatment site (the patient may be treated conservatively for approximately 4 weeks to permit the dissection to heal before treating the lesion with the Rotablator system).

Complications

Complications associated with the use of the Rotablator system have been compiled from a multicenter registry. PTCRA is associated with the following procedural complications.

Clinical Events.

- Access-site bleeding of significance (1.9%)
- Distal embolization (0.3%)
- Intimal dissection (13.7%)
- Acute vessel closure (4.0%)
- Vessel perforation or tear (0.7%)
- Emergency surgery; vascular repair, or bypass (2.5%)

- Contrast reaction (0.07%)
- Stroke (0.0.%)
- Myocardial infarction (1.1%)—creatine phosphokinase (CPK) enzyme elevation without myocardial infarction 13.0%
- Arrhythmia requiring treatment (2.7%)
- Cardiac tamponade (0.1%)
- Death (1.0%).

Angiographic Complications.

- No reflow (0–7%)
- Intimal dissection with dye stain (3%)
- Intimal dissection without dye stain (11%)
- Acute vessel closure (5%)
- Vessel perforation or tear (1%)
- Arrhythmia (3%).

No or Slow Flow After Rotablator Ablation

No/slow flow is the occurrence of no blood flow (no flow) or blood flow reduced by one angiographic thrombolysis in myocardial infarction study (TIMI) flow grade (slow flow) in the treated artery despite the fact that the treated segment is patent. No/slow flow is believed to occur because of the transient increase in blood viscosity due to the presence of

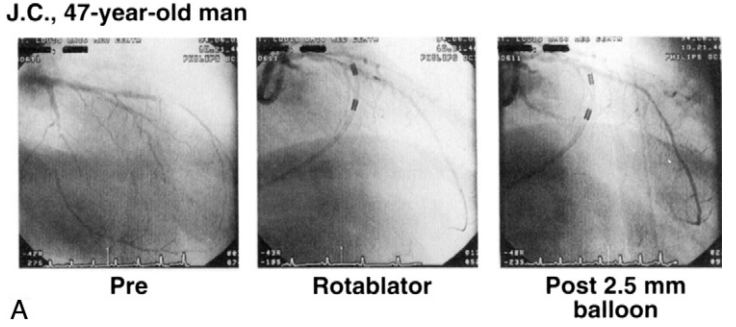

Fig. 6-2 Case example of rotational ablation. The mid left anterior descending artery has a calcified 99% stenosis associated with reversible ischemia after myocardial infarction. Long-standing coronary artery disease in the circumflex was not symptomatic. **A,** Angiograms of left coronary artery before (left), during Rotablator 1.75 mm burr (middle), and after Rotablator (right).

Continued

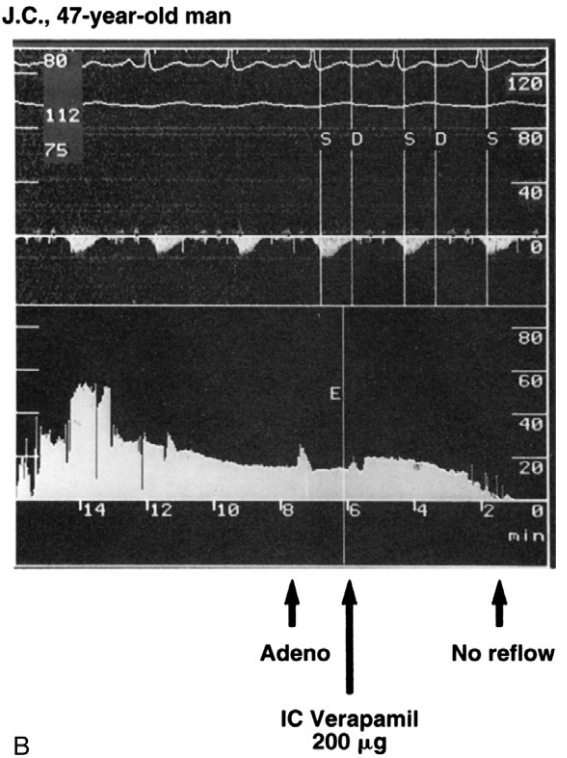

Fig. 6-2, cont'd **B,** Although the initial Rotablator result was satisfactory, angiographic and measured flow velocity (Doppler guidewire velocity trend) showed slow then no reflow, subsequently treated with balloon inflations, intracoronary nitroglycerin, and verapamil. Flow was restored and the procedure completed without complication. Flow velocity trend scale 0–100 cm/sec. (From Khoury AF, Bacj RG, Kern MJ. Influence of adjunctive balloon angioplasty on coronary blood flow after rotational atherectomy. *Cathet Cardiovasc Diagn* 1995;35:272–276.)

microparticles or vasospasm at the level of the distal microvasculature. No/slow flow has been observed in 6–7% of PTCRA patients (Fig. 6-2). No/slow flow can be minimized by:

- Advancing the burr slowly
- Using a stepped burr approach (smaller, then larger) in long or calcified lesions
- Using a "pecking" motion at the plaque to avoid blocking the arterial lumen

- Maintaining maximum blood flow by repeated flushing with saline (bolus of 10–30 ml)
- Using guide catheters with side holes
- Maintaining the left ventricular filling pressures (and mean arterial pressure) by increasing the patient's volume status appropriately.

No/slow flow generally resolves within a short period of time (<15 min) with or without the use of nitroglycerin. Intracoronary verapamil (200 μg) or nitroprusside (50–100 μg) has been reported to improve no/slow reflow.

Equipment Needed for Rotational Ablation

- Rotablator burr
- Turbine motor drive unit
- Heparinized, pressurized, warm flush solution
- Type A, C Rotablator wire
- Guidewire clip
- 6–10F guide catheter and arterial sheath
- Exchange catheter/guidewire
- Temporary pacemaker may be withheld if using aminophylline pretreatment, 250–500 mg bolus IV before Rotablator minimizes heart block.

The PTCRA system (Fig. 6-3) uses a high-speed, rotating, diamond-coated burr to ablate occlusive material and restore luminal patency. The burr tracks over a specially designed 0.009 inch (0.23 mm) guidewire that can be independently advanced and steered. The guidewire comes with a 0.017 inch (maximum) spring tip (0.43 mm inch diameter), facilitating negotiation of the wire through the vasculature.

The Rotablator burr is advanced on an independent extension, permitting accurate positioning and forward motion. A compressed-gas turbine located within the Rotablator advancer spins the flexible drive shaft and the diamond-coated burr. It is designed with minimal rotational mass so that it can be started and stopped abruptly.

Components of the Percutaneous Transluminal Coronary Rotational Atherectomy System

The seven main components of the PTCRA are the guidewire, the drive shaft and burr, the drive shaft sheath, the advancer, the control console, the foot pedal, and the compressed-gas supply.

RotaLink™ System Components

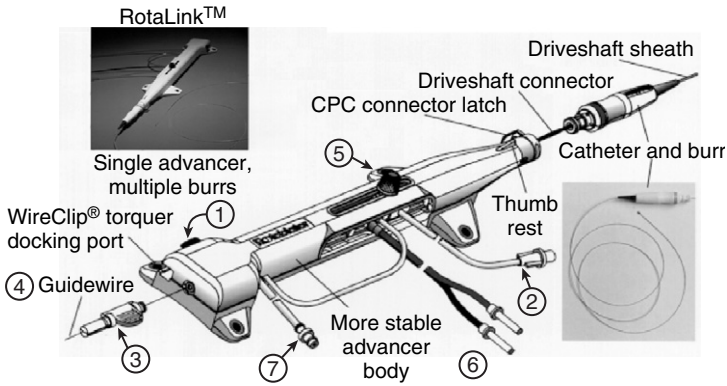

Fig. 6-3 Rotablator advancer unit with removable catheter and burr attachment. 1, Brake defeat knob; 2, Air pressure close; 3, Wire clip; 4, Rotablator wire; 5, Burr position control knob; 6, Fiberoptic tachometer; 7, Pressurized saline infusion port with lubricant. (Courtesy of Boston Scientific Corporation, Boston, MA.)

Rotablator Guidewire. Two types of guidewire are generally used. Type A has a diameter of 0.009 inch (0.23 mm) and has a spring tip (0.17 inch maximum, 0.43 mm inch diameter) and a safety core that extends to the end of the spring tip. Type C is 0.0009 inch (0.23 mm) in diameter and tapers at the distal end, terminating in a flexible, formable platinum spring that is 0.017 inch maximum (0.43 mm) with a safety core that extends to within 0.5 inch (12.71 mm) of the end of the spring. A Rota-floppy guidewire (0.007 inch) is also available as the softest of the Rotablator guidewire. The Rota-floppy guidewire is atraumatic, with a radio-opaque tip, and is the preferred wire. The wire shaft of the guidewires is constructed of polished stainless steel. The tip can be bent or preformed to form a steerable system (Fig. 6-4). The wire clip torquer is specially designed for manipulating the PTCRA wire (Fig. 6-5). Because of the torque responsiveness, the Rotablator guidewire is more difficult to handle than guidewires used in balloon angioplasty.

Burr and Shaft. The diamond-coated burr consists of a tapered body coated with fine diamond abrasive. The burr spins at high

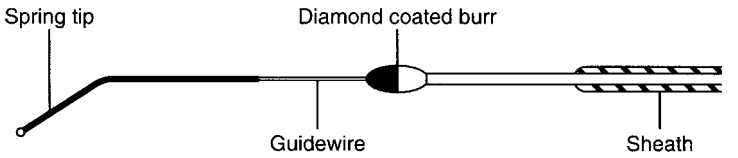

Fig. 6-4 Rotablator guidewire with burr through sheath assembly. (Modified from the Rotablator manual of operations, courtesy of Boston Scientific Corporation, Boston, MA.)

speed and ablates occlusive tissue into fine particles that are carried distally and removed by the reticuloendothelial system. If too large a quantity of particles is generated per unit time, a no- or slow-flow phenomenon may occur. The burr is driven by a flexible helical shaft that has a central lumen to permit passage of the guidewire. The shaft and burr are delivered to the coronary lesion using standard angioplasty guidewire technique. The shaft is capable of transmitting rotary motion at speeds up to 190,000 rpm. Table 6-1 provides recommended advancer turbine speeds.

Burr Shaft Sheath. The sheath is 0.058 inch (1.4 mm) in diameter, constructed of Teflon, and beveled at the tip to allow easy passage in the vessel. The Rotablator sheath can be detached from the front of the advancer body when a bigger burr is needed. The sheath functions are:

- To guide the helical drive from the point of entry to the site of the lesion

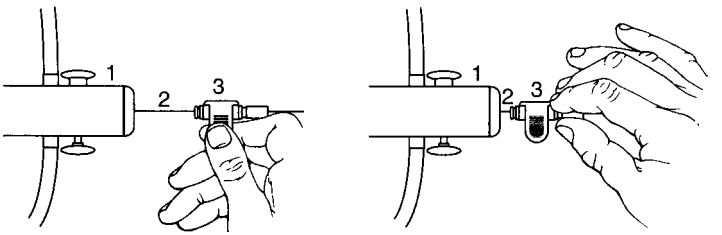

Fig. 6-5 Manipulation of the unique wire Clip on the Rotablator guidewire. (Left panel) Pinching wings of wire Clip permits attachment to guidewire. (Right panel) The end of the wire Clip can then be used as a torque tool to rotate and steer the guidewire in the distal coronary artery. 1, Advancer unit; 2, Rotablator guidewire; 3, Wire Clip. (Modified from the Rotablator manual of operations, courtesy of Boston Scientific Corporation, Boston, MA.)

Table 6-1

Recommended Rotablator Advancer Turbine Speed

Burr size (mm)	Burr size (French)	Design rotational speed range* (rpm)*	Optimum rotational speed range (rpm; no tissue contact)
1.25	3.75	150,000–190,000	180,000
1.50	4.50	150,000–190,000	180,000
1.75	5.25	150,000–190,000	180,000
2.00	6.00	150,000–190,000	180,000
2.15	6.45	140,000–180,000	160,000
2.25	6.75	140,000–180,000	160,000
2.50	7.50	140,000–180,000	160,000

Rotablator Catheter Sheath Outer Diameter		
Size (mm)	**Size (French)**	**Size (inch)**
1.35	4.0	0.058

* Preset speed outside of the body at the higher rotational speed—for example, for a 1.25 mm Rotablator advancer, set speed outside body at 190,000 rpm.
From: Boston Scientific Corporation. *Rotablator operations manual.* Boston Scientific Corporation, Boston, MA.

- To protect the arterial tissue from the spinning drive shaft
- To lubricate the drive shaft.

Burr Advancer. The advancer acts as a support for the air turbine and the control for the sliding elements for burr extension. A brake within the advancer body is designed to hold the guidewire firmly during burr rotation to prevent it from spinning or moving. Manipulation of the burr control knob and the wire clip torquer allows independent extension of the guidewire tip and burr. The use of compressed gas to generate the high rotational speeds necessary for ablation and low inertial mass permits the driving elements to be started and stopped quickly.

Control Console. The console monitors and controls the rotational speed of the burr and provides the operator with continuous performance information during the procedure.

Rotational Ablation Technique

The guide catheter is placed using balloon angioplasty guide catheter technique. Table 6-2 indicates the internal diameter of

Table 6-2

Recommended Guide Catheter Sizes for Use with the Coronary Rotablator

Rotablator burr size (mm)	Recommended guide catheter internal diameter (inch)	Guide size (French)*
1.25	0.053	6–8
1.50	0.063	6–8
1.75	0.073	6–8
2.00	0.083	7–9
2.15	0.089	7–9
2.25	0.093	7–9
2.50	0.102	9–10

* For a given size of catheter, the inside diameter varies from manufacturer to manufacturer. French sizes assume thin-wall (high-volume flow) catheters with side holes.
From: Boston Scientific Corporation. *Rotablator operations manual*. Boston Scientific Corporation, Boston, MA.

the guide catheter required for various burr sizes. 6 French guides use 1.5 mm burrs. 8 French guides use 2.15 mm burrs.

The maximum burr diameter should be no larger than 70–80% of the normal arterial luminal reference segment diameter. Start with a small burr first to create a pilot channel. Short run times (<30 sec) are recommended.

The Rotablator guidewire is positioned across the lesion. In some cases, a 0.014 inch angioplasty guidewire is needed to negotiate diffusely diseased vessels and is then exchanged, using a small tracking catheter, for the Rotablator guidewire. The infusion port on the advancer is connected to a 1 liter pressurized bag of saline and lubricant mixture with macro drip tubing and used to ensure steady infusion against arterial pressure. The recommended pressure is 150–200 mm Hg. The saline mix should flow through the advancer and sheath and exit from the sheath tip running bubble-free.

The combination of verapamil (10 mg/l), nitroglycerin (4 mg/l) and heparin (2000 u/l), when added to the saline flush, reduces spasm. Rotaglide is an egg yoke/olive oil/ethylenediaminetetraacetic acid (EDTA) mixture used to lubricate the drive shaft. It is mixed with saline and is compatible with the antispasm drugs and adenosine. **Warning:** The Rotablator advancer should never be operated without saline infusion.

The burr is advanced through the guide catheter and tested. Be sure the pressure flush is running; burr speed is 160,000–180,000 rpm depending on its size (Table 6-1).

After the burr is positioned immediately proximal to the lesion, advancer tension is released so the burr does not jump forward. The system is activated and the burr is advanced through the lesion in a slow and steady manner. Several burr passes are performed before the burr is removed and decisions are made about whether a larger burr size or complementary balloon inflation is required. Most (>85%) cases require additional angioplasty balloon inflations to decrease the residual stenosis.

Alignment of the coaxial guide and burr to the ostium and the vessel course is important.

Technical Notes and Rotablator Tips.

- A nitrogen compressed-gas cylinder with pressure regulator capable of delivering at least 140 l/min at 90–100 psi is required.
- The compressed-gas cylinder valve must be open to supply compressed gas to the console. The regulator should be adjusted so that it never supplies more than 100 psi.
- Use a glycoprotein IIb/IIIa receptor antagonist when treating heavily calcified lesions in addition to the stepwise burr approach, with short ablation times of less than 30 seconds.
- Angulated lesions and branch ostial narrowings have a higher incidence of dissection and perforation; downsize the initial burrs and stepwise increase size to achieve final result. Use a Rota-floppy guidewire for this application.
- When Rotablating bifurcation lesions, address the branch with the sharpest angle first, followed by the more directly treated vessel. Adjunctive stenting will jail an important side branch and thus full preparation of the side branch prior to stenting may be helpful.
- Chronic total occlusion can be Rotablated only if the guidewire is confirmed to be in the true lumen and is maintained in the distal vessel. IVUS may be required to confirm this condition.
- Perforations are uncommon and are often self-sealing. The covered stent should be available in the cardiac catheterization laboratory to treat perforated vessels after Rotablator.

Clinical Results

Percutaneous transluminal coronary rotational atherectomy is most suitable for rigid, calcified, and long lesions in which balloon angioplasty success is expected to be low. The complication and restenosis rates are similar to balloon angioplasty. A specific complication of PTCRA is temporary no-reflow phenomenon with creatine kinase (CK) enzyme rise (non-Q-wave myocardial infarction) in some patients.

The Rotablator multicenter registry (2953 procedures and 3717 lesions) evaluated the safety and efficacy of PTCRA as a standalone procedure or with adjunctive coronary angioplasty for the treatment of coronary artery stenosis. The clinical data based on the findings from 22 clinical sites as reported to the Food and Drug Administration indicated primary success (defined as a luminal diameter of ≤50% with at least a 20% reduction in overall stenosis and no major complications with or without complementary balloon angioplasty) was 95% with the use of adjunctive balloon angioplasty. The data showed no statistical difference in the overall primary success rate when segmented by lesion characteristics.

Restenosis

The results of most clinical studies indicate that the restenosis rate for patients treated with PTCRA is not different from the restenosis rate obtained for patients treated with balloon angioplasty or other interventional devices. An illustration of a case treated with Rotablator is shown in Figure 6-6.

Recommended Training

This system should be used only by physicians with angioplasty credentials who have received proper training in the technique of rotational angioplasty. The minimum requirements for the primary user include:

- The physician must be accredited and qualified to perform coronary angioplasty at his or her local institution.
- The physician must perform a minimum of 75 coronary angioplasties per year.

DIRECTIONAL CORONARY ATHERECTOMY

Directional coronary atherectomy is performed using a specially designed catheter with a cylindrical metal cutting

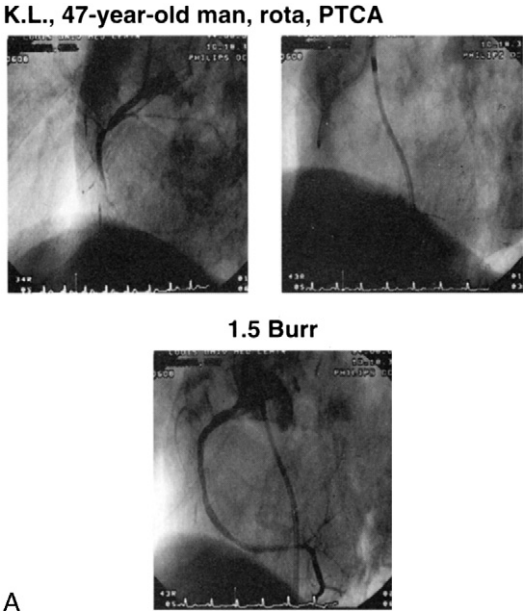

Fig. 6-6 Case example of rotational ablation and percutaneous transluminal coronary angioplasty (PTCA) with intravenous ultrasound (IVUS). **A,** Arteriograms of right coronary artery with 99% calcified stenosis in the mid portion of the vessel. A 1.5 mm burr is used and a 3.0 mm angioplasty balloon produces an acceptable angiographic result.

Continued

chamber that contains a rapidly rotating cylindrical cutter. The cutting chamber is 5–10 mm long and is pushed against the coronary lesion by a supporting balloon located on the opposite side of the cutting chamber opening. The cutter is rotated by a hand-held motor at 2000 rpm and is advanced within the cutting chamber. The operator shaves the plaque and deposits it in the nose cone of the catheter (Fig. 6-7).

Mechanisms

- The Dotter (pushing) effect created by the bulk of the catheter (5–7F) pushes the plaque aside
- A balloon dilatation effect is caused by inflation of supporting balloons
- Cutting or shaving removes the plaque—this mechanism is usually the dominant one.

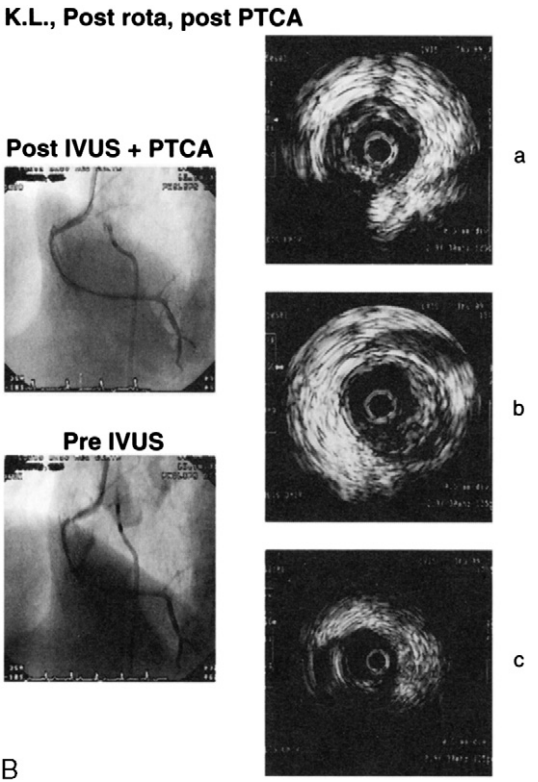

Fig. 6-6, cont'd **B,** IVUS after PTCA shows residual plaque and heavily calcified vessel at the lesion (site b). Site a and site c are proximal and distal to the lesion, respectively.

Indications

- Single or multiple coronary stenoses located in vessels 3.0 mm or more in diameter in proximal segments. Patients with large vessels (>3.0 mm) and ostial or eccentric lesions that decrease the success rate with balloon catheter dilatation are candidates for DCA. Calcified, angulated, long (>20 mm) narrowings, spiral dissections, and friable graft lesions are not suitable for DCA. Bifurcation lesions may be approached using special guidewires, but extra caution must be exercised to avoid perforation.

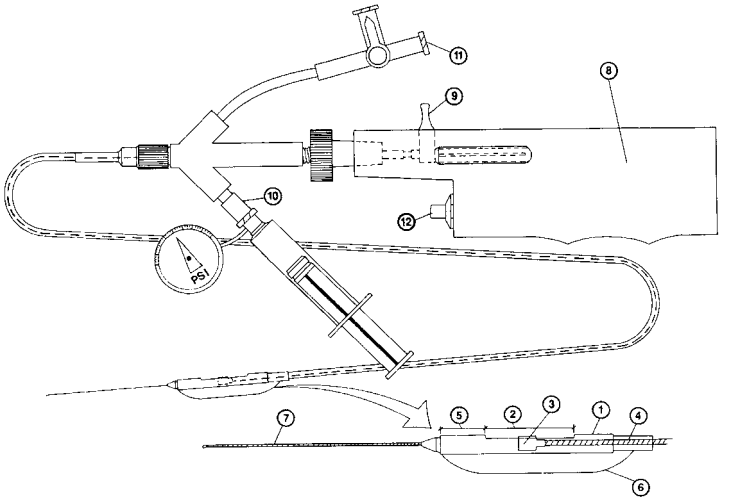

Fig. 6-7 Directional atherectomy catheter system. 1, Cylindrical housing; 2, Opening along housing; 3, Cutter; 4, Cutter drive cable; 5, Specimen collection chamber; 6, Balloon; 7, Guidewire; 8, Drive motor; 9, Cutter advance lever; 10, Balloon inflation port; 11, Flush port; 12, Motor switch. (From Simpson JB, *et al.* Directional coronary atherectomy: a symposium in interventional cardiology. *Am J Cardiol* 1988;61:97G.)

- Saphenous vein graft stenosis with the following characteristics:
  —Discrete
  —Subtotal
  —Accessible.
- Generally, lesions that are most accessible to DCA are those in the proximal or mid portion of coronary vessels. These are the same lesions that are best treated by stenting. The most common use of DCA is debulking prior to stenting or treating ostial lesions with or without stenting. The results of DCA are not superior to percutaneous transluminal coronary angioplasty or stenting.

Because of the need for relatively large sheaths and guide catheters, patients who have severe peripheral vascular disease are considered at high risk for complications with DCA procedures.

Contraindications

Contraindications for DCA are the same as for coronary balloon angioplasty. Relative contraindications include:

- Left main coronary artery disease
- Lesions located in or distal to severely tortuous or densely calcified coronary vessels
- Calcified aorto-ostial lesions
- Significant iliofemoral occlusive disease precludes insertion of large catheters and, therefore, is a relative contraindication for coronary DCA
- Absolute contraindications include:
  - —Patients who are not suitable for coronary artery bypass surgery
  - —Patients with totally obstructed arteries where a guidewire cannot be passed through the lesion.

Equipment

- Atherectomy catheter
- Motor drive unit
- Guide catheter
- Long large sheath (8–11F)
- Hemostatic valve
- Exchange guidewire
- Specimen preparation materials.

Selection of Guide Catheters

Because of the large size of the DCA catheter and rigid cutter housing, large-lumen guide catheters with specially designed curves are needed. The right coronary catheters are 9.5 French and the left coronary catheters are 10 or 11 French. The DCA guiding catheters are designed to provide more torque and more support compared to angioplasty/diagnostic catheters. Each guiding catheter is packaged with a guiding catheter introducer. It is important to use the guiding catheter introducer to ensure that there is minimal guiding catheter trauma during insertion and to maintain hemostasis. Six unique tip shapes are available, designed to accommodate the rigid housing of the DCA catheter (Fig. 6-8).

Guide Catheter Placement

The guide catheter is straightened with the long introducer catheter (7 or 8F) and advanced over a 0.38 inch J guidewire to

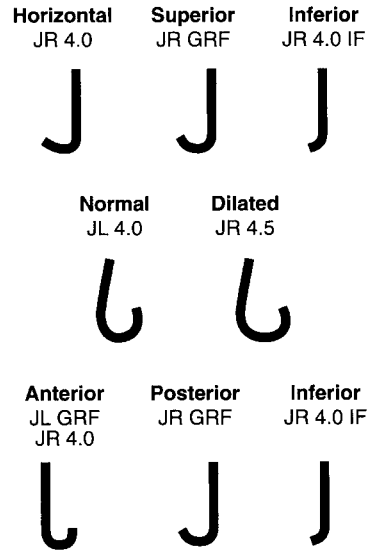

Fig. 6-8 Directional atherectomy guide catheter shapes. JR, Judkins right configuration; GRF, graft; IF, inferior; JL, Judkins left configuration. (From Freed M, Grines C, eds. *Manual of interventional cardiology*. Birmingham, MI: Physicians' Press, 1992: 277.)

avoid trauma to the aorta. This guide catheter and introducer system is advanced into the aortic root with the guidewire several centimeters in front of the introducer catheter tip. The guidewire and inner catheter introducer are then removed. The guide catheter is connected to a manifold and large-diameter Y connector and flushed. The Y connector accommodates larger catheters than those used for balloon angioplasty.

Because of the size and stiffness of the guide catheter, special care should be taken in manipulation, especially avoiding deep engagement of the coronary vessel. Contraindicated techniques for guiding catheter placement include:

- Deep engagement
- Vigorous torquing
- Non-coaxial alignment of the guiding catheter to the ostium.

The Atherectomy Catheter

Catheter Sizing. Selection of the cutting atherectomy catheter size depends on the diameter of the vessel lumen: 5F for vessels

2.5–2.9 mm diameter, 6F for 3.0–3.4 mm, 7F for 3.5–3.9 mm, and 7FG "graft" for vessels 4 mm or larger. In general, most vessels selected should be large enough to be treated with the 7F or 7G catheters (Table 6-2).

Atherectomy Catheter Placement. The target coronary stenosis is crossed in the usual manner with a coronary guidewire (0.014 inch diameter). The DCA catheter is advanced over the guidewire slowly by keeping constant forward tension and rotating the catheter slightly as the catheter is moved forward. Do not "jack-hammer" the catheter. The guide catheter may back up in the aorta at this time. Do not try to bring the guide catheter deep into the vessel. Slight withdrawal of the coronary guidewire while advancing the DCA device is helpful.

It may be necessary to predilate a tight lesion to allow easy passage of the cutting device. While advancing the cutting catheter over the artery curves, keep the cutting chamber opening toward the outer curvature and keep the cutter blade in a forward locked position at all times.

Atherectomy Cutting Sequence

Once the cutting window is in place within the lesion (Fig. 6-9):

1. The cutter is retracted using the thumb lever on the motor drive
2. The balloon pressure is increased to 20–30 psi (**Caution:** Balloon rupture may occur at more than 60 psi or 4 atm)
3. The motor is turned on
4. The cutter is advanced in a slow and steady manner until it is at the end of the cutting chamber (failure to advance and lock the cutter fully in place risks tissue embolization and/or produces an incomplete cut, leaving an intimal flap with the potential for abrupt reclosure)
5. The motor is turned off
6. The balloon is deflated
7. The cutting catheter is rotated about a quarter turn and the sequence is repeated
8. During cutting, it is important to keep the wire a few centimeters distal to the catheter tip to avoid entrapment in a small vessel.

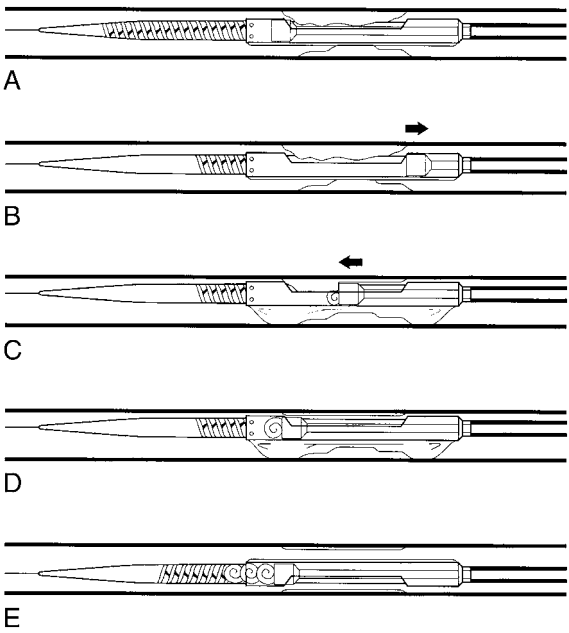

Fig. 6-9 Directional atherectomy catheter cutting sequence**. A**, Catheter housing is positioned in the coronary lesion. Cutter is forward. **B,** Cutter is retracted. **C,** Balloon is inflated and cutter motor turned on; cutter is slowly advanced, shaving tissue. **D,** Cutter is advanced to deposit the specimen into the nose cone chamber. **E,** Balloon is deflated. Keep the cutter forward until after the catheter is rotated to the next position. Repeat steps **A–E.** (From Simpson JB, Selmon MR, Robertson GC, *et al.* Transluminal atherectomy for occlusive peripheral vascular disease. *Am J Cardiol* 1988;61:96G–101G.)

Removal of the Catheter

There are two ways to remove the DCA catheter. Both ways require the cutter be located in the distal section of the housing.

- Use a 300 cm exchange guidewire
- Remove the guidewire and DCA together. A guidewire can be left across the lesion if it is not fixed in the distal nose cone. Experience has shown that it is best to use an exchange technique:
  —If wire negotiation was difficult at the initial pass
  —When working in vein grafts
  —When working in dissections
  —Following predilatation.

When removing the DCA catheter, put tension on the guiding catheter to prevent deep seating. If the guidewire becomes fixed, remove the guidewire with the DCA and empty the distal collection chamber. Guidewire recrossing is generally easy, because of the smooth surfaces at the atherectomy site.

Atherectomy Tissue Removal. Removal of tissue from the DCA catheter is best accomplished by pulling the cutter blade proximal, inserting the nose cone into a saline-filled syringe, and flushing specimens into a collection cup. Once they are dislodged from the collection chamber, specimens in the chamber may be retrieved with tweezers. Check for tissue specimens in the cutter cup. After removing tissue, the DCA catheter may be reinserted for further atherectomy. Prior to reinsertion, the guidewire lumen should be flushed with saline.

Criteria for Successful Atherectomy

Three criteria are used in combination to evaluate atherectomy results:

- An artery with angiographically smooth borders
- Residual stenoses in the range of 5–20%
- An average number of tissue samples removed—five to 13 samples weighing more than 5 mg.

Anticoagulation and Sheath Removal

Administration of heparin follows standard balloon angioplasty technique (40–70 u/kg bolus, consider glycoprotein blocking drugs). During the procedure, the patient should be well anticoagulated, with an activated clotting time (ACT) of 200–300 seconds. Early sheath removal (≥4 h after the procedure) is advocated and may require a vascular closure device. Because of the large size of the sheaths, distal leg pulses should be checked frequently. In case of limb ischemia, the atherectomy sheath should be removed promptly.

Complications

Arterial complications are the most significant source of morbidity and mortality, especially in elderly patients. Other complications of DCA include proximal vessel dissection, thrombotic or atherosclerotic emboli to uninvolved adjacent

branches, vasospasm, coronary ostial injury, myocardial infarction, and death.

Several helpful points to remember to minimize complications with DCA are:

- Cut only where disease is present
- Avoid large devices in small vessels
- Avoid extensive angioplasty dissections
- Use low inflation pressures (≤30 psi)
- Avoid bifurcation and ostial lesions where DCA techniques are prone to perforation (Fig. 6-10).

Post-Atherectomy Follow-up and Restenosis

Routine follow-up is similar to that for balloon angioplasty. Restenosis after atherectomy occurs at a rate and time similar to that for balloon angioplasty. A recent randomized study suggested a small restenosis benefit with DCA as compared to balloon angioplasty in patients with proximal left anterior descending artery lesions. Restenosis after DCA is less in patients with less than 20% residual narrowing and in those with a reference-vessel lumen diameter of 3 mm or more. Since some studies have indicated increased complications with DCA, the risk:benefit ratio should be carefully evaluated in each patient.

Figure 6-11 illustrates an angioplasty with adjunctive DCA guided by IVUS imaging.

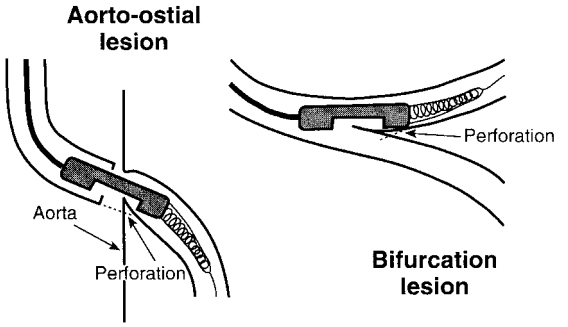

Fig. 6-10 Diagram of directional atherectomy catheter positioning in the ostial lesion (left panel) and at bifurcation points (right panel). Extra caution is needed to avoid perforating the vessel at these high-risk locations. (From Topol EJ. *Textbook of interventional cardiology*, 2nd ed. Philadelphia: WB Saunders, 1994:594.)

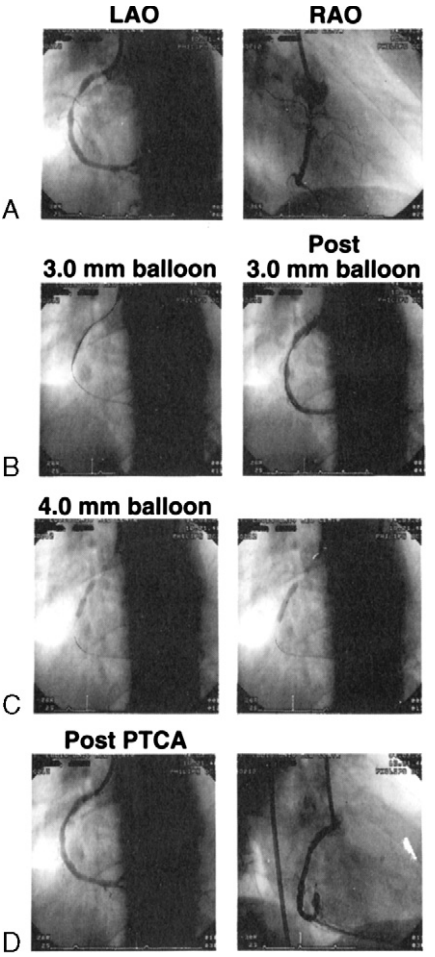

Fig. 6-11 Case example of sequential percutaneous transluminal coronary angioplasty (PTCA) and directional atherectomy catheter guided by intravascular ultrasound imaging (IVUS). **(A)** Angiograms demonstrating severe proximal right coronary stenosis in a 47-year-old man with rest angina. Calcifications are not visible angiographically. **(B)** A 3.0 mm balloon is inadequate at 12 atm and **(C)** a 4.0 mm balloon is used at high pressure with **(D)** a marginal result.

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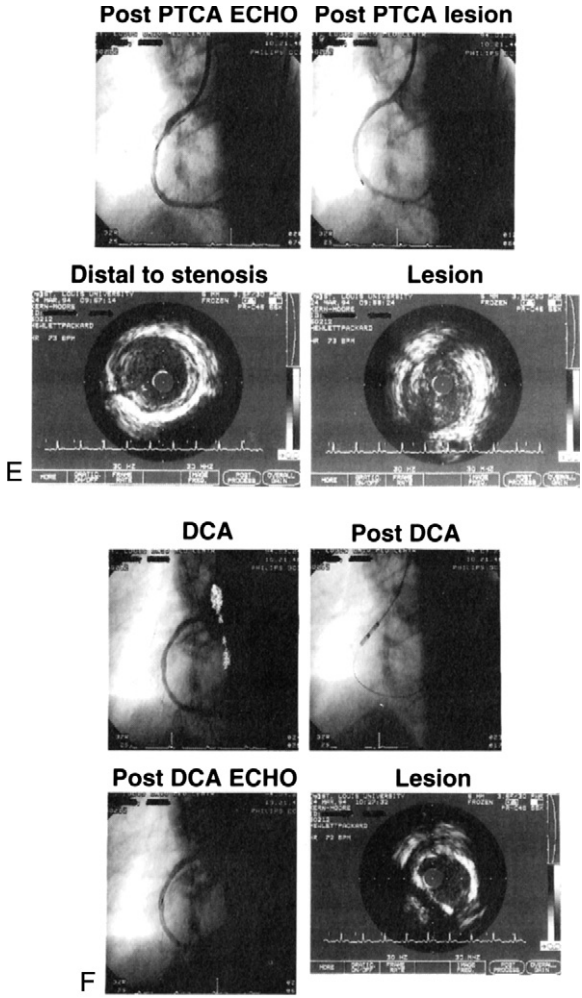

Fig. 6-11, cont'd **(E)** IVUS images with corresponding angiography. IVUS in lesion shows considerable residual plaque. **(F)** The DCA catheter in place with angiography after DCA cutting and IVUS. The IVUS image is considerably improved and now shows deep calcifications not previously identified.

THROMBUS ASPIRATION SYSTEM

Percutaneous thrombectomy devices are now available to promptly re-establish flow. These devices either pulverize and/or aspirate the thrombus. Aspiration devices (e.g., Possis AngioJet®) are preferred for arterial thromboses but are limited by the small size of the catheter compared to the reference vessel. Also, adherent thrombus (chronic) may not aspirate easily. Extracting thrombus from the distal occlusion initially before flow is re-established can minimize distal embolism and hemolytic complications.

The AngioJet® Rheolytic™ Thrombectomy System opens an artery by aspirating clot. High-pressure water jets directed backward into the catheter create strong suction at the space near the tip. Effective thrombus evacuation occurs (Fig. 6-12). Conventional treatment for thrombus in the past involved mostly a "lyse and wait" procedure using thrombolytic therapy to dissolve thrombus. The AngioJet system removes thrombus from the artery as safely as thrombolytic therapy and, in some lesions, with less risk of breaking up thrombus with the distal thromboembolism.

In the VeGAS 2 trial of AngioJet thrombectomy in saphenous vein graft thrombosis, procedural success—achievement of a

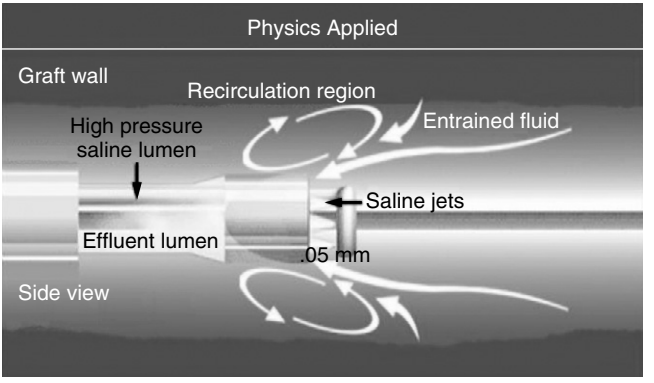

Fig. 6-12 AngioJet thrombectomy catheter mechanism. High-speed jets create local vacuum with balanced flow. Within the graft wall, the saline jets shooting into the catheter lumen create a strong Venturi effect with recirculation and entraining fluid. The high-pressure saline lumen aspirates the thrombus for expulsion into the collection unit.

final residual diameter stenosis of less than 50% and TIMI 3 flow post-procedure, in the absence of death, emergent bypass surgery, or Q-wave myocardial infarction—was high (>90%). Regardless of the age of the thrombus, success was significantly higher with the AngioJet. Procedural success was also achieved in 81.3% of interventions with chronic (>2 weeks old) thrombus. The incidence of major adverse events was 52% less with the AngioJet system. Myocardial infarction (Q or large non-Q) was reduced with the AngioJet system, an effect that was sustained for 1 year after the procedure. The AngioJet often requires temporary pacing and may be less effective in very-large-diameter conduits.

Components

The AngioJet System is made up of three components; a driver, a pump set, and replaceable catheters.

Driver. The driver is a pump system that monitors the aspiration and system flow during the procedure for patient safety. It generates 10,000 psi of water pressure to the pump set and catheter tip.

Pump Set. The pump set drives saline into the catheters as well as maintaining the balance between liquid leaving and entering the catheter so as to maintain a constant pressure within the artery. It also serves as the bridge between the sterile and non-sterile components of the system. It is color-coded for easy setup.

AngioJet Catheters. The Possis AngioJet catheter is a 135–140 cm long, 4–6 French catheter that tapers at the distal 5 cm. The catheter is attached to a driving unit and roller pump, which generates high-speed pulsed flow (50 ml/min) into a high-pressure tube. This tube forms a 180° loop at the catheter tip. From this tip, six high-pressure jets are directed retrogradely into the collecting lumen of the catheter. The saline, which exits the loop at a speed of about 450 km/h, creates a vortex that fragments the thrombus and the Venturi effect creates a vacuum that aspirates the thrombus material into the catheter.

Technique

The AngioJet catheter is advanced through the suspected thrombus and positioned distally. The unit is then activated and the catheter is pulled back slowly at the rate of 0.5 mm/sec. Multiple passes are performed until no further improvement is noted. AngioJet thrombectomy is used for coronary thrombosis in acute myocardial infarction, stent thrombosis, saphenous vein graft thrombus, thrombosed dialysis fistula, thrombosed peripheral vessels, and occasionally in acute pulmonary embolism for pulmonary artery thromboembolism. Thrombectomy may be combined with distal protection devices to limit the effects of embolization. Figure 6-13 shows a case of acute inferior myocardial infarction with a large thrombus burden treated with AngioJet and PCI.

EMBOLIC PROTECTION DEVICES

Distal embolization of particulate matter commonly complicates percutaneous coronary interventions, especially for acute coronary syndromes. A number of distal protection devices are under development. These use either an occlusion balloon to trap particles that are then aspirated or a filter that traps the particles, which are removed with the filter. The PercuSurge GuardWire™ is a protection balloon occlusion with a thrombectomy catheter device approved for saphenous vein graft interventions. The AngioGuard expandable filter is one of several filter devices mounted on an angioplasty guidewire that are being tested for the entrapment of particles and safe removal. Table 6-3 compares the advantages and disadvantages of embolic protection devices.

Distal Occlusion Balloon

The PercuSurge GuardWire (Medtronic) consists of a wire containing a central lumen that communicates with a low-pressure distal occlusion balloon incorporated into the tip (Fig. 6-14). The wire both serves as the angioplasty guidewire and provides protection from distal embolization. An inflation device allows controlled expansion and sizing of the occlusion balloon in the treated vessel. An aspiration catheter is used to remove the debris from the treated vessel before the balloon is deflated and antegrade flow in the treated vessel is restored.

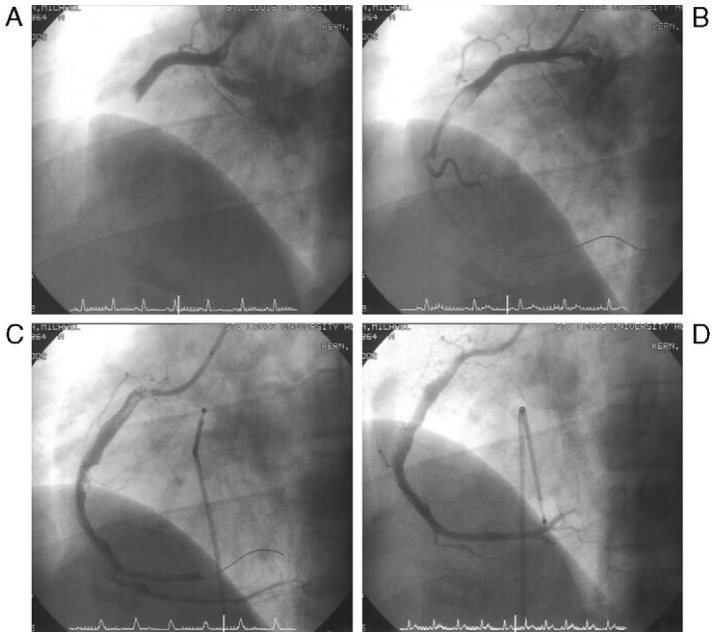

Fig. 6-13 Example of AngioJet thrombectomy in a 37-year-old man 8 hours after the onset of chest pain for acute inferior wall myocardial infarction. **A,** Right coronary artery with thrombus in proximal portion. **B,** Angioplasty guidewire traversing lesion with large amount of clot in the proximal portion of the artery. **C,** Right coronary artery after 4 French AngioJet. A temporary pacemaker was also inserted. **D,** The thrombus was almost completely extracted from the vessel. The final angiogram demonstrates residual distal embolic occlusions but good patency, with TIMI grade 3 flow.

The Saphenous Vein Graft Angioplasty Free of Emboli (SAFE) study evaluated the safety and feasibility of this device. Initial encouraging results in the European and Canadian registries were confirmed in the Saphenous Vein Graft Angioplasty Free of Emboli, Randomized (SAFER) trial conducted in the USA. The SAFER trial was conducted at 47 sites and enrolled 659 patients undergoing saphenous vein graft percutaneous coronary intervention. Inclusion criteria were stenoses of 50–99% in saphenous vein grafts 3–6 mm in diameter, more than 5mm from the ostium and 20 mm from the distal

Table 6-3

Advantages and Disadvantages of Embolus Protection Devices

	Filter	Balloon Occlusion
Perfusion	Permits blood flow; depending on pore size	Prevents perfusion during manipulation causes ischemia if no collateral flow
Emboli	Prevents emboli greater than pore size (e.g., 100 μm)	Inflated balloon, traps all emboli and debris
Vasoactive substances and cytokines	Flow through filter	Potentially prevents active substances from reaching the distal bed
Crossing considerations	Bulky	Lower profile than filter wires
Retrieval function	Full of debris, occasionally difficult to collapse and retrieve filter	Slow balloon deflation may prolong ischemic time
Embolization during device placement	Possible	Possible
Visualization of vessel	Satisfactory	Once occluded, no flow

anastomosis, and at least TIMI 1 flow at baseline. Exclusion criteria included acute myocardial infarction, ejection fraction less than 25%, creatinine above 2.5 mg/dl (unless on hemodialysis), and planned use of an atherectomy device. The primary endpoint was the occurrence of major adverse clinical events at 30 days, including death, myocardial infarction, emergency bypass surgery, and repeat target vessel revascularization.

Filter Wire

AngioGuard (Cordis Corporation, Minneapolis, MN) is an example of a filter device approved for marketing in Europe. AngioGuard is a filter on a guidewire that expands to 6 mm and is placed distal to the target lesion to capture and retrieve embolic debris. At the end of the procedure, the filter is collapsed, trapping the particulate matter and facilitating removal from the artery. The AngioGuard filter has multiple 100 μm laser-drilled holes that allow perfusion during device deployment, a major advantage over occlusive protection devices. Maintained perfusion is a critical clinical consideration

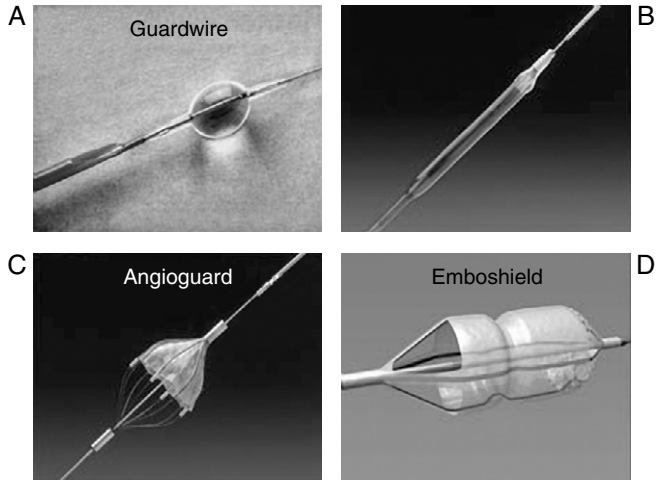

Fig. 6-14 Distal protection devices. **A,** The guard wire, PercuSurge balloon occlusion, and export catheter. **B,** AngioGuard in the undeployed state. **C,** AngioGuard in the deployed state acting as an umbrella-type filter. **D,** An Emboshield filter device.

in patients with reduced left ventricular function or in patients in whom the treated artery supplies a large amount of myocardium. Conversely, incomplete vessel occlusion with the filter devices may allow passage of debris through the holes of or around the filter devices. Indeed, analysis of debris retrieved by the balloon occlusion thrombectomy device in the SAFE trial device found that 80% of the particulate matter was less than 100 μm in diameter. The clinical significance of such small embolic particles is unclear. Figure 6-14 shows different distal protection devices and filter designs.

THE CUTTING BALLOON

The Cutting Balloon was designed specifically to reduce trauma on the vessel wall and on the plaque, by making small incisions into the plaque rather than splitting and tearing it, as occurs with standard balloon trauma. The Cutting Balloon has three or four tiny stainless steel blades called atherotomes, 0.1–0.4 mm thick, fixed to the surface of the balloon. These atherotomes are tucked within the folds of the balloon (Fig. 6-15). The folds of the balloon safely cover the blades

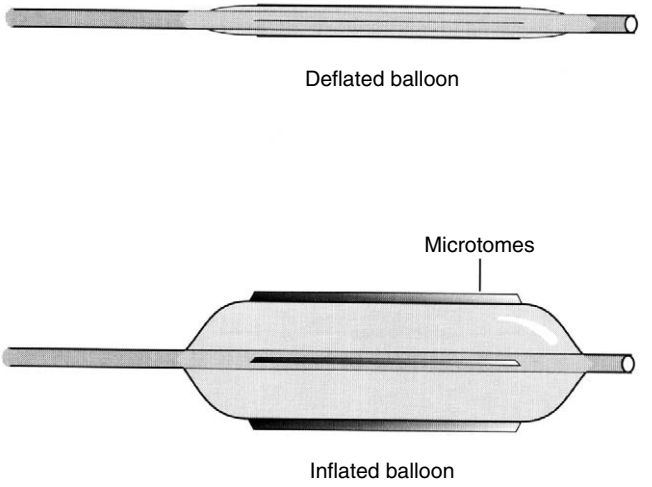

Fig. 6-15 The Cutting Balloon (above) in the deflated state and (below) in the inflated state. The microtomes can be seen extending outward from the balloon.

until the balloon is inserted in the artery at the lesion site. After it is inserted and inflated, the blades are exposed. These blades are purported to make microscopic surgical incisions in the plaque rather than cutting through the plaque completely. The pressure of the balloon on the precut tissue appears to be associated with less trauma than balloon angioplasty.

Indication

The Cutting Balloon is indicated primarily in three clinical situations (Box 6-1):

- Bifurcation lesions
- In-stent restenosis
- Ostial lesions.

Box 6-1

Uses of the Cutting Balloon

- In-stent restenosis
- Small vessels
- Bifurcations
- Aorta-ostial lesions
- Saphenous vein graft lesions
- Site preparation for stenting

Although heavily calcified lesions have been treated with Cutting Balloon, there is controversy about this application. Lesions best suited for Cutting Balloon angioplasty are those which are relatively short (<20 mm), concentric lesions in vessels with moderate or less tortuosity, and those without significant thrombus. Lesions in small vessels less than 2 mm diameter, total occlusions, heavily calcified lesions, or lesions more than 20 mm long are not considered appropriate for Cutting Balloon angioplasty.

Equipment

Guide Catheter. With appropriate wire support the Cutting Balloon can be used with 6 and 7 French systems. A 6 French guiding catheter can be used for Cutting Balloon sizes 3.25 mm or less. A 3.5 mm balloon with four atherotomes is best used with a 7 French system for adequate contrast visualization of the lesion. In addition, a small guiding catheter may not allow the Cutting Balloon to be retrieved without pushing the atherotome against the balloon material and possibly causing micropunctures. Standard guide catheter shapes are acceptable, except for the support needed for tortuous arteries. In an acute circumflex takeoff, Amplatz, Voda, or Q-curve shapes, or extra support guiding catheters will facilitate device delivery. When access to the lesion is difficult because of vessel tortuosity, a floppy guidewire may help by limiting wire bias. Some physicians have used the Wiggle Wire (Guidant Corporation, Santa Clara, CA; the Wiggle Wire has a series of pre-bent areas) in situations where the atherotome may be caught on calcium or a stent strut. Forming an artificial guidewire bias away from any obstruction or using a buddy wire technique can also be helpful for difficult anatomy.

Cutting Balloons. Cutting Balloons range from 2.0 to 4 mm in diameter from 10 mm to 15 mm in length. Sizing the Cutting Balloon is a critical step. The balloon:artery ratio, using IVUS, should be 1:1, measuring from media to media. In in-stent restenosis when the stent size is known, the Cutting Balloon can also be sized 1:1, or it can be 0.25–0.50 mm larger if the device is kept within the stent. When sizing by angiography, upsize the Cutting Balloon by a quarter size, except when treating ostial lesions, lesions on an extreme bend, or small

vessels, because of the risk of perforation. If the initial device is undersized, a hazy angiographic appearance may be present. Although a conventional balloon may be used following dilatation, the lack of atherotomes may cause deep vessel wall trauma.

Technique

Delivering the Cutting Balloon may be more difficult than conventional balloons. As a result of the atherotomes, the Cutting Balloon becomes a stiffer device. Some maneuvers that may be helpful are listed below.

- If the device fails to traverse a *de novo* lesion, use a 1.5–2.0 mm balloon to predilate the lesion. This will not compromise the end result
- Use the buddy wire technique in tortuous segments or into a stent
- Inflating the device when it is partially in the lesion and then advancing as it deflates may be helpful.

Inflation/deflation techniques help deploy and retrieve the balloon and atherotomes. Inflating or deflating the Cutting Balloon incorrectly can cause damage to it. When inflating the Cutting Balloon, do it slowly and gradually, at 1 atm/5 sec, to allow the atherotomes to unfold. When the Cutting Balloon is fully inflated to nominal pressure (6 atm), maintain inflation for 60–90 seconds to allow it to not only score the lesion but also flatten the incisions. Performing multiple slow inflations may improve angiographic results. The Cutting Balloon does not need to be rotated into a different position to perform multiple inflations.

Deflate the Cutting Balloon very slowly. Without slow deflation, it may wing and resistance to retrieval occurs on re-entry into the guide catheter. If the balloon remains inflated as a result of lost pressure, use a 60 ml syringe to deflate it, making sure there are no kinks in the catheter.

Special Lesion Techniques

Eccentric Lesions. Try at least three inflations at nominal pressures after repositioning the balloon to allow the atherotomes to find an elastic site. If the balloon continues to be underexpanded, use higher pressures, up to 10–12 atm.

Multiple Lesions. Dilate the most distal lesion first unless the proximal lesion is flow-limiting or prevents the passage of the Cutting Balloon.

Tortuous Anatomy. Straightening the vessel with an extra-support guidewire may help. If the treated vessel segment is tapered, size the Cutting Balloon to the distal portion of the vessel first. If the proximal portion is larger than the distal portion, two different sizes of Cutting Balloon may be needed.

In-stent Restenosis (ISR). Minimize multiple inflations. In aorto-ostial lesions, let the Cutting Balloon protrude 15 mm into the aorta. Consider IVUS before Cutting Ballon to select correct Balloon size.

Bifurcation Lesions

Cutting Balloon angioplasty may minimize branch dissection, because the plaque is incised at the ostial and bifurcation point, minimizing plaque shift compared to balloon angioplasty. The Cutting Balloon may also minimize the effects of elastic recoil at the bifurcation site. It does, however, have a large profile and less flexibility because of the blades, limiting its usage in sharply bifurcated lesions. The kissing balloon technique employed for bifurcations is problematic when one balloon is the Cutting Balloon. The Cutting Balloon has been recommended for bifurcation lesions under the following circumstances.

- When the stenosis at the bifurcation site is in the main branch, with no significant stenosis in the side branch
- When the difference between the proximal and distal reference diameters of either side branch bifurcation site is small
- When the side branch is small and occlusion by the balloon carries little or no risk.

Concerns about the use of the Cutting Balloon include the risk of restenosis, especially in small vessels, and the risk of perforation. The Cutting Balloon can cause deep cuts, with perivascular staining. In these situations, stenting is always used to decrease subsequent dissection and occlusion. Additionally, there are many small studies suggesting that using the Cutting Balloon before stenting has a lower restenosis rate.

Cutting Balloon Procedure—Key Points

- A balloon:artery ratio of 1.1 should be achieved with low inflation pressures of 4–6 atm over 1–2 minutes to permit gradual

expansion and exposure of the blades to firmly incise the plaque. Two to three low pressure balloon inflations may be needed

- Sudden inflating of the balloon should be avoided so that high pressure will not foul the opening of the blades and result in insufficient plaque incision.

Complications

In the REDUCE I trial, the angiographic complications of coronary perforation had an incidence of 0%, coronary dissection 26%, and clinical complications of death, Q-wave MI, emergency coronary artery bypass grafting, and non Q-wave myocardial infarction 0%, 0%, 0%, and 1%.

A potential disadvantage of the Cutting Balloon is that it does not easily cross standard lesions, and predilatation may be required in some circumstances.

Clinical Outcome

To determine the immediate and chronic results of Cutting Balloon angioplasty, Kondo *et al.* performed it on 127 lesions in 110 patients. The overall procedural success rates were 93.7% (119 lesions) and 92.7% (102 patients) with few complications. The successfully treated Cutting Balloon angioplasty group (95 lesions) was matched with the successful conventional angioplasty group for chronic result assessment. The Cutting Balloon angioplasty group showed a significantly lower restenosis rate (23.1%) than the conventional angioplasty group (42.1%).

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7

RESTENOSIS, BRACHYTHERAPY, AND DRUG-ELUTING STENTS

Souheil Khoukaz, Michael J. Lim, and Morton J. Kern

RESTENOSIS

Restenosis is defined as the reaccumulation of material within a vessel at the site of previous coronary angioplasty. The process of restenosis appears to be initiated by injury of the vessel, with a release of thrombogenic, vasoactive, and mitogenic factors. Endothelial and deep vessel injury leads to platelet aggregation, thrombus formation, inflammation, and activation of smooth muscle cells and macrophages. The production and release of growth factors and cytokines promotes further synthesis of such factors and release from the cells involved. The migration of smooth muscle cells is initiated from their location within the arterial media to the endovascular lumen. These cells become a synthetic type of cell that produces extracellular matrix, leading to cellular proliferation and mechanical obstruction of the vessel lumen. Recoil and remodeling of the arterial wall are also important components of the restenosis process. The vessel is further affected by scar contraction, which may reduce the appearance of the lumen.

This process occurs to a greater or lesser degree in all patients who undergo coronary angioplasty. Restenosis is not device-specific but rather a function of the anatomic substrate and the type of injury produced (Fig. 7-1).

Definitions of Restenosis

There are two types of restenosis recognized in patients, angiographic and clinical, which are not mutually exclusive.

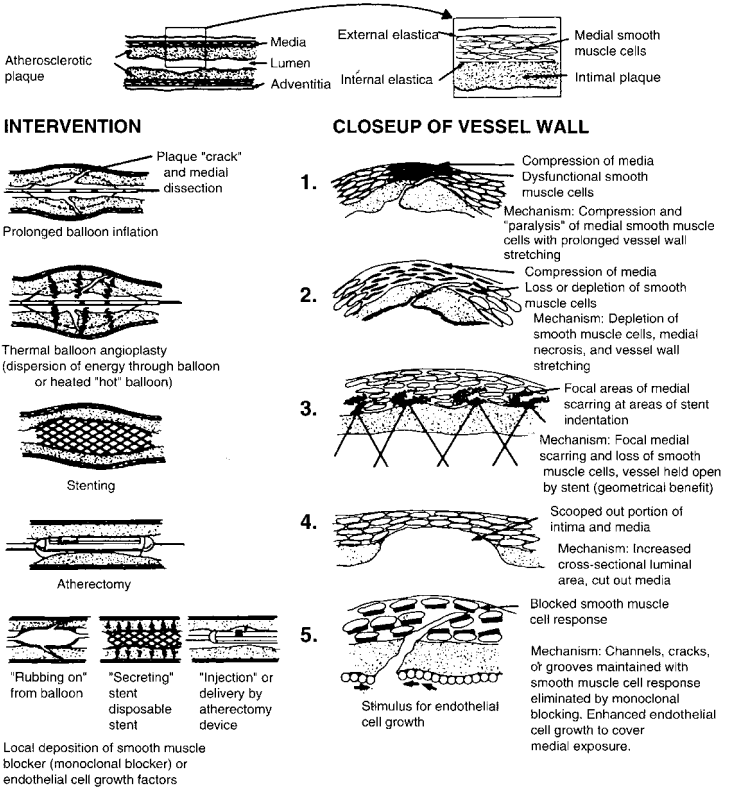

Fig. 7-1 Interventional devices and presumed mechanisms of action of arterial plaque in vessel wall lead to restenosis. The indication of immediate outcome and restenosis rates depend on both the device and the arterial substrate encountered. (From Waller *et al.* Mechanisms of restenosis after successful balloon angioplasty. *J Am Coll Cardiol* 1991;17:58B–70B.)

Angiographic Restenosis. Angiographically measured luminal renarrowing after coronary angioplasty has long been the "gold standard" for restenosis. Angiographic restenosis is a continuous phenomenon, with no obvious threshold separating "restenosers" from "nonrestenosers." Studies have shown that percentage stenosis or minimal lumen diameter has a near Gaussian (normal) distribution on followup angiograms after balloon angioplasty. Thus, restenosis is best measured as a continuous variable. Nevertheless, because of practicality, the

most commonly used definition of restenosis employs a dichotomous value (e.g., 50% diameter narrowing).

Several different angiographic definitions of restenosis have been published with different definitions overlapping in some patients. Binary angiographic restenosis rates are determined by followup lesion diameter of less than 50% of the reference vessel diameter. For coronary stenting, this is generally less than 20% of the diameter, depending on the type of lesion treated. The late loss of the acute luminal enlargement, or net gain in millimeters at the lesion site 6 months after treatment by quantitative angiography, should be around 0.7 mm for balloon angioplasty. The late loss index is the loss at the lesion site divided by the amount of acute gain (Fig. 7-2). The loss index is accepted as the most sensitive measure of the

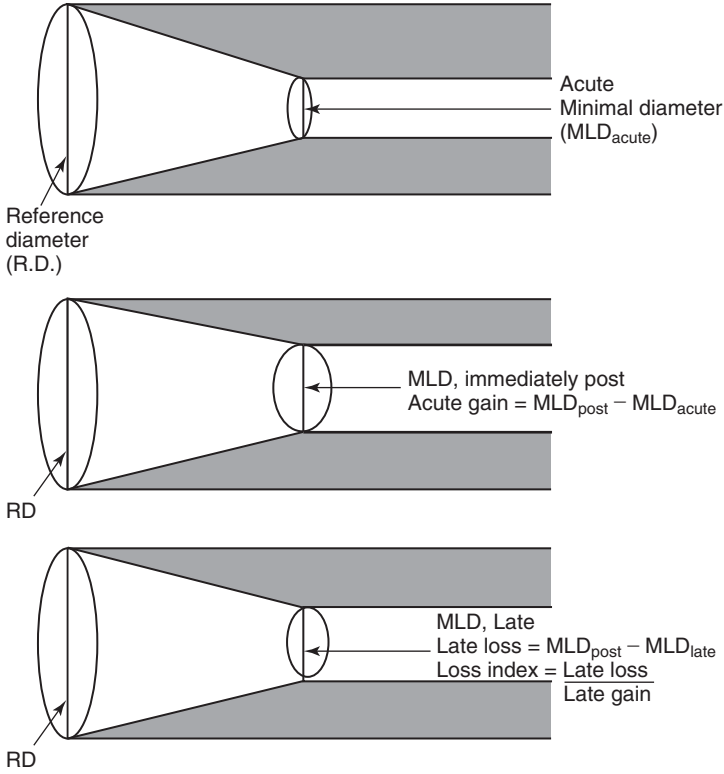

Fig. 7-2 Calculations of acute gain, late loss, and loss index.

effectiveness of the technique and should range from 0.4 to 0.6 mm for balloon angioplasty. The lower the loss index, the more effective the antirestenosis treatment.

Restenosis is both a lumen- and a wall-related phenomenon. It appears that 40–60% of the acute luminal gain is lost during followup in all patients treated, independent of the devices. A similar degree of intimal thickening (restenosis by wall measurements) may or may not cause a significant luminal narrowing (restenosis by lumen measurement). As expected, the vessel size itself exerts a significant positive influence on minimal lumen diameter at followup and an equally negative effect on late loss. A larger artery will have a larger lumen at followup and vice versa for a smaller artery. Using percentage stenosis rather than absolute lumen diameter will neutralize this effect by correcting automatically for artery site.

Intravascular ultrasound imaging is superior to angiography for anatomic and morphologic restenosis definitions. Recent intravascular ultrasound studies have shown that an important component of restenosis is vessel recoil, a feature prevented by stenting. Normal vessel modeling maintains the coronary lumen. Late negative remodeling of the injured vessel is also prevented by stenting (Figs 7-3, 7-4).

Clinical Restenosis. Clinical restenosis is defined as recurrent angina or anginal-equivalent symptoms after coronary angioplasty. Other causes of symptoms might be mistaken for clinical restenosis, such as disease progression in the nondilated

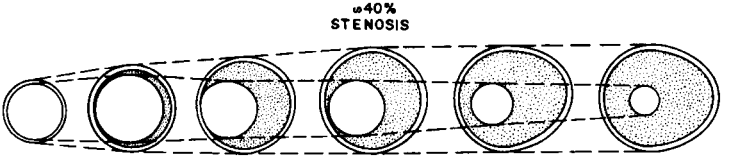

Fig. 7-3 The Glagov phenomenon. According to serial intravascular ultrasound, normal segments showed proximal enlargement of the vessel as plaque volume increases. Vascular dilatation is compensated for by substantial plaque formation inside the vessel wall leaving the vessel's angiographic appearance unchanged and normal looking. (From Glagov S, Wisenberg E, Zarins CK *et al.* Compensatory enlargement of human atherosclerotic coronary arteries. *N Engl J Med* 1987;316:1371–1375. © Massachusetts Medical Society.)

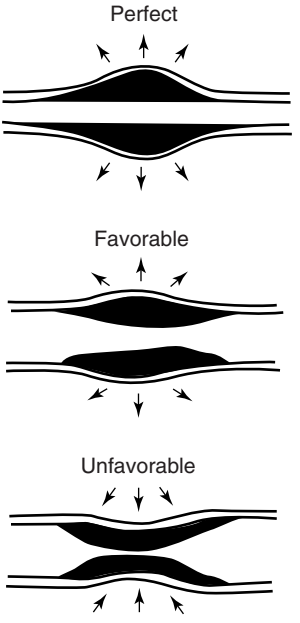

Fig. 7-4 Adequacy of arterial remodeling with respect to changes in vessel size. (From Schwartz RS. Pathophysiology of restenosis: interaction of thrombosis, hyperplasia, and/or remodeling. *Am J Cardiol* 1998;81:16E.)

arterial segment, which occurs in 10–15% of cases late after angioplasty. Incomplete revascularization also causes symptoms in about 10% of patients. Recurrence of typical angina after an asymptomatic period following angioplasty is a very specific clinical indicator for restenosis. On the other hand, atypical chest pain is a poor predictor. Restenosis maybe documented in 15% of asymptomatic cases.

Early (<1 month) exercise tests after percutaneous transluminal coronary angioplasty (PTCA) are often persistently positive and fail to predict future restenosis and recurrent events. An exercise test at 6 months in patients who have not yet presented with clinical recurrence shows a modest positive predictive value. Bengston and colleagues showed that exercise-tests were independent predictors of restenosis. Despite a multivariate approach, 20% of restenosis cases were not recognized. Compared to standard exercise testing, myocardial perfusion imaging stress studies may improve diagnostic accuracy.

Risk Factors for Restenosis

It is not possible to predict reliably whether restenosis will occur in a given patient. By a multivariate analysis, severe angina, left anterior descending artery lesions, diabetes, a higher degree of residual restenosis, hypertension, absence of an intimal tear, eccentric lesion morphology, and older age are predictors for restenosis.

It is difficult to obtain followup angiograms in all patients undergoing coronary angioplasty. Less than complete angiographic followup with preferential recatheterization of symptomatic patients creates bias, in that symptomatic patients artificially increase the restenosis rate in that population. At the same time, asymptomatic restenosis cases will go unrecognized clinically. Recurrence of symptoms, or other events such as fatal or nonfatal myocardial infarction, is a useful indicator of the efficacy of coronary angioplasty as a therapeutic procedure. This approach may underestimate the actual angiographic restenosis rate. Target vessel revascularization rate is another surrogate for angiographic restenosis rates.

Time Course

In a small proportion of patients, restenosis occurs very early (<24 h), due to acute elastic recoil. After this point, the incidence of restenosis increases rapidly up to the third month. New restenosis occurs uncommonly after 12 months. Restenosis after angioplasty using new devices follows a similar time course. The reported incidence of restenosis varies between 15% and 55%, with most studies averaging around 30%. Different mechanisms produce restenosis in a time-dependent manner. Early restenosis is due to thrombus, whereas late restenosis is related more to remodeling (Fig. 7-5).

Patient Subsets at Higher Risk of Restenosis

Diabetes Mellitus. Restenosis is more prominent in insulin-dependent diabetics. In a recent study comparing acute and 5-year outcomes in 1133 diabetic and 9300 nondiabetic patients, acute success and complication rates were only slightly worse than for nondiabetic patients but 5-year survival was significantly shorter in diabetic patients, with only 36% surviving 5 years without reinfarction or repeat target lesion

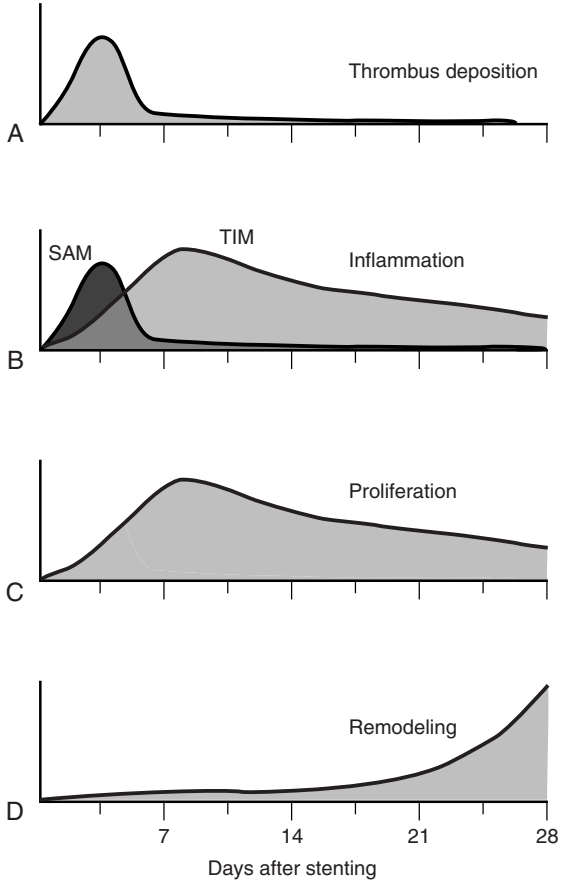

Fig. 7-5 The four phases of vascular repair after stent-induced arterial injury in terms of time after stenting. **A,** Platelet-rich thrombus accumulates at areas of deep strut injury and peaks at 3–4 days after stent deployment, accounting for most early lumen loss. **B,** Coincident with thrombus deposition, inflammatory cells are recruited to the injury site, both at and between stent struts. At 3–7 days after stenting, the surface-adherent monocytes (SAM) migrate into the neointima as tissue-infiltrating monocytes (TIM) and remain in place. **C,** Proliferation of smooth muscle cells and monocyte macrophages within the neointima peaks at 7 days after implantation and continues above baseline levels for weeks thereafter. **D,** Collagen deposition in the adventitia and throughout the tunica media and neointima leads to arterial shrinkage or remodeling, causing compression of the artery on stent struts from without. (From Garasic J, Edelman E, Rogers C. Stent design and the biologic response. In: Beyar R, Keren G, Leon M, Serruys PW, eds. *Frontiers in interventional cardiology*. London: Martin Dunitz, 1997: 95–100.)

revascularization (TVR). There was significant repeat TVR within the first year. The event rate was worse in insulin-dependent diabetics for each end-point as compared to non-insulin-dependent diabetics.

Chronic Renal Failure. Patients with chronic renal failure who undergo PTCA have a high restenosis rate (60–80%).

Transplantation. Restenosis is frequent after balloon angioplasty in heart transplant recipients. In a multicenter study, the restenosis rate 8 ± 5 months after balloon angioplasty of 76 lesions was 55%. Importantly, early or late failure of angioplasty had serious consequences. In a series of 162 transplant patients, 3-year survival was less than 50%. The results with new devices in this population are under study.

Acute Myocardial Infarction. Although the immediate and medium-term clinical outcome is very favorable, the angiographic restenosis rate after angioplasty for acute myocardial infarction is not well known. Compared to elective procedures, late restenosis after urgent angioplasty was found to be lower (35% versus 19%). Since the rate of in-hospital reocclusion was higher in the myocardial infarction group (13% versus 2%), the lower late restenosis rate likely reflects a difference in the time course of restenosis. Other studies report the 6-month restenosis rate after angioplasty for acute myocardial infarction as 52%, similar to the results of elective PTCA.

Total Occlusion. Total occlusion has a higher restenosis rate than subtotal stenosis. Total occlusion angioplasty restenosis rate may not plateau at 6 months. In an angiographic followup study after total occlusion angioplasty, 41% of patients had restenosis within 6 months and 66% had restenosis within 12 months. In other studies, the angiographic restenosis rate ranged from 45% to 60%. The recurrence of total occlusion lesions seldom results in myocardial infarction because of reformation of prior collateral protection.

Saphenous Vein Graft Lesions

Saphenous vein graft lesions are associated with a higher restenosis rate, particularly in the proximal anastomotic (58%)

and body (52%) portions of the graft. Distal anastomotic narrowing responds to angioplasty well, especially in patients with recurrent coronary artery bypass graft surgery. The time course of restenosis in vein grafts is different than in native coronary vessel, with continued significant attrition beyond 6 months.

Internal Mammary Artery Graft Lesions

The internal mammary graft anastomotic site responds very favorably to angioplasty, with 15% or less restenosis rate.

Nonballoon Device Restenosis

Percutaneous transluminal coronary rotation atherectomy (PTCRA) pulverizes the atheroma to create a satisfactory coronary lumen. The ablating mechanism creates a small lumen and is postulated to cause less flow turbulence in the healing vessel and therefore, less restenosis. Rotational atherectomy has distinct mechanical advantages for calcified lesions in the native coronary circulation. The 6-month restenosis rate after PTCRA approximates the rates observed after standard PTCA. In some circumstances, the PTCRA restenosis rate exceeds the expected PTCA rate. According to reports from the multicenter registry for rotational atherectomy, the overall restenosis rate is about 38%. Predisposition to restenosis after PTCRA occurs in patients with diabetes and those with a poor initial increase in lumen diameter after the procedure.

Ostial stenosis of a native coronary artery represents an anatomic circumstance in which PTCRA confers a procedural advantage over PTCA, with restenosis rates approximately 39–43%. The only comparative study of balloon versus Rotablator failed to show any improvement in the restenosis rate despite better initial angiographic success.

IN-STENT RESTENOSIS

In-stent restenosis (ISR) is primarily due to neointimal hyperplasia produced by vessel injury of the balloon and/or stent struts. The injured segments promote activation of platelets, mural thrombus, and inflammatory cells. Vascular injury, mural thrombus, and a metallic foreign body activate circulating neutrophils and tissue macrophages. These elements

release cytokines and growth factors, activating smooth muscle as well as stimulating upregulation and expression of genes promoting cell division, such as c-*myc*, leading to further cell proliferation. Metalloproteinases are produced, leading to increased matrix material and remodeling of the extracellular support matrix, initiating smooth muscle cell migration. Uncontrolled proliferation of vascular smooth muscles into the vessel intima and the deposition of extracellular matrix leads to significant in-stent luminal narrowing 3–6 months after PCI.

There are two major categories of in-stent restenosis—focal and diffuse (Fig. 7-6)—and within each category several

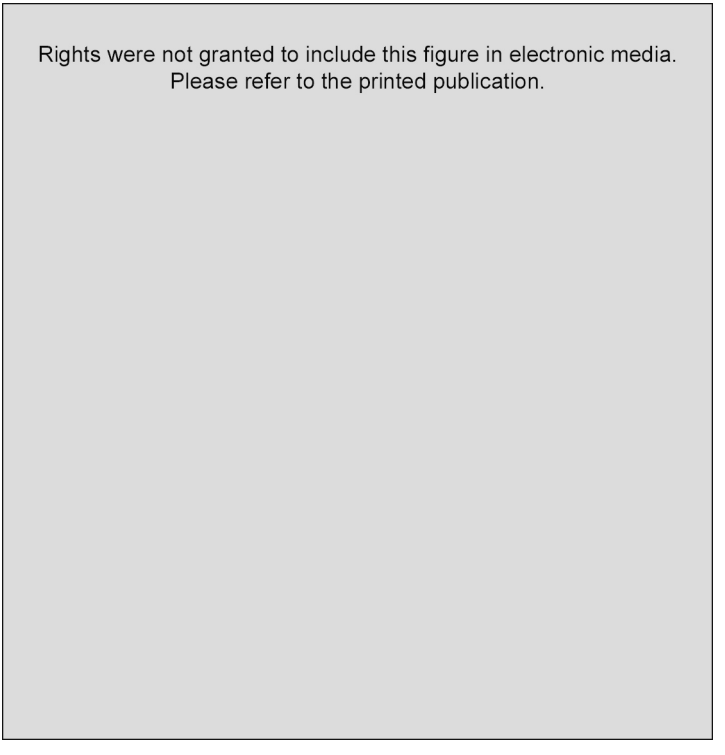

Fig. 7-6 Classification system proposed for in-stent restenosis. (From Mehran R, Dangas G, Abizaid AS, *et al.* Angiographic patterns of in-stent restenosis classification and implications for long-term outcome. *Circulation* 1999;100:1872–1878.)

subtypes of responses relative to the proliferation within and/or adjacent to the stent are noted.

Figure 7-7 suggests a scheme for the treatment of ISR.

CONVENTIONAL MANAGEMENT OF RESTENOSIS

Balloon Angioplasty

Restenosis can be treated successfully with repeat balloon angioplasty. The success and complication rates are lower than for the initial procedure because the restenosis lesion is primarily fibroproliferative rather than an atherosclerotic plaque. Restenosis after a second angioplasty ranges from 30% to 35%, although sustained revascularization may be present in more than 80% of patients after two procedures. Early (<3 months) restenosis, proximal left anterior descending locations, and multivessel disease increase the restenosis risk after a second angioplasty procedure.

Stents

Endovascular stents were developed to prevent abrupt closure and restenosis after angioplasty. Stenting improves the long-term angiographic and clinical outcome, compared to standard balloon angioplasty. Stenting of restenotic lesions is readily

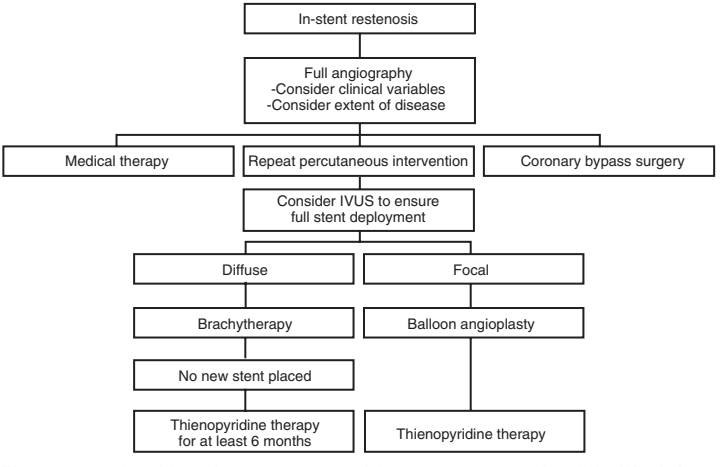

Fig. 7-7 Algorithm for treatment of in-stent restenosis. (Modified from Topol EJ. *Textbook of interventional cardiology*, 4th ed. Philadelphia, PA: WB Saunders, 2003: 468.)

performed but, in general, restenosis is not significantly reduced.

Key Points for Management of In-stent Restenosis

- Intermediately severe ISR lesions should be associated with evidence of ischemia related to the territory (use stress test or fractional flow reserve measurement [FFR]) before treatment
- If not using brachytherapy and lesion is focal; first dilate with standard angioplasty or cutting balloon
- After balloon, use intravenous ultrasound to decide whether to re-expand stent to true vessel size
- If stent not well expanded, use slightly oversized balloon and redilate
- Consider using cutting balloon because blades provide excellent stability inside stent
- If edge restenosis is present, additional stents may be needed.

BRACHYTHERAPY

Vascular brachytherapy, using beta and gamma emitters for treating percutaneous coronary intervention (PCI)-related restenosis, and especially in-stent restenosis, has demonstrated safety and efficacy. Routine use of brachytherapy depends on its continued efficacy in reducing complications such as late thrombosis and edge effects after radiation therapy. Operators need to optimize dosimetry, provide prolonged antiplatelet therapy, and apply adequate radiation margins to obtain the best brachytherapy results.

Basic Concepts

Radiation Sources. The external application of radiation to treat in-stent restenosis is limited by the high dosage, the inability to localize the dose to the treatment volume, and the moving target of the coronary artery. For coronary interventions, brachytherapy uses a radioactive source placed intravascularly adjacent to the angioplasty-treated segment. The radiation may be delivered by stents or a movable source of fluid, gas, or solid pellets.

Two types of radiation source are currently in use; beta and gamma radiation. Beta particles are high-speed electrons emitted from the nucleus of an unstable atom. Beta particles have a high-energy but low penetration, so that the dose

Table 7-1

Radiation Particle Physics

The result of a nuclear reaction is a release of small particles and/or energy

Particle	Emitted Quantity	Component
Alpha	Particulate	2 protons and 2 neutrons
Beta positive	Particulate	Positron
Beta negative	Particulate	Electron
Gamma	Electromagnetic	Photon

reduction over distance from the source is rapid. Gamma rays are high-energy protons emitted from the nucleus of an unstable atom. The penetration of gamma rays is higher than that of beta rays, with a longer dose reduction from the source distance. Gamma radiation provides increased total body dose to the patient and requires more extensive radiation protection measures. The dwell times of gamma sources are longer than those of beta radiation, approximately 15 minutes versus 3 minutes. To limit radiation exposure during gamma source dwell time, catheterization laboratory staff must leave the room.

The advantage of gamma radiation is its higher tissue penetrance, with a centering device being unimportant. Initial positive trials using gamma radiation have been reported for in-stent restenosis. Tables 7-1–7-4 summarize important concepts of radiation particle physics.

Mechanism of Action. The mechanism of action of brachytherapy is identical for all types of radiation particles. Radiation particles disrupt DNA by breaking strands in

Table 7-2

Radiation Physics—Characteristics of Particles

Particle	Penetrating Distance	Shielding	Clinically Useful
Alpha	Very short	Yes	No
Beta positive	Short 1–2 cm	No	No
Beta negative	Short 1–2 cm	Yes	Yes
Gamma	Far in meters	No	Yes

Table 7-3

Comparison of Beta and Gamma Brachytherapy

Characteristic	Beta	Gamma
Quantity emitted	Particulate	Electromagnetic wave
Energy	Low energy	High energy
Delivery time	Short, 2–10 min	Long, 15–30 min
Efficacy	Poor in large vessels	Broad spectrum of vessel sizes

cross-linkages. Breakage of these bonds leads to cell death at the next mitosis or cell division. Myofilaments in the adventitial layer and cells that migrate and transform into smooth muscle cells are the most likely targets of brachytherapy. Adventitial targeting for brachytherapy appears to be the optimal penetration depth.

Dosimetry. The dose of radiation is important to the results of the procedure. Too low a dose is ineffective or even becomes stimulatory to neointimal growth, and too high a dose is associated with aneurysm formation due to wall thinning and weakening. Long-term fibrotic complications have not been seen in patients. The therapeutic window for brachytherapy dosage remains under study but appears to be relatively narrow. The gray (Gy) is the unit that measures the mean energy imparted by ionizing radiation in a given volume divided by the mass of the target in that volume. The therapeutic dose of brachytherapy appears to be between 10 and 18 Gy at 1 mm from the endoluminal surface. Dosimetry is more difficult with beta sources because of low penetrance and vessel motion. Because of its low penetrance in the tissue, beta radiation has advantage of less radiation exposure to the laboratory staff and patient. Centering

Table 7-4

Brachytherapy Isotopes

Isotope	Emission	Half Life (days)	Dwell Time (min)
Iridium-192	Gamma	74	>25
Phosphorus-32	Beta	14	1–8
Strontium-90	Beta	28	2–4

catheters to deliver a homogeneous dose to the target area is important when employing beta radiation.

Edge Effect. The first descriptions of the edge effect related to radioactive stents. The lack of success of radioactive stents can be explained by the fact that the source could not cover the entire injured area, which occasionally extends a few millimeters beyond the stent, and this results in geographic miss and edge stenosis. The geographic miss can also occur with catheter-based brachytherapy but can be controlled by extending the treatment margins beyond the injured segment (Fig. 7-8). Animal and clinical studies suggest that a minimum of 5–10 mm from each end of the injured segment will eliminate the edge effect. With the drug-eluting stent, the drug is confined to the stent platform and could potentially leach from the proximal end of the stent. Thus brachytherapy may be used to treat the edge effect phenomenon in drug-eluting stent restenosis.

Brachytherapy Systems. The two most common systems in use today involve a solid radiation source deployed at the target

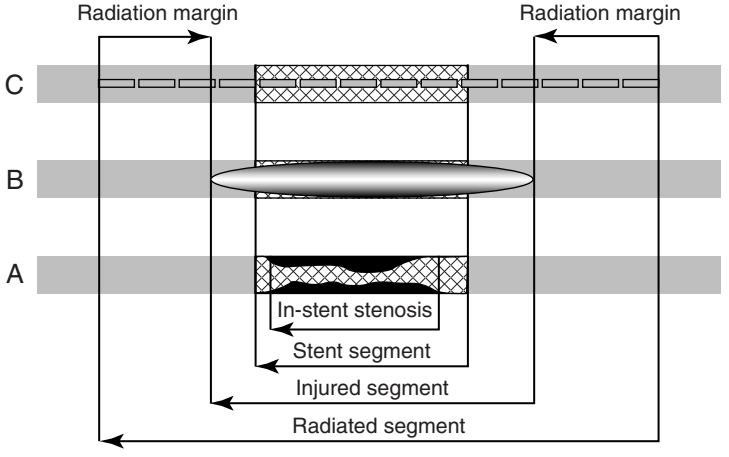

Fig. 7-8 Definition of vessel segments. **A,** Preprocedural in-stent stenosis in stent segments. **B,** Angioplasty determines the injured segments. **C,** After angioplasty, the radiation source is placed in the vessel. (From Cheneau E, Wolfram R, Leborgne L, Waksman R. Understanding and preventing the edge effect. *J Interv Cardiol* 2000;16:1–7.)

and delivered by catheter, subsequently removed after a prespecified dwell time. Two sources have been employed.

- A wire type with radiation seeds embedded in the distal portion
- A train of cylindrical sealed sources, which is flushed in and out of a blind end delivery catheter.

Other types of system are in development that use self-centering balloons and liquid or gas radiation. Balloon systems have the potential for radiation contamination if there is balloon rupture. Radioactive stents have also been developed. These stents are delivered using an identical technique to that for standard stenting. Table 7-5 summarizes FDA-approved brachytherapy systems.

Licensing Issues. Coronary brachytherapy must be performed with appropriate licensing and regulatory protocols in place. The institutional safety committee for use of radioactive materials must be informed and participate in the delivery of brachytherapy. Licenses for the appropriate use of radioactive material must be held by the operator and their support staff and be applied with approved radiation oversight protocols. Approvals must be obtained prior to performance of the procedure. These requirements are most easily fulfilled by a team consisting of an interventional cardiologist, medical physicist, and radiation oncologist. The team approach will

Table 7-5

FDA Approved Brachytherapy Systems—Clinical Indications and Usage

	Novoste Beta-Cath	Cordis Checkmate	Guidant Galileo
Lesion type	In-stent restenosis in native coronary artery	In-stent restenosis in native coronary artery	In-stent restenosis in native coronary artery
Lesion length	Treatable with 20 mm balloon	Treatable with balloon up to 45 mm	Treatable with balloon up to 47 mm
Vessel diameter	2.7–4.0 mm	2.75–4.0 mm	2.4–3.7 mm
Source	Beta (^{90}Sr or ^{90}Y)	Gamma (^{192}Ir)	Beta (^{32}P)
Contraindications	Cannot take antiplatelet or anticoagulants	Cannot take antiplatelet or anticoagulants	Cannot take antiplatelet or anticoagulants

provide the satisfactory licensing and expertise to perform this procedure on a daily or weekly basis.

Clinical Trials

Gamma Brachytherapy. The efficacy of intracoronary gamma radiation therapy in reducing angiographic and clinical restenosis in patients with in-stent restenosis has been demonstrated in the SCRIPTS, WRIST, and GAMMA 1 trials. The only gamma emitter used in clinical trials for in-stent restenosis is iridium-192 (^{192}Ir). Initial clinical studies recognize the limitations of gamma radiation, which include high activity and exposure, long treatment time, and the need for special shielding. In the long WRIST high-dose study, a higher radiation dose of 18 Gy as compared to 15 Gy was delivered 2 mm from the center of the radiation source in 120 patients. The 6-month followup with 6 months of antiplatelet therapy had a target vessel revascularization rate and major adverse cardiac event rate of 17% compared to overall adverse event rate of 36% in a low-dose group. Intravascular ultrasound examination at 6 months followup demonstrated the greatest minimal lumen area in the high-dose patients (4.0 ± 1.4 mm^2 compared to 2.9 ± 1 mm^2). These results indicate that, if the magnitude of these reductions persist, 3 Gy more for each isotope will achieve a single-digit restenosis rate, making the brachytherapy results comparable to some of the reported drug-eluting stent results.

Beta Brachytherapy. The development of intracoronary radiation using beta emitters has eliminated problems related to gamma radiation exposure. The START and INHIBIT trials for in-stent restenosis have provided the data for approval of the strontium-90 Beta-Cath systems. The positive results with Beta-Cath in-lesion segment analysis and the strong trend in clinical outcomes in the PTCA arm indicate a role for strontium-90/yttrium radiation in the treatment of *de novo* lesions. Direct stenting of *de novo* lesion and beta cath irradiation resulted in 15% restenosis for both the stented and analyzed segments, suggesting that the edge effect can be eliminated with correct dosing. Preliminary results from the SVG BRIGHT study with the use of the RDX system using phosphorus-32 radiation for the treatment of *de novo* lesions

in saphenous vein grafts had 0% restenosis and 11% late loss. Vascular brachytherapy can be utilized as an adjunct to ablative therapy, such as laser and atherectomy, when stents are not considered. Figure 7-9 summarizes results of several important clinical trials in coronary brachytherapy.

Late Thrombosis of Vascular Brachytherapy. Late thrombosis occurring more than 30 days after radiation therapy is one of the major complications of vascular brachytherapy (Box 7-1). Late thrombosis in early clinical trials was reported in up to 14% of patients. Late thrombosis also occurs with other vascular brachytherapy and relates to the healing arrest and lack of stent re-endothelialization. An effective strategy to prevent late rethrombosis is limiting restenting at the time of radiation treatment. It is essential to administer at least 12 months of antiplatelet therapy, preferably clopidogrel in addition to aspirin, for all radiation cases, both beta and gamma emitters.

De Novo *Lesions.* The results of beta and gamma radiation in the treatment of patients with in-stent restenosis are being extended to *de novo* lesions. The first pilot study demonstrated mixed results, with the presence of aneurysms between 60 days and 6 months. The overall stability in the clinical angiographic

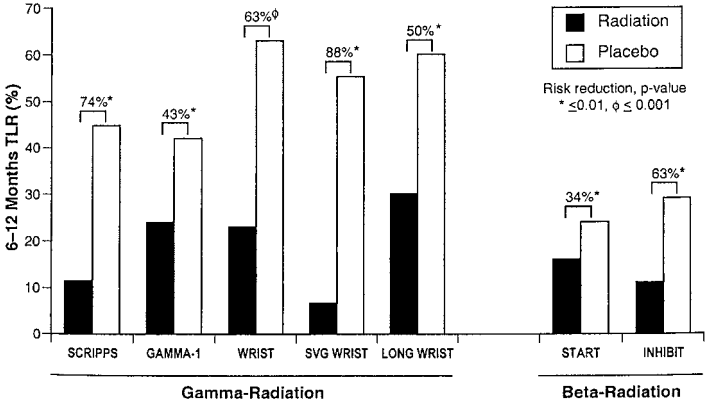

Fig. 7-9 Current clinical trials of gamma and beta brachytherapy for coronary restenosis. Each trial demonstrates significant reduction of repeat revascularization within 12 months after PCI. (From Topol EJ. *Textbook of interventional cardiology*, 4th ed. Philadelphia, PA: WB Saunders, 2003: 438.)

Box 7-1

Adverse Effects of Brachytherapy

- Edge effects
- Late thrombotic occlusion
- Stent vessel separation
- Long-term effects

results was maintained. Initial beta studies, such as the GENEVA trial using yttrium-90 and the BERT trial (Beta Energy Restenosis Trial) with strontium-90/yttrium, and the repeat 32 PREVENT trial (Proliferation Reeducation with Vascular Energy Trial) demonstrating late loss index and binary restenosis with radiation treatment showed the benefit of brachytherapy. The beta radiation in the EUROPE trial (BRE), REGISTRY, and the Beta-Cath randomized trial demonstrated a high rate of geographic miss (≥80%). The right dosimetry and adequate coverage of the injured segment mean that binary restenosis can be minimized, up to 4% and 15% for stented lesions with combined restenosis of 9%.

Peripheral Vascular Disease. Brachytherapy should limit the neointimal formation following vascular injury in peripheral arteries. The treatment strategy most applicable to superficial femoral arteries was explored in Frankfurt, in 30 patients with in-stent restenosis and superficial femoral arteries. No safety issues have been reported 10 days after this trial. Additional applications of peripheral vascular brachytherapy include in-stent restenosis in renal arteries, arteriovenous dialysis shunt fistula, subclavian stenosis, and maintenance of patency in stents from Port-A-Cable shunt (TIPS procedure). Important considerations for peripheral vascular brachytherapy include the use of a centering catheter, dose adjusted rate for vessel size, and treatment of long lesions.

DRUG-ELUTING STENTS

Drug-eluting stents (DESs) have markedly reduced in-stent restenosis. In 2003, there will be several DESs available in the USA and even more in Europe. DESs are classified according to the specific stent design, the use of polymer for drug absorption, the type of polymer, the type of drug, and release

characteristics. Currently, paclitaxel adhered to the stent surface with no polymer is delivered by the V-Flex Plus stent (Cook & Guidant, Inc.), and a flex stent (JOMED), which has a narrow pore ceramic coating and no polymer, delivers tacrolimus. Paclitaxel will also be applied to the NIR Express stents of Boston Scientific. For sirolimus the polymer carrier for local drug delivery is attached to a Bx velocity stent. The BiodivYsio matrix stent can deliver dexamethasone, prednisone, batimastat, estrogen, and Angiopeptin. The Tetra stent delivers actinomycin D. The S-7 (Medtronic) stent will deliver c-*myc* antisense. Clinical trials of the different types of DESs are in progress.

Drug-eluting stents inhibit endothelial proliferation, thus reducing restenosis. The many antiproliferative drugs that can be attached to the stent surface depend on stent designs to reduce arterial injury, modification of stent cell configuration, strut thickness, and implantation technique. Three major components of DES systems are the stent design, the polymer or coating, and the antiproliferative drug.

Stent Design

Drug delivery is dependent on strut spacing, the number of struts, and the homogeneity of strut placement over the target surface. The geometry of the stent cells must provide enough surface area for delivery of the agent. The drug-carrying units of the stent cells must allow a sufficient area for diffusion to deliver optimal tissue drug levels. Biodegradable stents with temporary scaffolding and drug delivery during the healing process are being tested.

Coatings

Passive stent coatings to modify the surface characteristics of stainless steel have included ceramics, noble metals, polishing, thermal plating, and biochemical mimicry with phosphorylcholine or fibrin.

Polymeric materials act as drug repositories and allow for controlled drug release over time. Pharmacologic agents can be maintained in the polymer reservoir when covered by a film on or within a polymeric matrix. Drug release occurs through three mechanisms—diffusion, chemical reaction, or solvent

activation. Nondegradable polymers enable drug release by particle dissolution whereas biodegradable polymers permit drug diffusion in concert with matrix degradation.

For the current generation of DESs, drug release from the polymer occurs by passive diffusion. The Cypher stent is a Bx velocity stent (Cordis Inc.) covered by a 10 μm thick sirolimus-containing layer of nonbiodegradable methacrylate and ethylene-based polymer allowing release of 140 μg of sirolimus per square centimeter of stent over a 15-day period. A slow-release formulation eluting 80% of the drug over 30 days can be achieved by a polymeric diffusion barrier applied over the base code.

Another useful coating is phosphorylcholine. This moiety is a highly hydrophilic molecule with a major phospholipid "head" grouping identical to that found on the outer layer of normal cell membranes. Synthetic phosphorylcholine polymers absorbed onto stent surfaces and cross-linked to the stainless steel by gamma irradiation have shown that this coating inhibits absorption of proteins such as fibrinogen and albumin and reduces platelet adhesion and complement activation. In animal models, phosphorylcholine-coated stents did not incite an inflammatory response over that of uncoated stents, with the additional ability to allow drug release at the time of implantation. The BiodivYsio stent (Biocompatibles, Inc.) uses a 50–100 nm thick phosphorylcholine coating resistant to elongation during stent implantation and providing an excellent depot for drug. Early studies show favorable outcomes: incidents of subacute thrombosis 0.4%, target vessel revascularization 6.1%, and 6-month angiographic restenosis 17.7% (SOPHOS trial).

Drugs for Coated Stents

Drugs used for stent coatings to reduce neointimal proliferation should have a large therapeutic window, a low inflammatory potential, inhibit multiple mechanisms of the complex restenotic biology, and reduce smooth muscle cell proliferation without unacceptable toxicity to the medial and adventitial cell layers. Unlike radiation, local drug elution should not inhibit stent re-endothelialization. Drugs for coated stents should have favorable local pharmacokinetics and distribution properties. Hydrophilic drugs, such as heparin, permeate into

tissue but are rapidly cleared. Hydrophobic agents, such as paclitaxel or sirolimus, are insoluble in the aqueous phase and bind to hydrophobic sites on the arterial wall. Both hydrophilic and hydrophobic drugs have large spatial concentration gradients across the arterial wall, with hydrophobic drugs distributing better and more homogeneously than hydrophilic agents. The two most promising agents at this time for DES are sirolimus and paclitaxel.

Sirolimus. Sirolimus is rapamycin, a naturally occurring macrocyclic lactone discovered in the soil of Easter Island (Rapa Nui) in the 1960s. Rapamycin is a product of fermentation of *Streptomyces hygroscopicus* and was used as an antifungal antibiotic. Sirolimus blocks the cell cycle of proliferating cells binding to the high affinity cytosolic receptor protein FK506 leading to the inhibition of mammalian target of rapamycin (mTOR), which prevents downregulation of tumor suppressive cell P27. The gene *p27* inhibits cell-dependent kinase activity and blocks G1- to S-phase cell cycle progression. Sirolimus is lipophilic and easily crosses the cell membrane. The inhibition of mTOR suppresses T-cell proliferation and is a powerful antiproliferative and antimigratory agent acting on smooth muscle cells. Systemic sirolimus reduces neointimal proliferation after balloon injury in porcine coronary arteries. Local sirolimus administration inhibits neointimal proliferation (Fig. 7-10).

The clinical use of sirolimus in patients was supported by the RAVEL trial, randomizing 238 patients with single *de novo* lesions less than 18 mm in length with vessels of 2.5–3.5 mm to the Cypher Bx velocity stent containing 140 μg of sirolimus/cm^2 or an uncoated Bx stent. The patients were treated with standard post-stent regimens for 6 months. Neointimal proliferation in the DES was 0 as compared to 0.8 ± 0.53 mm in the standard stent group. Angiographic restenosis (≥50% reduction in diameter) was 0% in the sirolimus group and 26.6% in the control group. Target lesion revascularization at 1 year was 5.8% in the sirolimus group and 28.8% in the control group. Neointimal hyperplasia was also markedly inhibited in the sirolimus group by intravascular ultrasound substudy.

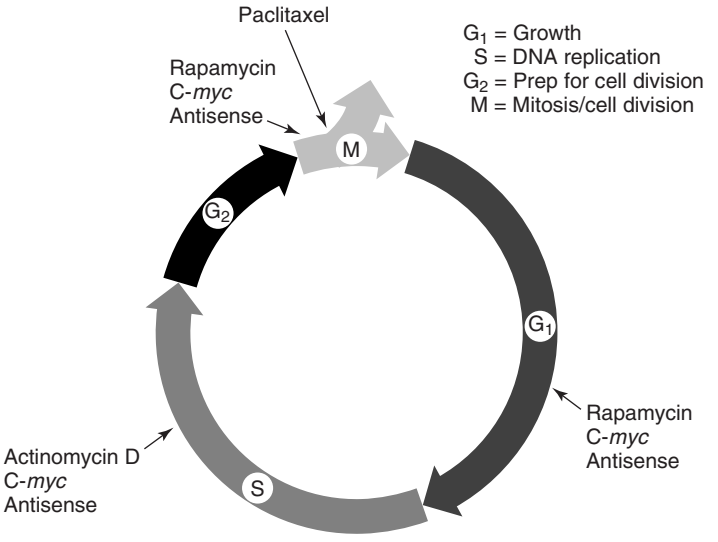

Fig. 7-10 Sites of action in the cell phase for selected pharmacologic stent coating to inhibit restenosis.

Similar results were found in the SIRIUS trial of 1101 patients, randomized to coated and uncoated stents. Binary restenosis was 3.2% in the sirolimus group and 35.4% in the control group. The in-stent restenosis within the margins of the stent 5 mm proximal or distal to the stent edge was 8.9% versus 36.3%. No evidence of edge effect was detected in this particular study. Stent thrombosis at 9 months in the two groups was 0.4% versus 0.8% (p = NS). The incidence of the combined endpoint of target lesion revascularization, cardiac death, or myocardial infarction at 9 months was 8.6% in the drug-eluting stent group and 21% in the control group. These studies demonstrated a significant reduction on in-stent restenosis with the sirolimus coated stent and support the application of this technique.

Paclitaxel. Paclitaxel is a powerful antineoplastic drug found in the Pacific yew tree (*Taxus brevifolia*) and is used in the treatment of malignant ovarian and breast cancer. Paclitaxel stabilizes polymerized microtubules and enhances microtubular assembly, forming unorganized and decentralized microtubules in the cytoplasm. Cell replication is inhibited

predominately in the G0/G1 and G2/M phase of the cell cycle. Paclitaxel is highly lipophilic, promoting rapid uptake through hydrophobic cell membranes and minimizing systemic loss. Paclitaxel is suitable for polymer-based delivery. It has long-lasting antiproliferative effects after a single administration, and can be directly applied to metal as a durable simple coating.

The Taxus I study randomized 61 patients with *de novo* coronary lesions to the paclitaxel-eluting NIRx stent or an uncoated NIR stent. The percentage diameter stenosis at 6 months was 13% in the paclitaxel group versus 27% in the uncoated stent group. The incidence of binary restenosis was 0% and 10% respectively in the two groups. In Taxus II, paclitaxel dosage was tested in 269 patients in the moderate-release arm, 267 patients in the slow-release arm and a control stent group. In the moderate-release arm, the in-stent volume obstruction was 7.8% versus 20.5% in the control group, with in-stent restenosis of 8.6% versus 23.8% in the control group. Target lesion revascularization was 6.2% versus 17.7% in the DES and control groups, respectively (all comparisons $p < 0.05$). Similar results were also produced in the slow-release arm.

The Future

When cost considerations are overcome, DES is likely to become a standard for PCI, reducing adverse events and the need for additional revascularization.

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8

DIFFICULT SITUATIONS IN PERCUTANEOUS CARDIAC INTERVENTIONS

Morton J. Kern and Morton R. Rinder

Difficult angioplasty can arise at anytime, even during the most straightforward procedure. For example, a simple mid-right focal coronary stenosis may dissect distally to involve a bifurcating posterior descending artery and posterolateral branch with myocardial ischemia and subsequent complications. A similar lesion could also be associated with an ostial dissection tracking upward toward the guide catheter and involve the aorta. A deeply seated guide catheter may be the cause of such a complication from this straightforward approach.

The initial approach to overcoming difficult percutaneous cardiac interventions (PCI) is planning and anticipation of problems. No operator should begin a procedure without having mentally performed all the necessary steps and visualize the optimal result.

The need for extra guide catheter support could be anticipated by the degree of difficulty experienced in seating the diagnostic catheter and from clues gained during the initial attempts to pass the guidewire.

Knowledge of guidewire difficulties will lead to extra caution during balloon catheter passage and eventually stent placement. For example, will the tortuosity encountered require a buddy wire or deep guide catheter seating to accomplish the task?

The additional concern of left ventricular function and the patient's tolerance for ischemia must be included in the

advanced planning for the procedure. The operator must address the need for additional arterial and/or venous access for intra-aortic balloon pumping or pacemaker placement. This ability to foresee difficult situations comes from good mentorship and case experience.

This chapter will review common approaches to commonly seen difficult PCI situations. Since many cases of difficult PCI also involve situations of higher or extreme risk, further discussion of these patients is provided in Chapter 9.

SIDE BRANCH AND BIFURCATION STENOSIS

Side-branch closure is the most concerning outcome of treating bifurcation lesions. The approach to bifurcation lesions is based on the angiographic configuration of the stenosis and the proximity of the side branch to the target lesion. Significant disease in the ostium of the side branch increases the clinical consequences of side-branch loss if closure should occur. The likelihood of side-branch closure is related most directly to the presence of disease in the ostium of the side branch.

Common combinations of side-branch plaque involvement during parent-branch PCI are shown in Figure 8-1. Side branches at *low risk*, those that are not likely to be compromised, include prestenosis branches, poststenosis branches, and those branches that do not straddle a stenosis. Angioplasty across an uninvolved side branch carries a less than 1% risk of occlusion. The requirement for side-branch protection for the three side-branch locations above (prestenosis, poststenosis, not straddling a stenosis) is minimal, as the technical difficulty of approaching the target branch is also low.

Bifurcation lesions that are at *high risk* for side-branch closure involve a side branch straddling the stenosis of the target vessel, and ostial stenoses of the side branch. The technical difficulty of treating these stenoses increases with the severity of side-branch narrowing. The risk of side-branch closure with an ostial narrowing approaches 15%.

When there is an equal distribution of coronary plaque across a bifurcation stenosis, dilatation of both branches and, in some cases, simultaneous balloon inflations in both branches to maintain vessel patency should be anticipated. Another balloon-only strategy involves sequential Cutting Balloons in each limb, which may prevent the significant

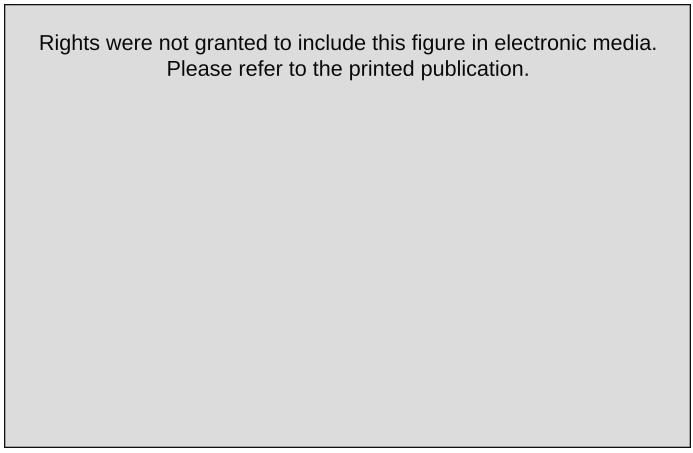


Fig. 8-1 Schematic representation of lesion and side-branch involvement. **A, B,** and **C** represent parent vessel involvement with no disease located in the side-branch vessel. **D, E,** and **F** represent parent and side-branch involvement with more than 50% ostial stenosis in the side branch. (From Freed M, Grines C. *Manual of Interventional Cardiology*. Birmingham, Michigan: Physicians' Press, 1992.)

plaque shifting associated with sequential plain balloon inflations.

Balloon angioplasty of one or both branches has been successful but long-term patency of high-risk lesions is low. For this reason many operators consider debulking the involved side branch with Rotablator or Cutting Balloon. A bifurcation "T" or "Y" stent does not provide greater side branch patency than single parent branch stenting with nonstent PCI of the side branch. Since most early studies with drug-eluting stents have excluded lesions with significant side branches, it is unclear whether there are any specific hazards associated with bifurcation stenting with two or more drug-eluting stents.

General Approach to Bifurcation Lesions

Guide Catheter Selection. A guide catheter should be selected considering whether large diameter equipment such as a Rotablator or Cutting Balloon is needed. Simultaneous balloon catheter and stent manipulations are easier with a large (7 or 8F) guide. Guide catheter internal dimensions

should be large enough to accommodate balloon/stent catheters and other PCI devices (e.g., Rotablator, Cutting Balloon). Guide catheters with internal diameters of 0.086 inch may accommodate two monorail balloon catheters and most stent systems. A single guide catheter with two guidewires for two balloon catheters (Fig. 8-2) is easier than two guide catheters and separate systems.

Balloon Catheter Selection. Two standard balloon catheters may be needed. Sequential inflations of two different-sized balloons are common. The simultaneous balloon inflation may be required to eliminate the shifting of plaque from one branch to the other that may occur with sequential inflations.

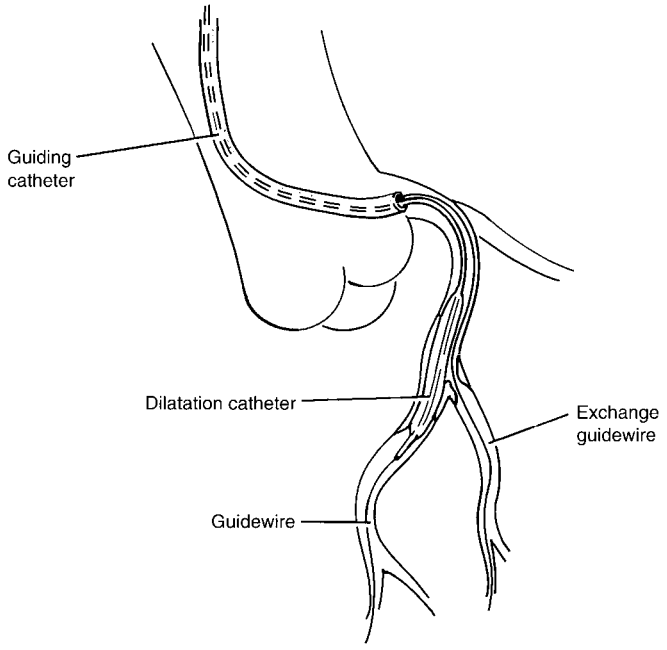


Fig. 8-2 Angioplasty procedure with single guide catheter, two wires, one balloon catheter. The exchange guidewire is positioned across the bifurcation in the vessel to be protected. The dilatation catheter is used for the principal lesion and the side-branch lesion in a sequential fashion. (Modified from Oesterle SN, McAuley BJ, Buchbinder M, Simpson JB. Angioplasty at coronary bifurcations: single-guide, two-wire technique. *Cathet Cardiovasc Diagn* 1986;12:57–63.)

Guidewire Technique. To protect side branches, two guidewires can be placed, one in the side branch and one in the main vessel, before beginning inflations (Fig. 8-3). The order of inflation is relatively unimportant. It may be helpful to keep a marker on one of the wires to reduce confusion during balloon inflations and wire repositioning. When using a two-guidewire system, the guidewires may become trapped after multiple wire manipulations because they have become entangled. Efforts should be made to avoid guidewire entrapment, which will prevent advancement of the balloon and may result in failure to recross the stenosis.

Adjunctive Pharmacotherapy. Meta-analyses from glycoprotein receptor IIb/IIIa inhibitor trials suggest that these agents can reduce the incidence of side branch occlusions when stenting or balloon only techniques are used.

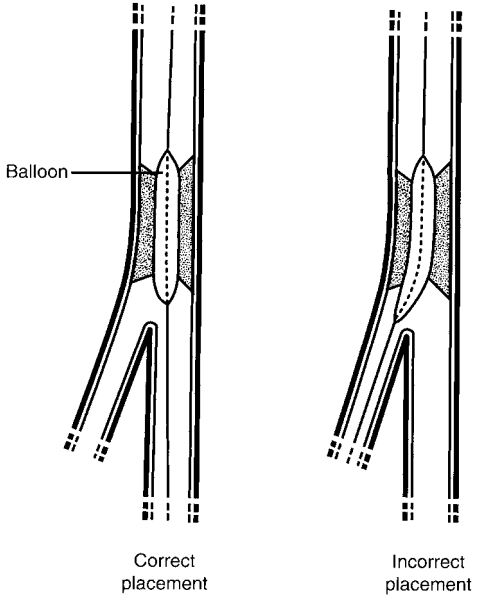


Fig. 8-3 Correct and incorrect placement of balloon catheter in guidewire for prebranch dilatation in a lesion with a proximate side branch. (From Kulick DL, Rahimtoola SH. *Techniques and applications of interventional cardiology*. St Louis: Mosby, 1991: 67.)

Anticipate a Dissection. An unprotected major vessel dissection will require reinstrumentation and jeopardize further attempts to open the side branch. Serial inflations, first in one branch then in the other, as opposed to simultaneous balloon inflations in both branches, may limit the need for extra maneuvers.

Sequential Branch Inflations (Table 8-1). Dilate the main vessel first, the side-branch second, and finish dilation in the main branch. A sequential main-side–main-branch inflation strategy provides a safe and straightforward approach. However, shifting of atherosclerotic plaque during sequential inflations may result in suboptimal main vessel dilation, requiring repeated dilatations. Simultaneous balloon inflations may be used when plaque shifting causes unsatisfactory results.

Simultaneous Balloon Inflations. When a shifting plaque causes opposite branch narrowing after sequential inflations, simultaneous balloon inflations are needed. The combined diameter of two balloons in the parent branch should be considered carefully in order to avoid dissection. The loss of the side branch must be weighed against potential damage to the main vessel from two simultaneous balloon inflations.

Nonballoon Interventions. Bifurcation lesions have been addressed with rotational ablation and Cutting Balloon devices. The routine application of directional atherectomy is not recommended because of the directional cutting nature of the atherectomy catheter.

Bifurcation Stenting. If the decision to stent both branches is made, several techniques can be used depending on the angle of the side branch origin. Keep in mind that none of the techniques described provides "better" results. "T" or "Y"-stenting is used when the angle of origin approaches 90° and the side branch is smaller than the main vessel. "V" stenting can only be used when the proximal main vessel is sufficiently large to accommodate two stents. The "Culottes" technique provides excellent coverage but is a challenge even for experienced interventionalists. Premounted bifurcation stents will be commercially available in the future. Figure 8-4 shows a stepwise approach to bifurcation stenting from Pan *et al.*

Table 8-1

Approach to Bifurcation Stenosis

Approach	Advantages	Disadvantages
Guide catheter selection		
Two-guide catheters	Large variety of catheters available	Two artery punctures Two-guide catheter manipulation Long procedure time
One-guide catheter	One arterial puncture Fewer catheter manipulations, low risk of ostial trauma Reduced procedure time	
Balloons and guidewires		
Two wires, one balloon catheter	Maintain access for balloon upsizing, perfusion catheters, or stents Less obstruction to protected side branch coronary flow than deflated balloon-on-a-wire catheter Less expensive Good vessel opacification	
Two balloon-on-a-wire catheter	Immediate dilatation capability during acute closure	Expensive Must use guidewires for exchanges
One over-the-wire balloon, one on-a-wire catheter	Allows immediate dilatation of side branch Reduces procedure time	Expensive Limited fixed-wire exchanges
Balloon inflation strategy		
Sequential balloon inflation	Uses same balloon for both vessels	More catheter manipulations
Simultaneous balloon inflations	Minimizes atheroma shifting to opposite branch Allows dilatation without oversizing the balloon relative to small postbifurcation vessel diameter	More balloons and inflation devices

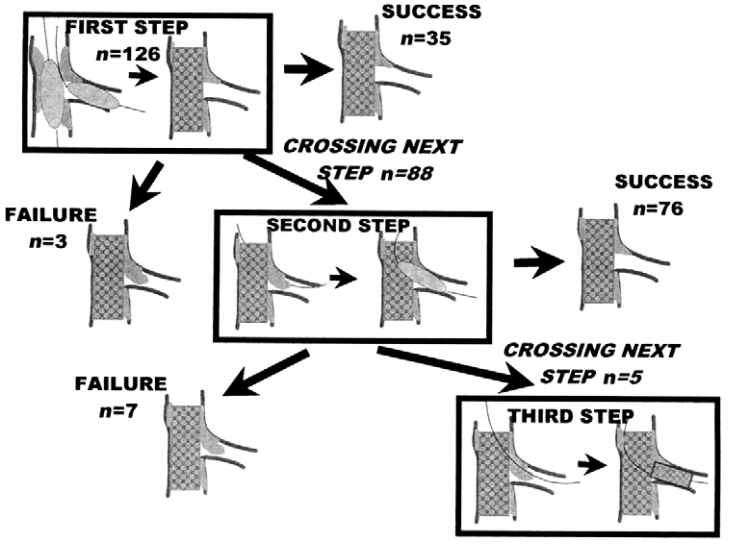


Fig. 8-4 Steps undertaken for bifurcation stenting. Success indicates a number of patients in whom results were acceptable. Failure indicates number of patients in whom additional steps were required. (From Pan M, Suarez de Lezo J, Medina A, *et al.* A stepwise strategy for the stent treatment of bifurcated coronary lesions. *Catheter Cardiovasc Interv* 2002;55:50–57.)

Key points for bifurcation stenting are shown in Box 8-1.

ECCENTRIC STENOSES

An eccentric lesion is a stenosis with an oval configuration and a lumen-to-vessel centerline position ratio of more than 0.7 (Fig. 8-5). Moderate to severely eccentric stenoses are considered to be complex B2-type or C-type lesions, depending on the involvement of side branches, calcification, vessel

Box 8-1

Key Points for Bifurcation Stenting:

- Use two wires if side branch loss is important
- Dilate smaller branch first or use Rotablator or Cutting Balloon
- Dilate and stent main branch; reassess side branch
- Redilate side branch
- Stent side branch through main stent only when absolutely necessary

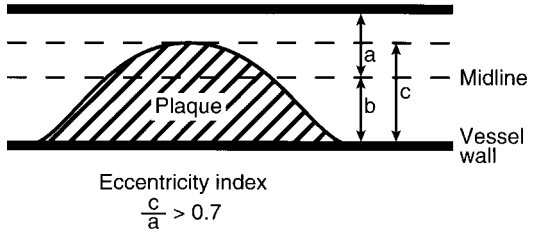


Fig. 8-5 Schematic description of lesion eccentricity.

complexity, and morphology. Outcomes for eccentric lesions are related to calcification lesion length (>20 mm), vessel tortuosity, thrombus, and vessel diameter. For most lesions stenting has been performed with acceptable long-term success rates. Long-term outcomes for directional atherectomy, thought to remove eccentric plaque more efficiently, are inferior to stenting.

SEVERELY CALCIFIED STENOSES

Percutaneous Transluminal Coronary Angioplasty and Stenting

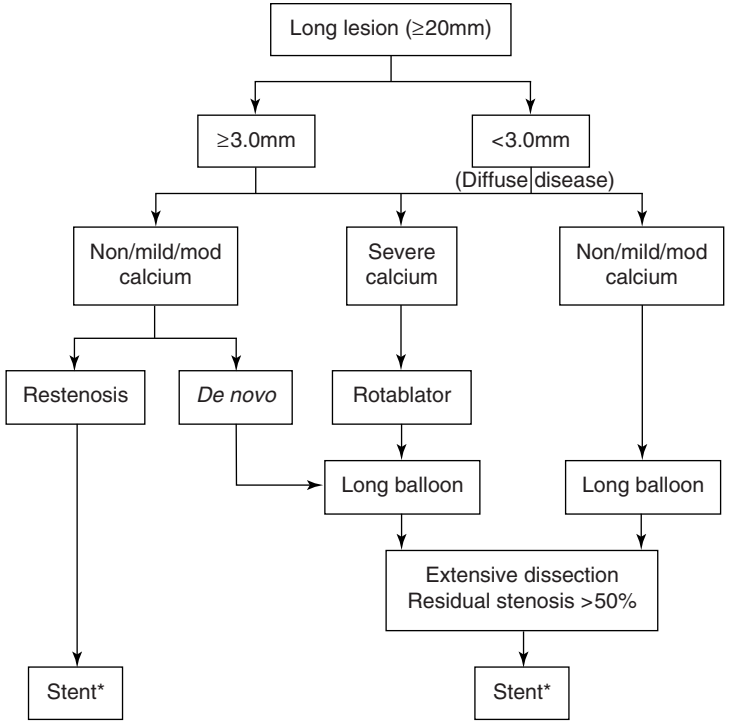
Balloon angioplasty of heavily calcified stenoses stretches the noncalcified vessel wall, promoting dissection (or rarely rupture) originating at the border region of the calcified and elastic regions of the stenosis. Severe calcification is associated with reduced primary success rates and increased complications. Because balloon angioplasty of calcified lesions requires high-pressure inflations (>15 atm), noncompliant balloon catheters are preferred. However, heavily calcified lesions may puncture polyethylene terephthalate (PET) balloon material. Entrapment of ruptured balloon material, although rare, has been reported, making removal of the balloon catheter difficult. In calcific lesions, direct stenting without balloon dilation may result in incomplete stent deployment and a potentially ruptured and entrapped balloon catheter.

Rotablator

Because of the increased complications and dissection rates, Rotablator ablation is the technique of choice for heavily calcified lesions. Cutting of a calcified artery segment may be

difficult or impossible with Cutting Balloon or directional coronary atherectomy.

Technique for Long Diffuse Lesions. Angioplasty of long, diffuse lesions with or without calcification can be managed by long balloon catheters, Rotablator, and stents. After rotablation, balloon lengths of 30 mm and 40 mm followed by similar lengths of stent can cover long diseased segments, reducing the risk of dissection. A graduated dilation approach can be used by introducing progressively larger inflation balloons. If the lesion is heavily calcified, Rotablator should be used first (Fig. 8-6).



*One long stent preferred

Fig. 8-6 Potential approach to the treatment of long lesions and diffuse disease. (From Topol EJ. *Textbook of cardiovascular medicine*. Philadelphia: WB Saunders, 2000: 376.)

Box 8-2

Key Points for Calcified Lesions

- Use Rotablator first – be conservative with burr sizing
- Predilate before stenting
- Use firm backup support
- Use high pressure to implant stent
- Consider intravascular ultrasound to ensure full stent expansion

Key points for calcified lesions are shown in Box 8-2.

OSTIAL LESIONS

Narrowing of the vessel ostium presents a difficult problem for two reasons: (1) technique and (2) long-term outcome. Many ostial lesions limit guiding catheter support. Balloon inflations in the coronary ostial locations have the potential for aortic dissection, especially during right coronary artery (RCA) angioplasty or for dissection of the left main artery from dilations in the ostium of the circumflex or left anterior descending arteries. Ostial lesions are best managed by rotablation and stent. Balloon angioplasty works, but is considered a suboptimal technique.

Types of Ostial Lesion

Aorto-Ostial Stenoses. The most common ostial stenosis is the aortocoronary lesion, which involves the opening of the vessel from the aortic cusp. The origins of the left main or right coronary artery, or a saphenous vein graft, are the most common ostial lesions.

Branch Ostial Stenoses. A coronary branch ostial stenosis is a narrowing at the origin of the branch takeoff from a main coronary vessel. Left anterior descending coronary ostial, diagonal, or circumflex marginal ostial lesions are common. One of the most difficult lesions is the ostial left anterior descending artery. Angioplasty of this lesion may cause trauma to the left main artery during device delivery or deployment. A left anterior descending stenosis within 2–3 mm of the origin should be considered as an ostial lesion with similar technical risks, because most angioplasty devices are designed to cover vessel segments more than 10 mm in length.

Techniques for Ostial PCI

Because elastic recoil is very common in aorto-ostial or ostial branch balloon angioplasty, the effectiveness of any balloon catheter technique is limited. Rotational atherectomy and stents eliminate elastic recoil, with improved results.

Guide Catheter Selection. Angioplasty of right coronary artery ostial lesions is the most difficult, because standard right Judkins catheters often cannot provide satisfactory device support. Configurations such as modified Amplatz (left or right), multipurpose, Arani, or El-Gamal catheters have been used successfully. Ostial occlusion by the guide catheter, as seen by damping of arterial pressure, requires guide catheters with side holes. While these are adequate for coronary perfusion after re-establishment of a patent lumen, side holes permit contrast to escape and may limit coronary visualization, especially when using large devices (e.g., rotablation atherectomy).

Careful engagement of the guide catheter should minimize aortic trauma to avoid complicating a difficult procedure. The use of Rotablator catheters requires nearly coaxial alignment of the guide catheter upon device entry in the ostium. Significant angulation between the guide catheter and the ostial takeoff will reduce procedure success.

Balloon Catheter Placement. After Rotablator, balloon angioplasty requires seating such that, during inflation, the balloon will not be ejected from nor compressed forward past ("watermelon seed") the coronary ostia (Fig. 8-7). Removal of the guide catheter into the aorta immediately before balloon inflation will permit the balloon to be inflated partly in the coronary ostium and the aorta and outside the guide catheter. The lesion will be appropriately spanned by the two ends of the balloon inflating at equal pressure. Inflation in the guide may result in failure of the distal end of the balloon to inflate properly. Stenting the ostial lesion may produce strut "hangout" into the main vessel, a common complication of ostial branch stenting, especially when angiographic angles do not allow good visualization of the takeoff.

Figure 8-8 demonstrates good stent techniques for RCA ostial stenting. Recovering access to the main vessel after ostial

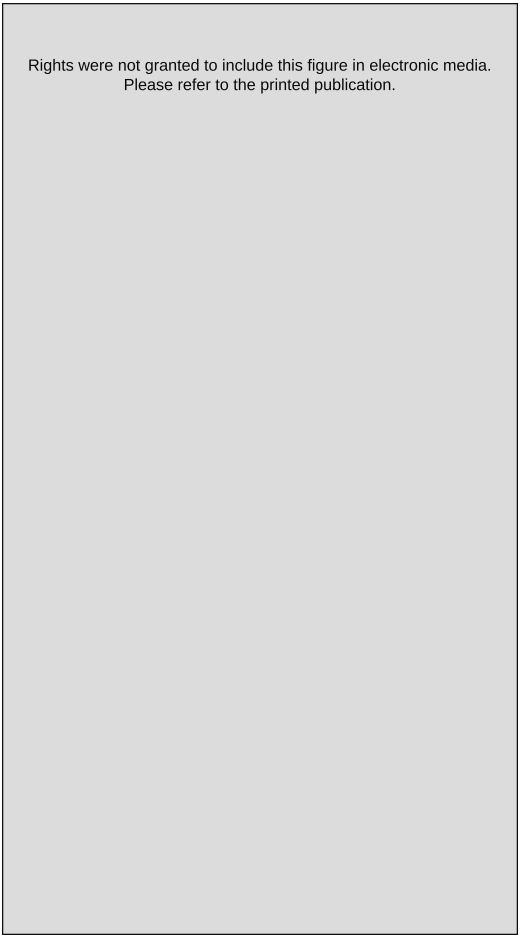


Fig. 8-7 **A,** Effect of squeezing the balloon out of an ostial lesion. The top panels show the balloon being ejected from the ostium, and the lower panels show the balloon advancing inside the artery during inflation. **B,** Proper guide catheter positioning helps to seat the balloon in an ostial lesion. (From Safian R, Freed M, eds. *The Manual of Interventional Cardiology*, 3rd edition. Birmingham, Michigan: Physicians' Press, 2001, p. 267.)

stenting can be difficult depending on the angle of the ostium origin and the amount of stent. A strategy to prevent strut hangout uses a main vessel balloon inflated at low pressure (balloon: artery ratio 0.7:1), placed prior to branch stent deployment. By pulling the ostial stent back against the

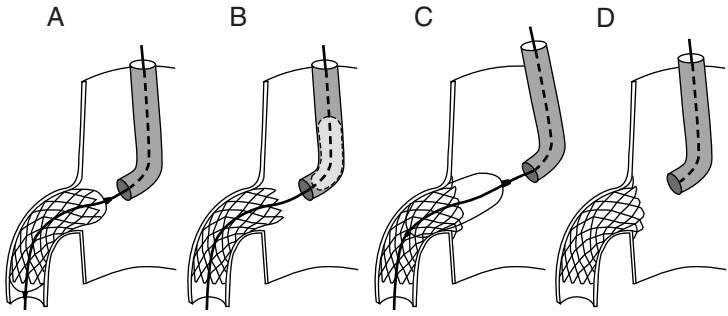


Fig. 8-8 Aorto-ostial lesions: stent technique. A, Position the stent-delivery balloon so 1–2 mm of stent extends into the aorta. The guide must be retracted 1–2 cm before deploying the stent. **B,** Remove the delivery balloon while maintaining backward tension on the guide, to prevent it from advancing into the ostium and damaging the stent. **C,** Perform adjunctive percutaneous transluminal coronary angioplasty with a high-pressure balloon to ensure full stent expansion and apposition. Flaring the proximal end of the stent with a slightly larger balloon is useful. **D,** Final result. (From Freed M, Grines C, Safian R, ed. *The new manual of interventional cardiology*. Birmingham, MI: Physicians' Press, 1996.)

inflated balloon and then deploying the stent, one prevents significant strut hangout into the main vessel.

A systematic approach to angioplasty of saphenous vein graft ostial coronary stenoses suggests using balloon and stenting (Fig. 8-9). For moderate to severe calcified lesions, rotational atherectomy is the technique of choice, followed by stenting.

Very Proximal Left Anterior Descending Stenosis PCI

A PCI complication of a very proximal or ostial left anterior descending artery stenosis can be life-threatening, especially if it involves the left main or circumflex ostium. Balloon dilatation and device manipulation in part of the left main coronary artery segment is unavoidable. Since the circumflex branch is often diseased in patients with an ostial left anterior descending artery lesion, bifurcation technique and probable side branch closure should be carefully considered. A left main coronary dissection is a forerunner to catastrophic vessel closure before stent placement. Angioplasty balloon success in ostial lesions was 86%, compared to 90% for nonostial left

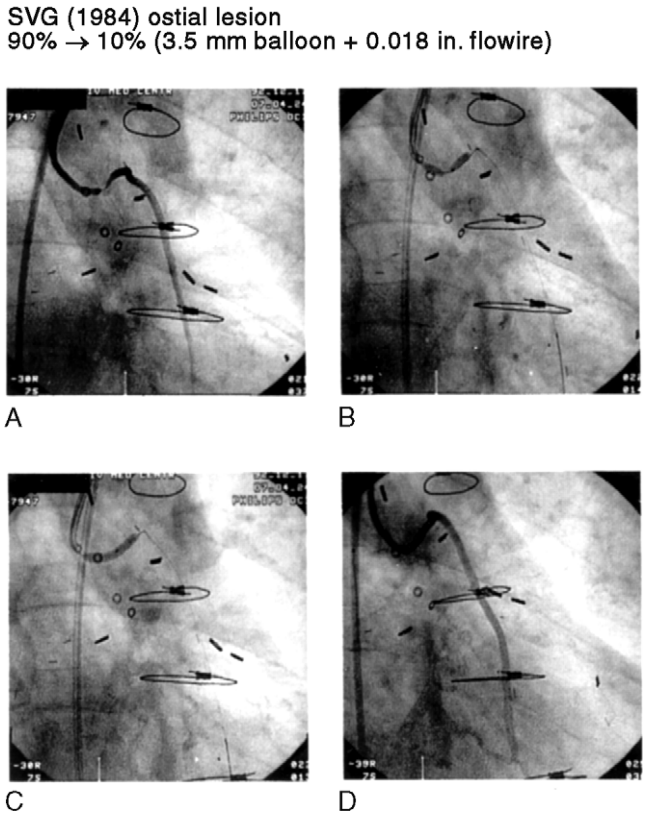


Fig. 8-9 Example of ostial coronary artery stenosis in a saphenous vein graft. **A,** The cineangiogram shows ostial stenosis in the right anterior oblique projection. An 8 French guide is positioned coaxially with the origin of the graft. **B,** A 3.5 mm high-pressure balloon was used to dilate the stenosis. **C,** The balloon is fully inflated. **D,** The ostial lesion after balloon angioplasty. This lesion is better treated with a stent placed primarily. (From Kern MJ, Donohue TJ, Flynn MS, *et al.* Interventional physiology: limitations of translesional pressure and flow velocity for long ostial left anterior descending stenoses. *Cathet Cardiovasc Diagn* 1994;33:50–54.)

anterior descending artery lesions. Although stent success is higher, no special technique for balloon angioplasty alone can be recommended. Based on the incidence of left main dissection and the potential for abrupt vessel closure, stent placement (with or without preceding Rotablator) has

superseded routine balloon angioplasty for ostial left anterior descending lesions. However, in the uncoated stent era, ostial left anterior descending artery stenting still has a high restenosis rate (>25–30%).

Key points for ostial lesion stenting are shown in Box 8-3.

TOTAL CORONARY OCCLUSIONS

The success rates for PCI of total coronary occlusions (TCO) ranges from 50% to 80%, with mortality rates of 0–2% and emergency coronary artery bypass surgery rates of 1–3%. Abrupt vessel closure following total occlusions may occur in up to 10% of patients, but may be clinically silent, depending on the collateral supply.

Total coronary occlusions are among the most technically challenging and largest group of complex lesions for which PCI is attempted. Procedural success rates for TCO depend on how long the artery has been occluded. For occlusions <3 months old, the success rate is higher (60% to 70%), compared to 50% to 60% for those older than 3 months. Other problems in dealing with total occlusions include the potential for distal embolization, closing collaterals, perforation, and rarely guidewire entrapment.

When crossing TCO, it is difficult to be sure that the guidewire is in the true lumen vessel rather than subintimal. One technique to insure proposition is to advance a small tracking or balloon catheter over the wire and remove the wire and inject contrast distally to verify the lumen. In rare cases, the severe narrowing will not permit a low-profile balloon catheter to cross the lesion. In this situation, a fixed-wire system (ACE balloon) may be successful to create a passageway.

Box 8-3

Key Points to Ostial Lesion Stenting

- Use Rotablator for calcified ostial lesions, especially of right coronary artery
- Use guide with side holes
- Use balloon long enough to span ostial lesion and remain inside artery and partly in aorta
- Use stents longer than 9 mm to prevent them from being "pulled out of position" by guide catheter manipulation
- Use larger balloon to flare aorto-ostial segment

Suitable Morphologies

The selection of chronic total occlusion lesions best suited to PCI should be based on favorable angiographic morphology (Fig. 8-10) such as:

- Tapered stumps
- Short length (< 10 mm) occlusion with evidence of antegrade angiographic flow
- An occlusion with a tapered (bird beak) shape
- Absence of bridging collaterals
- Absence of calcification.

Unfavorable morphologic features include:

- Flush or abrupt occlusions
- Long length of vessel occlusion
- Occlusions at side-branches with no tapering morphology
- The presence of bridging collaterals
- Heavy calcification.

Unfavorable features are associated with poor PCI outcomes. Retrograde collateral filling of the target vessel is helpful to identify the vessel course and lesion length. Stenting the TCO will provide the best long-term results.

Strategies for Total Coronary Occlusions

Strategies for PCI of total occlusions have included the use of extra stiff or coated (hydrophilic) guidewires, specialized laser guidewires, or radiofrequency guidewires (Fig. 8-11). None of these newer techniques have produced results exceeding those for conventional balloon/stenting approach.

Approaches to PCI for Total Occlusion of Saphenous Vein Grafts

Percutaneous cardiac intervention in an occluded saphenous vein graft represents a highly complex, high-risk procedure and requires a specialized approach. Bypass surgery should be strongly considered before selecting PCI methods for these conditions. The recanalization of chronically (>3 months), occluded saphenous vein grafts should not be considered unless there is an antegrade channel. For recent (<3 months) saphenous vein graft occlusions presumed to be thrombotic, thrombectomy catheters or thrombus aspiration systems (AngioJet) have been used successfully.

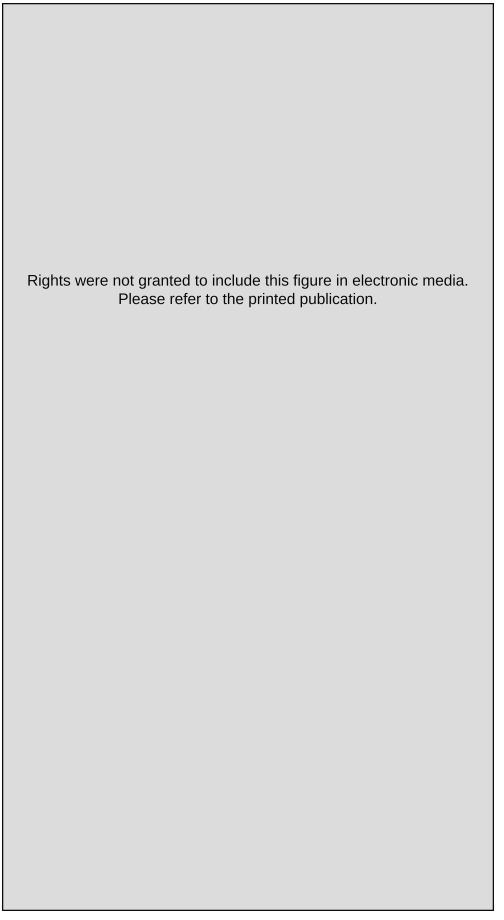


Fig. 8-10 Morphology of total coronary occlusion: favorable and unfavorable morphology for procedural success. (Modified from Freed M, Grines C, eds. *The Manual of Interventional Cardiology*, 3rd edition. Birmingham, Michigan: Physicians' Press, 2001, p. 295.)

Key points for total coronary occlusion PCI are shown in Box 8-4.

PCI OF CORONARY ARTERY BYPASS CONDUITS

Some 15–20% of all patients entering the catheterization laboratory have undergone previous coronary artery bypass graft surgery and may require further revascularization. For

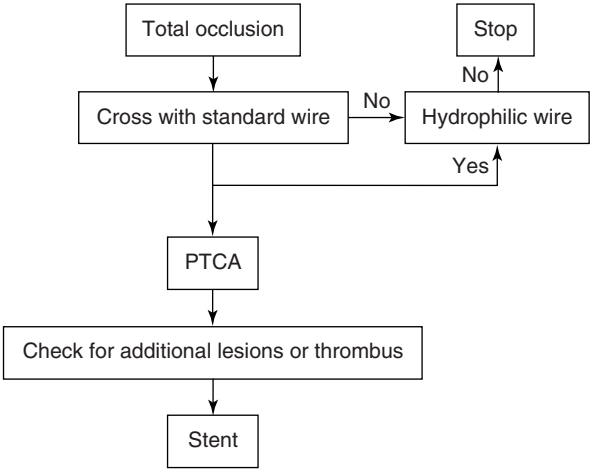


Fig. 8-11 Scheme for PCI of total chronic occlusions.

these patients, the objective of PCI is to provide symptom relief through re-establishing perfusion to ischemic zones instead of a second or third bypass operation. Repeat coronary artery bypass graft operations have increased operative mortality rates (2–8%), postoperative myocardial infarction rates (2–8%), and postoperative bleeding rates (1.3–11%). PCI for saphenous vein graft stenosis has a success rate of more than 85%, and rates for complications of less than 10%, urgent bypass surgery 4%, myocardial infarction 3%, and mortality 1–3%. The risks and benefits of saphenous vein graft PCI must be weighed against those of repeat coronary artery bypass graft surgery.

Compared to native arteries, PCI for saphenous vein graft stenosis has an increased risk of complications and lower long-term success rates, particularly if the vein graft has a

Box 8-4

Key Points for Total Coronary Occlusion PCI

- Limit attempts to open lesions with highly unfavorable morphology
- Use regular guidewires first before hydrophilic guidewires
- Confirm wire positioning in the coronary lumen before balloon inflation
- Balloon-dilate sufficiently to see extent of occlusion and additional lesions
- Stop if perforation occurs

degenerated appearance (usually an irregular surface or ulcerations on angiography), or if the graft is older than 3 years. Because the aorto-ostial anastomosis may be upward, flush or downgoing without aortic cusp support, guide catheter support can be compromised. Several technical aspects of saphenous vein graft PCI also contribute to lower success rates. Also, the saphenous veins may be too large for coronary stents (i.e., >4.5 mm diameter). No adjunctive pharmacotherapy has ever been demonstrated to reduce ischemia during saphenous vein graft PCI.

An especially critical consideration is the potential for distal embolization during saphenous vein graft angioplasty. Distal protection devices that limit the effects of embolization are not only useful but have resulted in a 50% reduction in peri-procedural complications. The one limitation of the currently available distal protection device (GuardWire™) is that it requires significant occlusion of the graft, thus causing poor visualization and some degree of ischemia.

Limiting the number of device exchanges and occlusion times is thought to improve outcomes. Thus direct stenting and minimal post-stent manipulation is recommended. Figure 8-12 shows an example of saphenous vein graft PCI.

The major factor associated with successful saphenous vein graft PCI is graft age. Grafts more than 3 years old are associated with lower success rates than grafts less than 1 year old. Saphenous vein grafts less than 1 year old are often narrowed more by thrombus than by plaque. Grafts more than 3 years old degenerate because of atherosclerotic material and are more prone to emboli than grafts under 1 year.

Because of the predisposition to occlude by thrombosis, embolization during PCI is the most common complication. Despite a satisfactory angiographic appearance, PCI associated with myocardial no-reflow may produce myocardial ischemia/infarction, a problem in 5–10% of procedures. Stenting is the preferred approach for saphenous vein graft lesions because of excellent early success rate and lower rates of restenosis, subacute closure, embolization, myocardial infarction, and death. Restenosis rates after saphenous vein graft stenting are approximately 25–30% at 1 year.

Lesion location within a saphenous vein graft is associated with different PCI success rates.

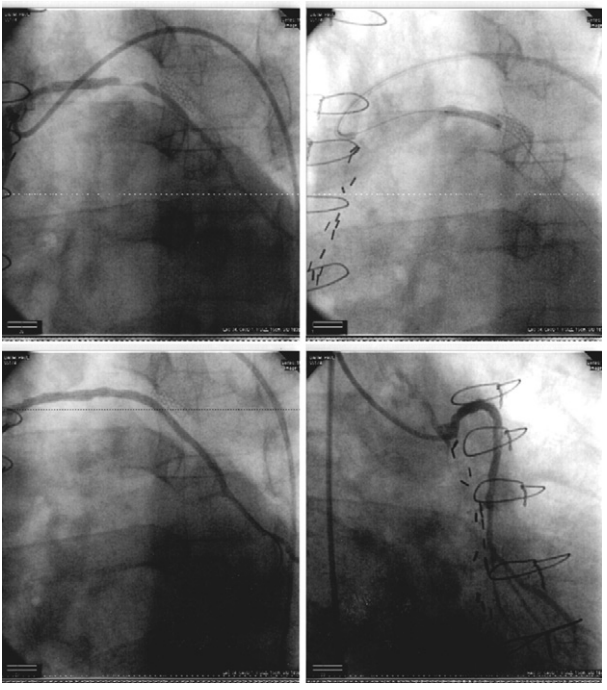
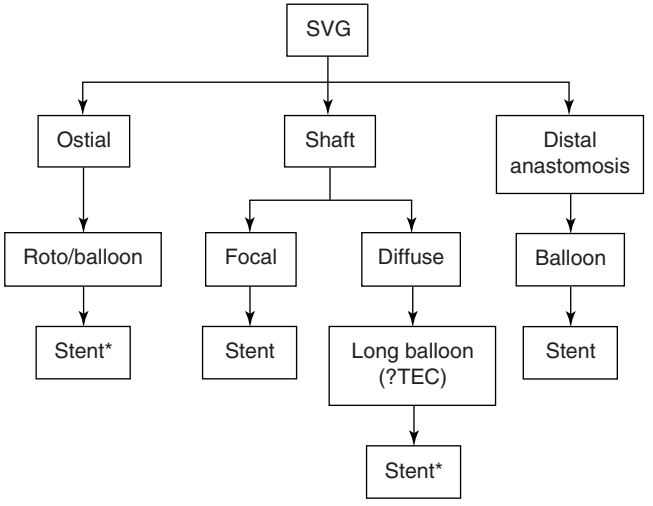


Fig. 8-12 Saphenous vein graft angioplasty: *Top left panel*, Lesion is seen in shaft of saphenous vein graft. Stent from prior interventions can be seen beyond lesion in additional vein. *Top right,* Stent positioned and expanded in lesion. *Bottom left,* Final result in a left anterior oblique view after stent placement. *Bottom right,* Final result in right anterior oblique view showing stent fully expanded.

- Aorto-ostial lesions have lower success and higher restenosis rates than mid-body stenoses
- Mid-body locations have lower complications and lower restenosis rates relative to aorto-ostial locations
- Distal saphenous vein graft–native vessel anastomosis sites have results similar to native vessels. The morphology of the stenosis carries the same implications in saphenous vein grafts as for those in native vessels, with an increased propensity for thrombogenic complications.

A strategy for saphenous vein graft angioplasty is described in Figure 8-13. Key points for saphenous vein graft PCI are shown in Box 8-5.



*One long stent preferred

Fig. 8-13 Scheme for PCI of saphenous vein graft (SVG).

INTERNAL MAMMARY ARTERY PCI

Internal mammary artery PCI presents another difficult technical challenge. Internal mammary artery stenosis commonly is located at the ostial or distal anastomotic site. The distal anastomotic site lesion represents a technical problem, both for intubation and for delivery of stents to the distal location. Selection of vascular access to provide a sufficient length of the angioplasty catheter and successful negotiation of the tortuosity of the artery must be considered. Vasospasm of the internal mammary artery during passage

Box 8-5

Key Points for Saphenous Vein Graft PCI

- Consider all material in graft as embolic debris
- Pretreat with intracoronary verapamil or nitroprusside for possible no-reflow
- Use distal embolic protection device
- Consider AngioJet for high probability thrombus
- Use minimal touch techniques to limit balloon inflations—consider self-expanding stents

of the device can be managed with generous doses of intracoronary nitroglycerin.

Also noteworthy is that guide catheter selection is limited and good seating may be difficult. A left arm approach may be needed for some internal mammary artery stenosis. Careful catheter manipulation should prevent ostial dissection.

MULTIPLE-VESSEL PCI

Multiple-vessel PCI approaches a series of stenoses one at a time, using any and all methods applicable to simple and complex single-vessel PCI. Multivessel PCI addresses at least two of the three major arterial territories (left anterior descending, left circumflex, and right coronary arteries). For example, the dilatation of a mid-left anterior descending and an obtuse marginal is multivessel angioplasty, but dilatation of a mid-left anterior descending and a diagonal branch is considered single-vessel, multilesion angioplasty. Multivessel angioplasty and stenting compete favorably with surgical revascularization in nondiabetic patients if the coronary stenoses morphology and location are likely to yield a high success rate. However, it appears that freedom from cardiovascular events is directly related to completeness of overall revascularization.

Strategies

All Lesions, Single-Setting PCI. For the multivessel patient, the primary lesion to be treated is the one that is responsible for objective myocardial ischemia or is the most critical in supplying a moderate to large area of functioning myocardium. After primary lesion success (<10% narrowing without dissection, thrombus or hemodynamic compromise), the secondary lesions are carefully approached and treated consecutively (Fig. 8-14).

Stage Procedures. In some patients, only the target lesion identified by objective ischemic indicators (ischemia testing or direct translesional hemodynamic assessment) is treated. The remaining secondary stenoses are addressed at a session days or weeks later. In patients with intermediately severe multiple stenoses without objective ischemia, the need for further lesion treatment should be identified by stress testing or direct

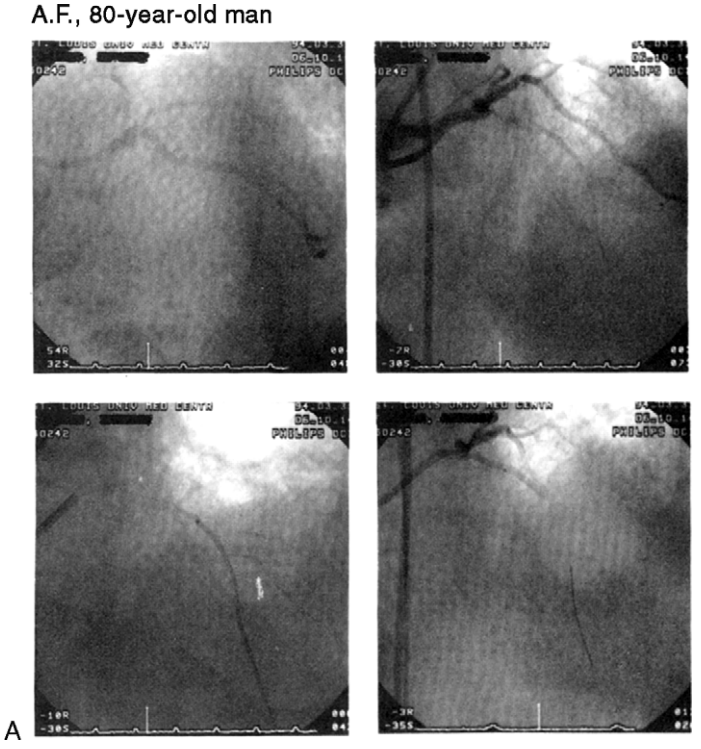


Fig. 8-14 **A,** *Top left,* Left anterior caudal view showing total occlusion of the left anterior descending artery, stenosis of intermediate ramus, and patent circumflex vessel in an 80-year-old man with class IV angina. *Top right,* Right anterior oblique cranial projection with total occlusion of the left anterior descending with faint antegrade left-to-left collateral filling. *Bottom left,* Guidewire and tracking catheter successfully advanced across the total occlusion. Contrast opacification of the distal vessel confirmed intraluminal location of the guidewire. *Bottom right,* Long angioplasty balloon advanced and lesion dilated.

Continued

hemodynamic lesion assessment using fractional flow reserve (FFR) measurement (see Chapter 10).

Indications for Multivessel Angioplasty.

- Symptomatic patients with evidence of ischemia during noninvasive testing
- Patients who are resuscitated from sudden death with significant coronary artery disease

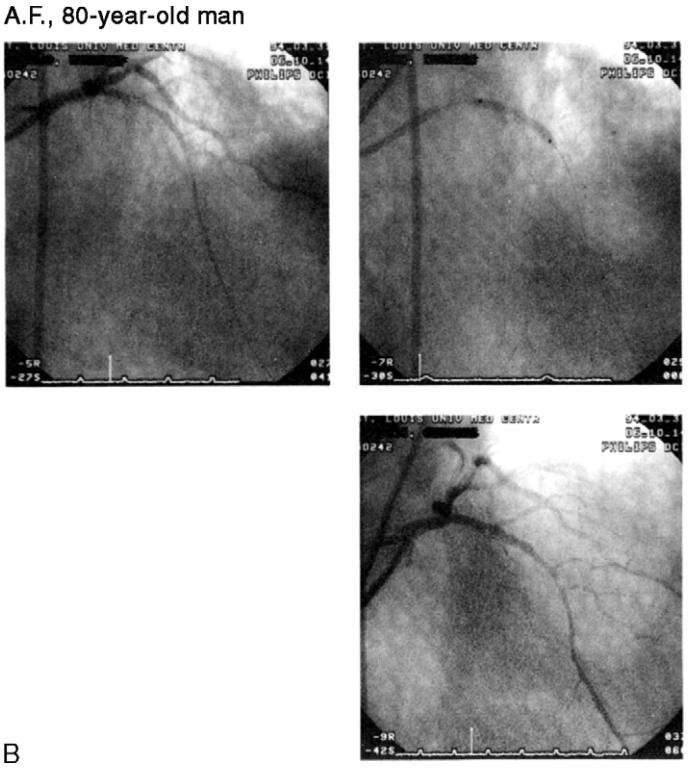


Fig. 8-14, cont'd **B,** *Top left,* A filling defect is prominent at the proximal left anterior descending site. *Top right,* A 3.0 mm × 20 mm stent placed across the proximal left anterior descending. *Bottom right,* Final result showing widely patent, previously totally occluded left anterior descending artery, now with excellent coronary blood flow.

Continued

- Patients who plan to undergo high-risk noncardiac surgery
- Patients with class II–IV angina with symptoms poorly controlled on medical therapy or who are intolerant of medical therapy.

Contraindications. Relative contraindications to multivessel PCI are similar to those for single-vessel PCI for each individual stenosis:

- Left main stenosis or left main equivalent lesions
- Type B2 or C lesions supplying large areas of viable myocardium

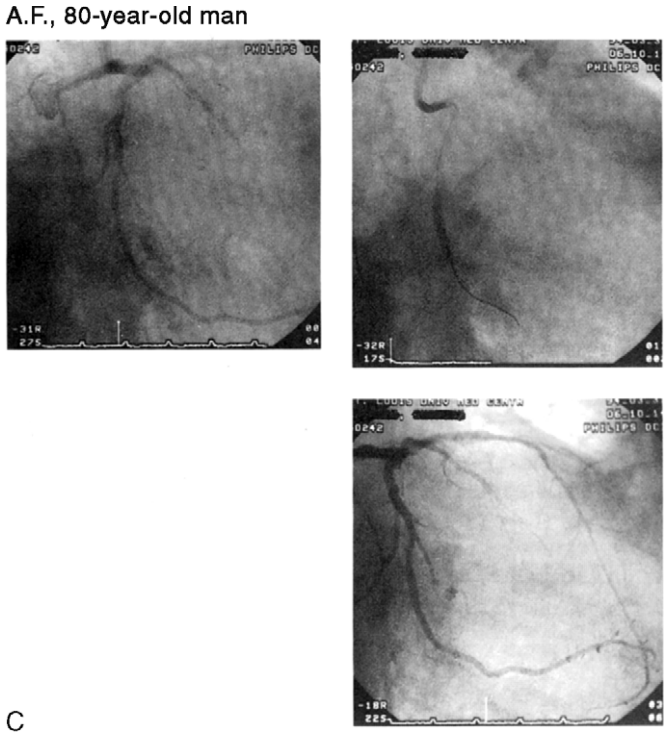


Fig. 8-14, cont'd **C,** Angioplasty of second vessel (circumflex) in this multivessel procedure was performed after recanalization of the left anterior descending coronary artery.

- Multiple severe lesions in major vessels of which one or more is technically unsuitable for revascularization (e.g., total chronic occlusion).

High-Risk Multivessel Angioplasty. Patients at high risk of dying during PCI include those with recent myocardial infarction, recent emergency bypass surgery, or poor left ventricular function. Patients with unprotected left main or left main equivalent disease or single-vessel disease supplying all remaining viable myocardium, high-risk angiographic subsets (degenerated vein graft, intraluminal filling defect, severe angulation, severe calcification), and patients who are very elderly all present higher risk for severe complications and death.

Before considering high-risk angioplasty, discussions should be held with the referring physicians, supporting surgeons, and family members. Should disaster occur, the management approach should already be identified. High-risk angioplasty should weigh the risks of nonoperative revascularization against the benefits. There should be objective evidence of ischemia or myocardial viability before accepting the risk of death versus the benefit of restoring patency to potentially nonfunctional myocardium.

Technical aspects of high-risk multivessel angioplasty include the following.

Identification of the Target Lesion. The primary stenosis should be clearly identified and characterized as causing ischemia.

Vascular Access. Access to the contralateral artery and vein should be maintained with 5 French sheaths, in case hemodynamic support, such as an intra-aortic balloon pump, is needed. Venous access for temporary pacing and/or monitoring of pulmonary artery pressures may also be required. Abdominal angiography should be performed before beginning PCI to identify patients who are not candidates for or may not tolerate prophylactic or emergency intra-aortic balloon placement.

PCI Equipment. All stents, guiding catheters, wires, and sheaths should be selected beforehand and alternative equipment and support supplies (e.g., intravenous dopamine) should be ready for immediate use.

Hemodynamic Support. An intra-aortic balloon pump may be inserted prophylactically or be available on standby for immediate insertion for hypotension or ischemic complications. Active blood perfusion pumps or perfluorocarbon perfusions have not proven more successful than rapid stenting and intra-aortic balloon pumping.

Portable cardiopulmonary bypass (CPS) is limited to specialized experienced laboratories for specific situations with particular grave consequences of coronary occlusion. The use of CPS should be discussed with the patient's family and

surgeon before the procedure. It is especially disturbing to all participants to have a patient with no functioning myocardium yet who is still alive, maintained only by CPS. Discussions about cardiac transplantation are appropriate for this type of patient.

Multivessel PCI in Patients with One Total Occlusion. A relatively common clinical anatomy in multivessel PCI is a totally occluded right coronary artery or left anterior descending artery with a critical narrowing in the contralateral supplying vessel. This anatomy creates a high-risk situation, especially when the totally occluded vessel is not suitable for angioplasty or when it supplies a large area. However, in selected cases the totally occluded vessel may be ignored, with dilatation of the contralateral artery. A typical example might be stenting of a left anterior descending artery in the presence of a totally occluded, small to medium-size right coronary artery supplying an infarcted area and receiving collaterals from both the left anterior descending and circumflex arteries. For PCI multivessel stenoses with one totally occluded vessel needing revascularization, the first lesion approached should be the total occlusion, using standard techniques for TCO and restoring antegrade flow to the region supplied by collaterals. This approach may provide potentially reversed collateral flow to the subsequent target lesions. If total occlusion dilatation is successful, the operator proceeds to the secondary lesions. If angioplasty of the total occlusion is unsuccessful, the patient can be referred to surgery for complete revascularization.

Clinical Results of Multivessel PCI. Procedural success varies widely (80–95%) among trials. The Multivessel Angioplasty Prognosis Group recommended bypass surgery in patients with two-vessel disease who had type B2 or C lesions in large-caliber vessel or when two adverse prognostic factors were present, especially when the risk territory score was more than 15 or left ventricular dysfunction was present.

During follow-up, patients with incomplete revascularization experience more frequent recurrent angina and require coronary artery bypass graft surgery more often than those with complete revascularization. Some of the differences between these groups may also be related to the higher incidence of

unfavorable baseline clinical characteristics of the incomplete revascularization group. Nonetheless, patients who have incomplete revascularization may experience highly satisfactory clinical improvement despite evidence of some myocardial ischemia on future clinical evaluations. Bypass surgery is a preferred alternative for patients with complex, low-success lesion morphology who are likely to have incomplete revascularization.

Restenosis for multivessel disease averages approximately 40%. Restenosis rates have decreased since the introduction of routine coronary stenting and will probably decrease further with drug-eluting stents.

Key points in multivessel stenting are shown in Box 8-6.

PCI FOR UNSTABLE ANGINA

Percutaneous coronary intervention is a highly effective technique for unstable angina. Angioplasty mortality in this patient subset is low (0.5–1%). A common strategy for patients with unstable angina begins with aggressive medical therapy (aspirin, glycoprotein blockers, heparin, beta blockers, and nitrates). If the patient stabilizes within 1–2 hours, urgent angioplasty may be deferred. In patients with recurrent symptoms during the first 6–12 hours of medical management, urgent coronary angiography is performed. The extent and characteristics of coronary artery disease will determine whether coronary bypass surgery or PCI is needed.

Because unstable angina is associated with thrombus and acute plaque transformation, PCI should be preceded by antiplatelet (aspirin, clopidogrel, glycoprotein blockers) and

Box 8-6

Key Points in Multivessel Stenting

- Plan a strategy and stick to it
- Do all lesions needing stent and skip those that do not. Measurement of fractional flow reserve (FFR) can help greatly here
- Direct stenting will shorten overall procedure time
- Do most important lesions first
- Stage procedure for additional lesions if the patient or operator becomes fatigued or unanticipated difficulties arise—e.g., excess contrast, arrhythmias, or patient discomfort

antithrombin (unfractionated or low-molecular-weight heparin) therapies. Pretreatment with clopidogrel is beneficial even if the patient does not require a stent. Hemodynamic support with intravenous fluids, pulmonary artery pressure monitoring, and intra-aortic balloon pumping may be needed in some patients.

PCI FOR ACUTE MYOCARDIAL INFARCTION

Direct or Primary Angioplasty

Direct angioplasty (angioplasty undertaken without prior thrombolytic therapy) is indicated in patients with acute myocardial infarction who can be recanalized in less than 120 minutes after onset of symptoms. PCI is also indicated for patients in whom thrombolytics are contraindicated, and patients in cardiogenic shock. Patients having an acute myocardial infarction who have immediate access to a cardiac catheterization without an onsite surgical backup facility but with experienced PCI operators and a plan for acute myocardial infarction treatment can also have PCI. The advantages of direct angioplasty include early and complete reperfusion, identification of associated coronary artery disease and reduced bleeding complications relative to thrombolytic therapy. The disadvantage of direct angioplasty is the delay in treatment caused by on-call services.

Post-Thrombolysis PCI

In asymptomatic patients, a strategy of routine PCI of the stenotic infarct related artery immediately after successful thrombolysis shows no benefit with regard to salvage of jeopardized myocardium or prevention of reinfarction or death. In some studies, this approach was associated with increased incidents of adverse events, which include bleeding, recurrent ischemia, emergency coronary artery surgery, and death. Routine PCI immediately after thrombolysis may increase the chance of vascular complications at the catheterization access site and hemorrhage into the infarct related vessel wall.

Rescue or Salvage Angioplasty

Rescue PCI is defined as PCI after failed thrombolysis for patients with continuing or recurrent myocardial ischemia. Rescue PCI has resulted in higher rates of early infarct artery

patency, improved regional infarct zone, wall motion, and greater freedom from adverse in-hospital and clinical events compared to deferred strategy. The randomized evaluation of the rescue PCI with combined utilization endpoint trial (RESCUE) demonstrated a reduction in rates of in-hospital death and combined death and congestive heart failure maintained up to 1 year after study for patients presenting with anterior wall myocardial infarction who failed thrombolytic therapy. Improvement in the thrombolysis in myocardial infarction study (TIMI) grade flow from 2 to 3 may offer additional clinical benefit. Rescue or salvage angioplasty is used in patients with acute myocardial infarction after failed thrombolysis. Rescue coronary angioplasty for anterior myocardial infarction reduces the risk of death and congestive heart failure and improves exercise ejection fraction. Re-establishment of coronary perfusion and myocardial salvage is especially important in high-risk patients.

The rescue angioplasty approach is supported by non-randomized observational trials in which increased survival rates were observed in patients less than 75 years old in cardiogenic shock or after failed thrombolysis with continued evidence of myocardial infarction.

Intra-aortic balloon counterpulsation is a commonly applied adjunctive modality for rescue PCI in patients with hypotension.

Immediate Angioplasty

Immediate angioplasty (angioplasty performed less than 24 h after thrombolysis) following successful thrombolysis is indicated if continued myocardial ischemia is present. Results of randomized trials do not demonstrate benefit of routine post thrombolysis PCI in stable patients over elective angioplasty, based on objective evidence of ischemia in the immediate post infarction period. However, despite a paucity of supporting data, it is common practice in some centers to perform routine coronary angiography after all myocardial infarctions, especially anterior myocardial infarction, or in young patients.

Elective (Deferred) Angioplasty

Elective or deferred angioplasty (angioplasty performed several days after thrombolysis when there is evidence of myocardial

ischemia) is an acceptable approach after acute myocardial infarction in patients without complications or continued myocardial ischemia after they have received thrombolytic therapy. In asymptomatic patients, ischemic risk stratification is performed before coronary angiography and revascularization. Catheterization and angioplasty are undertaken if there is evidence of increased ischemic risk.

Coronary Bypass Surgery for Patients with Acute Myocardial Infarction

In patients who have left main stenosis, left main equivalent stenoses, severe multivessel disease not suitable for angioplasty, or severe multivessel disease with cardiogenic shock, coronary artery bypass grafting is preferred to PCI.

Technical Considerations for PCI in Acute Myocardial Infarction

Anticoagulation. Because acute myocardial infarction always involves thromboses, pretreatment with oral aspirin, clopidogrel, glycoprotein receptor IIb/IIIa inhibitors, and intravenous heparin (activated clotting time >250 sec) are often required.

Angioplasty Equipment and Vascular Access. Before the coronary angiography, the operators should anticipate proceeding to coronary intervention and thus initiate the diagnostic catheterization with a 6 French arterial sheath for arterial access. The operator may anticipate that a pulmonary artery balloon-tipped catheter for hemodynamic monitoring might be needed at the end of the procedure. Early venous access should be obtained. Do not forget a sterile catheter sleeve for the pulmonary artery catheter before insertion. A 5 French arterial sheath in the contralateral femoral artery for intra-aortic balloon pumping may be useful.

Hemodynamic Support and Intra-Aortic Balloon Pumping. In patients with hypotension, mitral regurgitation, multivessel disease, or decreased left ventricular function, an intra-aortic balloon pump should be inserted before the procedure begins. After successful angioplasty for acute infarction PCI, intra-aortic balloon counterpulsation is associated with reduced recurrent ischemia. In some patients who are stable during the

intervention, the intra-aortic balloon pump can be inserted at the end of the PCI through the femoral artery access.

Figure 8-15 illustrates angioplasty for acute myocardial infarction. Key points for PCI in acute myocardial infarction are shown in Box 8-7.

PCI FOR CARDIOGENIC SHOCK

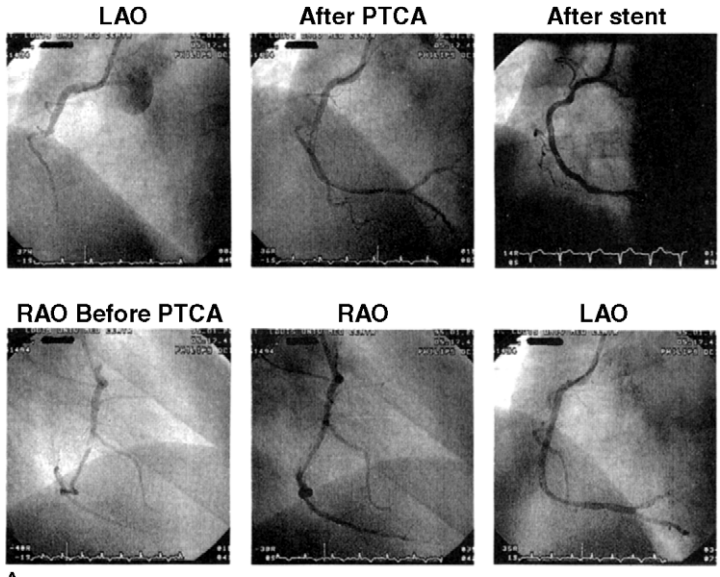
Untreated cardiogenic shock is associated with mortality rates of more than 90%. The prognosis and management of patients in cardiogenic shock due to myocardial infarction are determined by the duration of myocardial ischemia, the cardiac index, left ventricular end-diastolic pressure, and the presence of pulmonary edema.

The SHOCK trial was a multicenter trial that randomized patients with myocardial infarction and shock due to pump failure to either early revascularization or medical therapy (86% of all patients received an intra-aortic balloon pump). The 30-day mortality was not significantly different overall; however, at 1 year those patients who received early revascularization had slightly improved survival. In elderly patients (age >75 years), early revascularization was associated with worse survival rates than medical treatment, although only 56 patients over the age of 75 years were randomized during the 4-year trial. Furthermore, the nonrandomized elderly patients in the SHOCK registry appeared to derive a survival benefit from early revascularization. This trial, along with large registries, confirms the benefit of an early revascularization strategy in patients with shock complicating a myocardial infarction.

Key points for PCI in cardiogenic shock are shown in Box 8-8.

ANGIOPLASTY IN THE ELDERLY

Although PCI techniques are being used increasingly in patients more than 80 years old, the risk of PCI is increased in elderly patients. Strategies for complications should be carefully discussed with patient, family, and surgeon because some emergency procedures may not be feasible (e.g., intra-aortic balloon pumping) or may carry extraordinary risk (e.g., left main stenting). Myocardial perfusion and hemodynamics should be optimized before beginning the procedure. Adequate hydration and limiting the amount of



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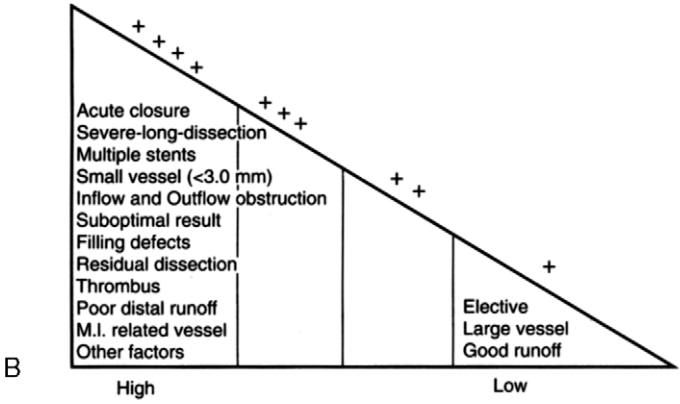

B

Fig. 8-15 **A,** Case example of stent thrombosis. Angiograms show proximal total right coronary artery occlusion. Recanalization by percutaneous transluminal coronary angioplasty (PTCA) is performed with evidence of dissection (top middle). A stent is placed and a lucent filling defect is seen at the distal end of the stent (top right). Further balloon inflations reduced the presumed thrombus. LAO, Left anterior oblique view; RAO, right anterior oblique view. **B,** Diagram of factors contributing to risk of stent thrombosis. Factors associated with highest risk are shown at left. (**B,** Courtesy of Gary Roubin, MD, University of Alabama, Birmingham, AL.)

Box 8-7

Key Points for Acute Myocardial Infarction PCI

- Prepare for complications of heart block, hypotension and arrhythmia
- Consider AngioJet for large amount of thrombus
- Balloon-dilate and then observe for no-reflow
- Stent, observe, and treat no-reflow.
- Insert pulmonary artery catheter, intra-aortic balloon pump or temporary pacemaker to assist future management in the coronary care unit

Note. Controversy exists regarding multivessel PCI in patients with acute myocardial infarction. Since the use of stents, some operators believe that additional vessels with critical stenosis can be treated in a single session after stenting of the infarct-related artery.

contrast medium are important. Anticoagulation regimens should be individualized. After the procedure, extra precautions need to be taken during sheath removal and hemostasis.

PCI IN CARDIAC TRANSPLANT RECIPIENTS

Coronary disease is a significant cause of morbidity and mortality in cardiac transplant recipients. A diffuse vasculopathy is usually present, characterized by concentric intimal thickening and coronary artery dilation (positive remodeling). Coronary angioplasty has been used in this group of patients to treat epicardial artery obstruction. The acute results appear similar to those in nontransplant patients. Long-term changes in morbidity and mortality do not appear to be different for

Box 8-8

Key Points for Cardiogenic Shock PCI

- Establish systolic pressure greater than 80 mm Hg. Use dopamine, norepinephrine, intra-aortic balloon pump, or intubation as required
- Use same techniques and approaches as for PCI in acute myocardial infarction
- Anticipate ventricular tachycardia/ventricular fibrillation treatments. Use amiodarone, cardioversion, intubation, and cardiopulmonary resuscitation as needed
- Dilate and stent essential lesion(s)
- Limit duration of procedure in lab. Stabilize the patient and manage clinically in coronary care unit

Note. The dilemma of multivessel PCI in cardiogenic shock is unresolved. Employ operator's best judgment for additional lesion interventions.

PCI with and without stenting versus medically managed coronary artery stenoses in transplant patients.

TREATMENT OF THROMBOSIS DURING PCI

At one time, intracoronary thrombus was common, occurring in up to 15% of all patients undergoing coronary balloon angioplasty and up to 40% in acute coronary syndromes. In the current era, better therapies for thrombus (clopidogrel, glycoprotein blockers, early heparin) have reduced the incidence of intracoronary thrombus. However, these lesions do present a challenge for PCI. Mechanical approaches to thrombus lesions carry an increased risk of abrupt occlusion, embolization, acute myocardial infarction, emergency bypass surgery, and death compared to nonthrombotic lesions. An optimal mode of PCI for thrombotic lesions has not yet been defined. Many interventionalists advocate mechanically decreasing thrombus bulk before proceeding. The AngioJet® (Possis Medical) rheolytic thrombectomy device is currently the only approved thrombus debulking device for intracoronary use. Intracoronary thrombolytic agents are no longer employed before coronary angioplasty.

Adjunctive Agents for PCI of Thrombotic Lesions

Heparin. All patients with IC thrombus should be treated with intravenous heparin to reduce clot propagation. Heparin does not reduce the thrombus but it permits the body's intrinsic thrombolytic mechanisms to stabilize and reduce thrombus mass. For patients who cannot receive heparin because of heparin allergy or heparin-induced thrombocytopenia, direct antithrombins such as bivalirudin can be given.

Antiplatelet Agents (ASA, Clopidogrel, Glycoprotein IIb/IIIa Blockers). To reduce platelet aggregation, adhesion and activation antiplatelet agents should be given to all patients. These agents are associated with a reduced incidence of complications.

Contraindications to Anticoagulation.

- Active bleeding
- Recent surgery or major trauma (within 2 months)
- Recent stroke (<6 months)

- Central nervous system malignancy
- Bleeding diathesis (check prothrombin time, partial thromboplastin time, platelets, hematocrit)
- Severe uncontrolled hypertension (diastolic blood pressure >120 mm Hg, systolic blood pressure >200 mm Hg)
- Pregnancy.

Difficult-lesion PCI is encountered on a daily basis in most laboratories. New operators will benefit by careful planning and a logical approach to lesions. Consulting with more experienced operators is also helpful and would be a method through which more difficult lesions can be taken on. Finally, recognizing the limits of the patient, procedure, and operator is critical key in performing safe PCI in difficult situations.

Suggested Readings

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9

HIGH-RISK PERCUTANEOUS CORONARY INTERVENTIONS

Oscar M. Aguilar and Glenn N. Levine

The risk of major complications such as myocardial infarction, life-threatening arrhythmias, need for emergency coronary artery bypass surgery (CABG), and death during a percutaneous coronary intervention is influenced by angiographic, patient-related, and clinical factors. These factors can be utilized to identify the patient at high risk of complications, to evaluate and discuss the risk:benefit ratio of proceeding with the intervention with both patient, family, and hospital personnel (e.g., cardiac surgeons), to allow for appropriate measures that can be taken during the high-risk percutaneous coronary intervention (PCI) procedure to minimize the risk of a major adverse event, and to allow the catheterization laboratory and hospital personnel to be optimally prepared to deal with complications if they occur.

IDENTIFYING THE HIGH-RISK PCI PATIENT

The first important step when performing high-risk PCI is identifying which patients are indeed high-risk. Retrospective studies and databases have been utilized to identify risk factors for adverse events occurring during PCI.

Angiographic Factors

The American College of Cardiology/American Heart Association (ACC/AHA) created a scoring system that was utilized to classified lesions according to their complexity, likelihood of successful dilation, and the likelihood of adverse event. This system classified lesions as either type A, B, or C,

with C the most high-risk lesions. The system was based on lesion characteristics, including lesion location, the presence of thrombus, length of the diseased segment, lesion angulation, the presence of calcification, and the tortuosity of the vessel (Box 9-1).

Box 9-1

The ACC/AHA Lesion Classification Scheme

This scheme has been slightly modified in that lesions with one "type B" characteristic are designated as "type B1" while lesions with two or more "type B" characteristics are designated as "type B2" lesions.

Type A Lesions

- Discrete
- Concentric
- Ready accessibility
- Location in a nonangulated segment (<45°)
- Smooth contour
- Little or no calcification
- Absence of total occlusion
- Nonostial location
- Absence of major branch involvement
- Absence of thrombus

Type B Lesions

- Tubular (10-20 mm in length)
- Eccentric
- Accessibility influenced by moderate tortuosity of proximal segment
- Location in moderately angulated segment (45–90°)
- Irregular contour
- Moderate or severe calcification
- Presence of thrombus
- Ostial location
- Bifurcation lesion requiring double wiring
- Total occlusion <3 months old

Type C Lesions

- Diffuse disease (>20 mm in length)
- Excessive tortuosity of proximal segments
- Location in an extremely angulated segment (>90°)
- Total occlusion >3 months old
- Inability to protect major side branches
- Degeneration of older vein grafts with friable lesions

The system was later slightly modified to subdivide type B lesions into type B1 lesions (those that had only one moderate "type B" risk characteristic) and type B2 lesions (those that had two or more moderate "type B" risk characteristics). In the pre-stent era, lesions in the B1, B2 and C category were found to have a lower probability of success and a higher risk of complications. Since this classification, which was first implemented in 1988, significant changes have been made to the approach to PCI, which have allowed treatment of more complex lesions with lower risks. In the current era of improved equipment and coronary stenting, it is primarily the type C lesions that are associated with lower success and higher complication rates.

In the mid 1990s, an era in which coronary stents and platelet glycoprotein IIb/IIIa inhibitors were frequently utilized, Ellis and coworkers analyzed a large database of patients undergoing PCI. Nine angiographic factors were identified that correlated with greater risk of complication. The two factors associated with the greatest increased risk were degenerated saphenous vein grafts (relative risk 4.18) and nonchronic total occlusion (relative risk 4.74). Other factors included long lesions, lesions with large filling defects, calcified angulated lesions, eccentric lesions, and old saphenous vein grafts (Table 9-1). The finding of marked increased risk in degenerated vein grafts supports the practice of using distal protection devices during PCI of such lesions.

Patient-Related and Clinical Factors

Several clinical factors can be utilized to identify high-risk PCI. Such factors include the presence of multivessel disease, angioplasty to more than one lesion, suboptimal activated clotting time (ACT), residual stenosis above 30%, depressed ejection fraction, old age (>65 years), unstable angina and recent myocardial infarction (Box 9-2). A retrospective study from the Mayo Clinic, examining the risk of PCI performed in the era of glycoprotein IIb/IIIa inhibitors and coronary stent implantation, found that clinical factors such as left main or multivessel disease, an ejection fraction below 35%, or a recent myocardial infarction, were more important than angiographic factors for predicting complications. Other studies have identified the presence of diabetes mellitus and renal disease as

Table 9-1

Lesion Characteristics and the Increased Risk of Ischemic Complications (Based on Multivariate Analysis)

Lesion Characteristic	Odds Ratio
Nonchronic total occlusion	4.74 (2.69–8.38)
Degenerated saphenous vein graft	4.18 (2.39–7.31)
Length ≥20 mm	2.77 (1.51–5.09)
Irregularity	1.88 (1.32–2.66)
Large filling defect	1.41 (1.17–1.70)
Length 10–20 mm	1.88 (1.26–2.82)
Moderate calcification with angulation >45°	4.44 (1.24–15.96)
Eccentric	2.12 (1.04–4.57)
Severe calcification	2.19 (1.04–4.57)
Saphenous vein graft age ≥10 years	1.81 (1.00–3.31)

Adapted from Ellis SG *et al.* Relation between lesion characteristics and risk with percutaneous intervention in the stent and glycoprotein IIb/IIIa era. *Circulation* 1999;100:1971–1976.

indicators of higher risk for complications and in-hospital mortality.

Operator Experience. Another factor that has been correlated with PCI risk is operator volume and experience. Several studies have found that those operators with greater experience have lower complication rates; the correlate being that those with lower procedural volume and experience have higher complication rates. This factor should also be considered for decisions regarding the performance of high-risk PCI.

Box 9-2

Patient and Clinical Factors Associated with Higher-Risk PCI

- Presence of left main coronary artery or multivessel disease
- PCI of more than one lesion
- Suboptimal activated clotting time
- Residual stenosis above 30%
- Depressed ejection fraction (<35%)
- Age over 65 years
- Unstable angina and/or recent myocardial infarction
- Severe concomitant disease

Myocardium at Risk. In patients with depressed ejection fraction, it is particularly important to assess which myocardial territories (anterior, lateral, inferior) are akinetic and/or consist of infarcted myocardium, and which myocardial territories are doing the majority of the "work" of left ventricular functioning. In a patient with infarcted lateral and inferior walls in which the only functioning territory is the anterior wall and septum, PCI of the left anterior descending coronary artery obviously carries extreme risk, in that, if there is compromised blood flow during the procedure, the patient can be expected to do extremely poorly. To some extent, a situation such as this should be considered as a "left main equivalent," and decisions regarding PCI and pharmacological and/or mechanical support made accordingly.

Vascular Disease. An often underappreciated factor in the high-risk PCI patient is vascular access and vascular disease. During the high-risk PCI procedure, an intra-aortic balloon pump (IABP) may be placed either prophylactically or emergently, requiring knowledge of the presence of iliac/femoral arterial disease beforehand. Palpitating strong bilateral femoral pulses should be coupled with angiography. When possible, in patients identified as high-risk PCI during their diagnostic procedure, angiography of the iliac and common femoral arteries should be performed to determine if the patient is a candidate for IABP support.

Specific High-Risk Subsets

Left Main Coronary Artery PCI. Several registries and retrospective studies have examined procedural success and short- and intermediate-term complication rates in patients undergoing PCI of unprotected left main lesions. Some patients underwent PCI because they were poor candidates for CABG, some because of strong patient preference, and a few because of acute myocardial infarction.

Balloon Angioplasty. The results of left main balloon angioplasty alone have been poor, with in-hospital mortality rates of up to 9.1% and a 3-year survival rate of 36%. Factors associated with high complication and mortality rates in percutaneous transluminal coronary angioplasty studies

include distal left main lesion with bifurcation involvement and low ejection fraction.

Han and colleagues reported the results of a multicenter registry of 279 patients. Approximately 50% of all patients were supported with intra-aortic balloon counterpulsation, 6% underwent prophylactic percutaneous cardiopulmonary support, and 66% received stents. Although the technical success rates of the procedure were high, the in-hospital mortality rate was 14%, and 1-year all-cause mortality was 24%. Factors associated with greater procedural risk and worse 1-year outcome included acute myocardial infarction with cardiogenic shock, ejection fraction below 30%, significant or severe mitral regurgitation, creatinine above 2 mg/dl, and severe calcification. A low-risk subgroup of patients was identified that consisted of those less than 65 years of age, with an ejection fraction above 30% and without cardiogenic shock. The 1-year mortality in this group was 3.4%.

Stenting. Reports of coronary stenting provide relatively encouraging data, including some with no in-hospital or late death attributable to the PCI procedure. However, these reports are generally retrospective, are likely to involve extremely carefully selected patients, and are generally from institutions with a high degree of experience and expertise. Thus, despite enthusiasm and encouraging results from small patient series, until randomized data are available comparing unprotected left main PCI to CABG, an unprotected left main stenosis should generally be considered as a contraindication to PCI and should be preferentially treated with CABG. In patients who are not candidates for or who adamantly refuse CABG and lack the high-risk features discussed above, PCI of the left main appears to be a viable option but should still be considered a very high-risk PCI, undertaken with all the precautions discussed above. Any decision regarding unprotected left main PCI should be made in consultation with the cardiothoracic surgery service. In those who do undergo unprotected left main PCI, Ellis and colleagues have strongly suggested that these patients undergo routine surveillance angiography during the restenosis window, in light of the high rate of

mortality observed in their registry in the first 6 months post-procedure.

Cardiogenic Shock. The highest clinical factor associated with risk of PCI is the presence of cardiogenic shock. Despite recent advances in pharmacotherapy, the incidence of cardiogenic shock as a complication of acute coronary syndromes has not diminished over time, nor have mortality rates. Cardiogenic shock occurs in approximately 2.1–2.9% of patients presenting with non-ST elevation acute coronary syndrome ("unstable angina" or "non-Q wave" myocardial infarction) and approximately 5.4–7.6% of patients presenting with ST-segment elevation acute myocardial infarction. Mortality for this condition is on the order of 60%. At least 14 single-institution retrospective studies have suggested that, in patients with cardiogenic shock who underwent PCI, mortality was reduced. The average successful reperfusion rate in these studies was 73%. Mortality in such patients was 44%. Mortality was 30% if reperfusion was successful but 80% if reperfusion attempts were unsuccessful. The overall lower mortality rates with PCI reported in these studies were, however, potentially subject to extreme selection bias, in that cardiogenic shock patients with fewer comorbid conditions and those believed more likely to survive may have been preferentially selected to undergo cardiac catheterization and PCI.

The SHOCK trial provided randomized data of the role of PCI in patients with cardiogenic shock. In this multicenter trial, urgent revascularization was compared to initial medical stabilization in 302 patients with cardiogenic shock from acute myocardial infarction. In patients who underwent urgent revascularization, 64% had angioplasty and 36% were revascularized via CABG. At 30 days, the mortality was 46.7% and 56.0% for the revascularization and the medical therapy groups, respectively (p = NS). At 6 months, however, the mortality was significantly lower in the revascularized group (50.3% vs 63.1%), and this difference remained significant at 1-year follow-up. Of note, in the prespecified group of patients older than 75 years, there was no benefit from revascularization. Based on the SHOCK trial and prior reports, acute angioplasty for elderly cardiogenic shock patients may not be generally beneficial.

RISK REDUCTION AND SUPPORT OF THE HIGH-RISK PCI PATIENT

Pharmacotherapy

Pharmacotherapy must be considered both prophylactically and during complications in high-risk PCI patients. Concomitant with this, it is essential to have good intravenous access allowing rapid infusion of medications. At least one and ideally two large-bore well-working peripheral intravenous lines should be present in high-risk patients. Although discouraged in most routine PCI procedures (due to an increased risk of bleeding complications), venous femoral sheaths should be strongly considered in a patient with a high possibility of needing aggressive anti-ischemic, pressor, and/or inotropic support, or transvenous pacing.

Anti-Ischemic Agents.

Nitroglycerin. Although nitroglycerin (intravenous or intracoronary) is commonly utilized before and after balloon dilations, it has not been demonstrated to provide prolonged ischemic benefit. Nitroglycerin may, however, reduce coronary spasm in selected situations. When using nitroglycerin, it is important to maintain an adequate margin of blood pressure (mean arterial pressure > 70 mm Hg) so that transiently induced severe ischemia does not reduce blood pressure below a critical perfusion level (mean arterial pressure <60 mm Hg), leading to a downward ischemia spiral. High-risk PCI patients should be well hydrated before the procedure so that nitroglycerin does not cause an excessive decrease in blood pressure.

Beta Blockers and Calcium Channel Blockers. Although beta blockers and calcium channel blockers may reduce local myocardial ischemia during balloon inflation through a regional decrement in myocardial oxygen consumption, this benefit has little clinical impact, particularly in the era of brief balloon inflations and coronary stenting. Pretreatment with these agents may become counterproductive in fact if the patient "crashes" during the high-risk PCI procedure, as these agents act as negative inotropes and/or vasodilators.

In high-risk PCI patients on these agents, operators should have access within the catheterization laboratory to drugs that

at least partially reverse the actions of beta blockers and calcium channel blockers. Calcium chloride (1 ampoule; 13.6 mEq) may reduce some of the vasodilatory effects of calcium channel blockers, and perhaps to a lesser degree the negative inotropic and negative chronotropic/conduction effects. It has been suggested that glucagon (1 mg) may partially reverse the actions of beta blockers.

Antiplatelet Agents (Oral).

Aspirin markedly decreases the incidence of abrupt vessel closure and is mandatory in all patients undergoing routine as well as high-risk PCI. In patients who have true aspirin allergy, pretreat with clopidogrel, either started several days before the procedure (75 mg daily) or given as a loading dose (300 mg) the night before (if possible) or early on the morning of the procedure (at least 6 hours pre-procedure). Patients pretreated with clopidogrel hours to days before their procedure (in those who are also being treated with aspirin) have a lower incidence of complications than those not receiving clopidogrel pre-procedure. Although this would seem to suggest that all high-risk PCI patients should receive clopidogrel pre-procedure, the nature and design of these studies does not permit definite conclusions. Clopidogrel significantly increases the risk of bleeding complications during surgery and, as an irreversible platelet inhibitor, exerts its effects for days. Therefore, pending further data, in the high-risk patient where there is a reasonable chance that the patient may require emergency CABG, it may be prudent to defer clopidogrel pretreatment until PCI is successfully completed.

To decrease the incidence of subacute stent thrombosis, patients who do undergo successful PCI with coronary stent placement should be treated with clopidogrel (300 mg loading dose given immediately post-procedure if patient has not yet been treated with clopidogrel; then 75 mg daily for 4 weeks) in addition to aspirin therapy.

Antiplatelet Agents (Intravenous). The platelet glycoprotein IIb/IIIa inhibitors block platelet aggregation and adhesion. Intravenous administration of these agents peri-procedure usually leads to between 80–95% inhibition of platelet aggregation. These agents reduce ischemic complications in

patients undergoing PCI. The greatest absolute magnitude of risk reduction is in those with high-risk angiographic and/or clinical features. Therefore, these agents should be strongly considered during high-risk PCI. The characteristics of the three platelet IIb/IIIa inhibitors, and a suggested dosing regimen, for the three platelet IIb/IIIa inhibitors are given in Table 9-2.

Antithrombin Therapy. There is little data specific to anticoagulant selection or dosing specific to high-risk PCI patients.

Unfractionated Heparin. Unfractionated heparin is the antithrombin most commonly used during PCI. Although the optimal ACT with heparin may be 350 sec, this result was obtained from older studies in patients generally not treated with IIb/IIIa inhibitors or coronary stents. In current practice, in patients treated with IIb/IIIa inhibitors, the ACT should be maintained at 200–300 sec.

Low-Molecular-Weight Heparin. Although some patients undergo PCI on enoxaparin therapy, no data is available on enoxaparin in the high-risk PCI patient.

Table 9-2

Platelet Glycoprotein IIb/IIIa Inhibitor Characteristics and a Suggested Dosing Regimen

	Abciximab	Eptifibatide	Tirofiban
Molecule	Antibody fragment	Hexapeptide	Tyrosine derivative
Thrombocytopenia	None or extremely rare	None or extremely rare	1–4%
Effective half-life	≈12–24 hours	≈24 hours	≈2–4 hours
Possible dosing regimen	0.25 mg/kg IV bolus; then 0.125 μg/kg/min (max 10 μg/min) infusion	180 μg/kg bolus; then 2.0 mg/kg/min infusion; 10 min after first bolus give second bolus of 180 μg/kg	10 μg/kg bolus; then 0.15 μg/kg/min infusion
Dosing adjustment necessary with renal insufficiency	Use with caution	Yes	Yes

Bivalirudin. The direct thrombin inhibitor bivalirudin (Angiomax) has been compared to unfractionated heparin in three small or antiquated studies, and to unfractionated heparin (plus IIb/IIIa inhibitor) in the larger REPLACE-2 study. In the three smaller/antiquated studies, bleeding complication rates were consistently significantly lower with bivalirudin than with unfractionated heparin. This decreased bleeding rate may be related to its mechanism of action and/or very short half-life (approximately 20 min). In REPLACE-2, bivalirudin and provisional IIb/IIIa therapy was associated with a lower risk of bleeding than unfractionated heparin and routine IIb/IIIa use, but was associated with a trend toward slightly higher rates of myocardial infarction. It should also be noted that REPLACE-2 is not generally considered a "high-risk" PCI trial. Therefore, it seems prudent to consider bivalirudin in patients at very high risk for bleeding complications, but to preferentially utilize unfractionated heparin and IIb/IIIa therapy for patients at very high risk of ischemic complications.

Vasopressors and Inotropic Agents. For high-risk PCI, vasopressors and inotropic agents should be readily available. In very high-risk PCI procedures, have at least one vasopressor agent premixed and available for immediate infusion. Commonly used vasopressors include dopamine and norepinephrine.

Dopamine produces primarily renal and splanchnic vasodilation at low doses, exerts a positive inotropic and chronotropic effect at moderate doses, and exerts a vasoconstrictive effect at higher doses. A starting dose in symptomatic hypotensive patients is 5 μg/kg/min. A higher starting dose (10 μg/kg/min) can be considered in the severely hemodynamically compromised patient.

Norepinephrine can be given either via intermittent intravenous boluses or continuous infusion. In our laboratory, for high-risk PCI, we always have premixed intermittent boluses of norepinephrine (2.5–5 μg) available. This produces rapid improvement in blood pressure, is easily titratable, and has a relatively short half-life. The premixing regimen we use is given in Box 9-3.

Box 9-3

Premixing Regimen to Prepare Norepinephrine for Bolus Therapy for Blood Pressure Support

1. Dilute 10 mg norepinephrine in 90 ml normal saline
   =10,000 μg/100 ml
   =100 μg/ml
2. Take 1 ml of above mixture and further dilute in another 9 ml normal saline
   =100 μg/10 ml
   =10 μg/ml
3. Administer bolus doses of 2.5–5.0 ml approximately every 5 min as needed. Blood pressure usually responds within 3–5 min.

Dobutamine. If necessary, dobutamine may be considered for inotropic support. However, because as a beta receptor agonist it can also lead to peripheral dilation, it is not ideal in a hypotensive patient. Usual doses of dobutamine are 2.5–10 μg/kg/min.

Transvenous Pacing

Prophylactic pacemaker insertion is determined by (1) the patient's risk of developing a bradyarrhythmia and (2) the patient's ability to tolerate the arrhythmia if it occurs. Conditions in which a prophylactic pacemaker may be considered include:

- Severe fascicular block
- Heart block greater than first degree
- Marked sinus bradycardia
- PCI involving the (dominant) artery supplying the AV node (particularly if the right coronary artery is occluded during a left circumflex artery PCI and vice versa).

Short of actually placing a temporary pacemaker, in the high-risk PCI patient obtain femoral venous access and place a 5–7 French femoral venous sheath to allow rapid placement of a pacemaker, as well as serving as a conduit for rapid infusion of pressors and/or inotropes if necessary.

Intra-Aortic Balloon Pump

Mechanisms. Intra-aortic balloon pump counterpulsation increases myocardial oxygen supply and decreases myocardial

oxygen demand. Balloon inflation at the onset of diastole (at the dicrotic notch on the central arterial pressure tracing) results in augmentation of diastolic pressure, which increases coronary artery (and systemic) perfusion. Deflation of the balloon just before systole (end diastole on the arterial pressure tracing) results in decreased ventricular afterload, which decreases myocardial oxygen consumption and increases cardiac output. In patients with low-output states, cardiac output may be increased by 20–30%. These effects are illustrated in Fig. 9-1. An example of the arterial waveform during correctly timed intra-aortic balloon counterpulsation is shown in Fig. 9-2.

There appears to be a 20–30% increase in cardiac output in patients with low-output syndromes and a significant amount of afterload reduction as demonstrated in reduction of mitral regurgitation. Direct measurement of coronary blood flow during IABP demonstrated augmentation in nondiseased and

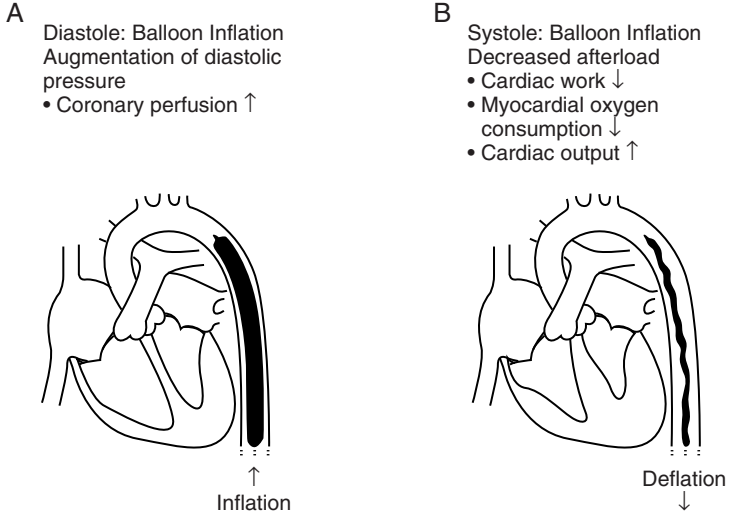

Fig. 9-1 A schematic representation of balloon inflation (**A**) during diastole and (**B**) just before the onset of systole. Diastolic augmentation increases coronary artery perfusion while deflation of the balloon just before the onset of systole decreases afterload, which results in decreased myocardial oxygen demand, decreased cardiac workload, and increased cardiac output.

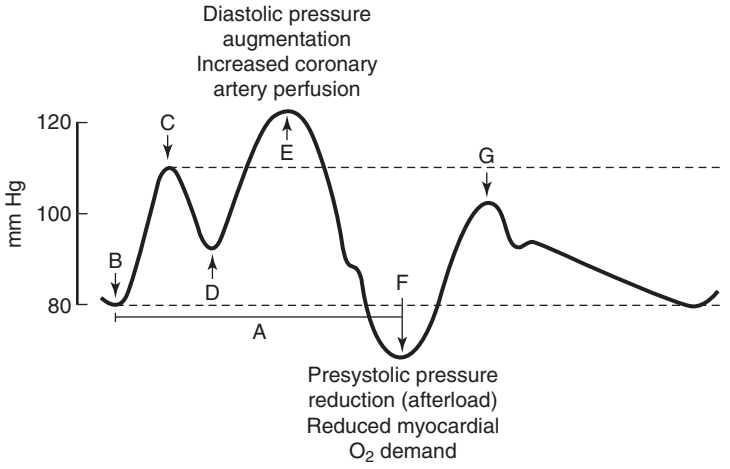

Fig. 9-2 Arterial waveforms during 2:1 intra-aortic balloon pump (IABP) counterpulsation. A, one complete cardiac cycle; B, unassisted aortic end-diastolic pressure; C, unassisted aortic systolic pressure; D, dicrotic notch (balloon inflation); E, diastolic augmentation; F, assisted aortic end-diastolic pressure; G, assisted systole.

post-angioplasty vessels, but no increase in vessels distal to significant stenosis (Fig. 9-2).

Indications. Before a diagnostic cardiac catheterization or interventional procedure, the patient should be treated medically to optimize hemodynamics and reduce myocardial ischemia. In unstable patients, an IABP may be required before proceeding with the catheterization. During diagnostic cardiac catheterization or interventional procedures, hypotension (not responding to volume loading or intravenous vasopressors) and medically refractory angina are important indications for IABP placement. In a prospective randomized study of IABP support for PCI in 1100 patients with acute myocardial infarction, Stone *et al.* found that IABP support did not improve the combined end point of death, reinfarction, infarct-related artery reocclusion, stroke, new-onset heart failure, or sustained hypotension compared to patients in the control arm.

Situations in which prophylactic IABP insertion should be considered in high-risk PCI are summarized in Box 9-4. As discussed previously, iliac-femoral angiography (or distal

Box 9-4

Indications and Contraindications for Intra-aortic Balloon Pump Counterpulsation During High-Risk PCI

Indications for Prophylactic Balloon Pump Placement

- Severely depressed ejection fraction
- PCI of sole remaining or primary remaining coronary artery or bypass graft
- Unprotected left main coronary artery PCI (especially if the right coronary artery is occluded)
- Ongoing ischemia
- Intractable ventricular arrhythmia believed due to ischemia
- Hemodynamic instability
- Hypotension
- Cardiogenic shock

Indications for "Rescue" Balloon Pump Placement

- Hemodynamic instability, hypotension, or cardiogenic shock
- Intractable ventricular arrhythmia believed due to ischemia
- Abrupt vessel closure (particularly if TIMI 3 flow cannot be quickly restored)

Contraindications

- Severe iliac/femoral atherosclerotic disease or tortuosity
- Aortic dissection or aneurysm
- Moderate or severe aortic regurgitation
- Bleeding diathesis
- Bypass grafting to femoral arteries or aorta
- Patent ductus (augments the abnormal shunting)
- Sepsis

aortic angiography with runoff) should be performed at the time of diagnostic catheterization, or at least preceding high-risk PCI, to assess for iliac-femoral atherosclerotic disease, extreme tortuosity, or arterial occlusion that would preclude placement of IABP. In patients with moderate degrees of iliac-femoral atherosclerosis or vessel tortuosity, select a lower profile (8F) or sheathless IABP. In high-risk PCI patients, even if an IABP is not placed prophylactically, obtain arterial access with a 4 or 5 French sheath in the femoral artery not being utilized for the PCI procedure in case emergent IABP placement becomes necessary.

Contraindications. Contraindications to IABP placement are listed in Box 9-4. The presence of a potential contraindication to IABP must be factored into decisions on whether to proceed with a high-risk PCI.

Complications of IABP most commonly result from a low puncture site, perforation of the superficial femoral artery, or forceful advancement of the catheter, damaging the arterial entry site. The most common serious complication of IABP is ischemia of the lower extremity, which occurs in approximately 5–10% of patients. Patients in whom an IABP is placed need to have careful assessment of distal pulses and tissue perfusion both prior to and after IABP insertion. Prolonged intra-aortic balloon counterpulsation is also associated with hemolysis and platelet destruction, and thus the blood counts of patients need to be closely monitored.

Despite the use of IABP during angioplasty in high-risk patients, there remains an in-hospital mortality of 6.2–19%, with a rate of vascular complications of 2.1–14%. The combination of IABP and a perfusion balloon is a particularly attractive physiologic combination in patients who appear to be at high risk.

Insertion Technique. Before the percutaneous insertion of an intra-aortic balloon, careful assessment of contraindications is required. A low abdominal aortogram will identify the course and extent of disease in the iliac and femoral vessels before the IABP insertion. For high-risk procedures, anticipate IABP placement and recall that the puncture site should be 2 cm below the inguinal ligament, similar to or slightly more proximal than a standard femoral puncture for cardiac catheterization. A puncture lower than the prescribed site may introduce the balloon into a superficial femoral artery too small to accept the large IABP catheter.

The balloon is inserted into either groin using standard Seldinger technique, as described in detail in *The Cardiac Catheterization Handbook.* Some manufacturers are making "sheathless" IABP balloon catheters, which are especially useful in the elderly and those with some peripheral vascular disease that requires support. Fluoroscopic observation of the balloon inflated above the renal arteries confirms optimal placement.

Percutaneous Cardiopulmonary Support

Percutaneous cardiopulmonary support (CPS) is rarely utilized during PCI and only at a few institutions. CPS involves the placement of a 16–20 French catheter inserted into the femoral vein, advanced into the right atrium, and the placement of a 16–20 French catheter into the femoral artery and advanced to the aorta. These catheters are connected in series to a flow probe, heat exchanger, and membrane oxygenator. Some CPS systems use a centrifugal pump that creates negative pressure to augment venous outflow as well as positive pressure to promote arterial inflow and circulatory flow and pressure.

As with IABP, iliac-femoral angiography must be performed prior to initiation of CPS. CPS should only be considered prophylactically in the extraordinarily high-risk PCI patient, and in such cases preferentially by a team experienced in CPS (including a trained perfusionist). It may be considered as an emergency procedure in patients who have severe cardiopulmonary compromise, although the time necessary to prepare and initiate the system is usually more than the patient's clinical status will allow. Although "stand-by" CPS can be considered in some patients, the necessity for CPS support is unpredictable. "Stand-by" CPS is generally not available at most institutions. CPS should only be used with the support of Cardiothoracic Surgery and Anesthesia specialists. In patients with no option for CABG, and no potential for cardiac transplant, there would be no purpose to institute CPS, maintaining hemodynamics without a heart function. It is important to remember that, because CPS does not increase regional myocardial blood flow or alleviate myocardial ischemia, acute coronary dissection or occlusion needs to be urgently addressed.

MANAGEMENT OF COMPLICATIONS IN HIGH-RISK PCI PATIENTS

Hypotension

Hypotension may result from myocardial ischemia, coronary perforation with cardiac tamponade, arrhythmia, contrast- or medication-induced anaphylaxis, or acute occult bleeding (e.g., retroperitoneal hematoma). This differential diagnosis of

hypotension must be evaluated quickly and therapy directed to the cause of the hypotension. After the first step of aggressive fluid resuscitation with normal saline, support blood pressure with inotropic agents such as dopamine (initial starting infusion rate of 5–10 μg/kg/min depending on degree of hypotension) or bolus dose norepinephrine (2.5–5 μg IV boluses as needed). An emergent echocardiogram will help assess cardiac tamponade. The treatment of anaphylaxis is discussed below.

Bradyarrhythmias and Heart Block

Bradyarrhythmias may occur as a result of ischemia of the sinus node (most commonly supplied via the right coronary artery). Symptomatic sinus bradycardia can initially be treated with atropine (0.5–1.0 mg IV), with repeat doses every 3–5 min as indicated, up to a usual total maximal dose of 3.0 mg.

Heart block may occur as a result of occlusion of the "dominant" artery (either the right coronary artery or the left circumflex artery) that supplies the atrioventricular node. Though there is no data to support this, intuitively one might believe that a patient with abrupt closure of the right coronary artery is more likely to develop heart block if the left circumflex artery is already occluded (and vice versa). Complete heart block with a slow ventricular escape rhythm should not be treated with atropine. Dopamine (initial infusion 5 μg/kg/min) may in certain circumstances ameliorate sinus bradycardia or heart block.

Patients on beta blockers or calcium channel blockers can be treated with glucagon (1 mg) or calcium chloride (1 ampoule, 13.6 mEq), although the effects of such therapy is more theoretical than established. For both bradycardia and heart block, severely symptomatic patients can be treated with transcutaneous or transvenous pacing. Ultimately, the treatment for both these conditions includes restoration of the compromised coronary artery blood flow.

Ventricular Tachyarrhythmias

Ventricular tachycardia (VT) and ventricular fibrillation (VF) may result from severe myocardial ischemia. Patients who develop VT and are hemodynamically stable can first be treated with intravenous antiarrhythmic therapy, amiodarone and

lidocaine (Box 9-5). Stable patients not responding to antiarrhythmic therapy can be treated with synchronized cardioversion (beginning at 100 J and increasing stepwise up to 360 J as necessary).

Patients who develop pulseless VT or VF have been treated with a precordial thump. However, immediate defibrillation is indicated (beginning at 200 J and increasing stepwise to 360 J). No other intervention should interfere with the immediate and rapid first three sequential attempts at defibrillation. Patients who do not convert after these three initial attempts at defibrillation should be treated with either epinephrine (1 mg IV, repeated every 3–5 min) or vasopressin (40 u IV × 1), with repeat defibrillation afterward. Patients who still do not respond can then be treated with amiodarone (300 mg IV

Box 9-5

Medications and Dosing Regimens for Arrhythmias Developing During High Risk PCI

Bradycardia

- Atropine 0.5–1.0 mg—can repeat as indicated every 3–5 min up to total dose of 2.0 mg

Stable Ventricular Tachycardia

- Amiodarone—can administer in one of two regimens, as dictated by clinical setting:
  - —Regimen 1: 150 mg over 10 min, followed by infusion rate of 1 mg/min
  - —Regimen 2: 300 mg IV rapid infusion after dilution in 20–30 ml fluid—can give additional 150 mg rapid infusions in similar manner as indicated
- Lidocaine 1.0–1.5 mg/kg IV bolus—can give additional boluses of 0.5–0.75 mg/kg IV as indicated, up to total dose of 3.0 mg/kg

Pulseless Ventricular Tachycardia/Ventricular Fibrillation

- Epinephrine 1 mg IV bolus—can repeat every 3–5 min
- Vasopressin 40 u IV bolus × 1 (either epinephrine or vasopressin can be given after three unsuccessful defibrillation attempts)
- Amiodarone 300 mg IV rapid infusion after dilution in 20–30 ml fluid—can give additional 150 mg rapid infusions in similar manner as indicated
- Lidocaine 1.0–1.5 mg/kg IV bolus—can give additional boluses of 0.5–0.75 mg/kg IV as indicated, up to total dose of 3.0 mg/kg

bolus after dilution in 20 ml fluid) or lidocaine (1.0–1.5 mg/kg IV bolus), with repeated attempt at defibrillation afterward. Simultaneous with these interventions should be the initiation of cardiopulmonary resuscitation. The advanced cardiac life support (ACLS) algorithm for the treatment of pulseless VT/VF is presented in Fig. 9-3.

During resuscitative efforts, coronary access, as well as guidewire position across the lesion, should be maintained to complete the ultimate recanalization of the coronary artery. It is unknown how defibrillation affects the coronary artery with the guidewire in place.

Pulselessness

Asystole. Asystole is usually the result of extensive myocardial ischemia. Asystole should be confirmed in two leads because it

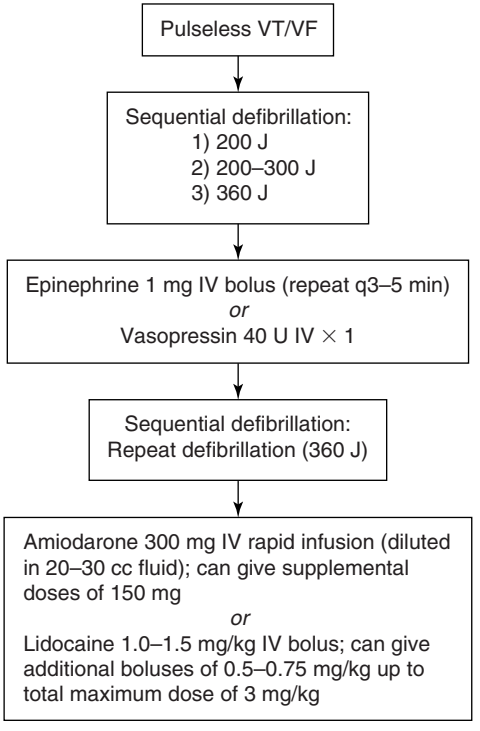

Fig. 9-3 ACLS algorithm for pulseless ventricular tachycardia (VT) or ventricular fibrillation (VF).

can be difficult to distinguish fine ventricular fibrillation from asystole. If the diagnosis is unclear, one should assume that fine ventricular fibrillation is present and treat accordingly. Right ventricular pacing should be instituted as quickly as possible. Atropine (1 mg) should be given and can be repeated in 5 min if necessary. External pacing can be employed if right ventricular pacing cannot be established immediately. Metabolic abnormalities, including hyperkalemia or severe pre-existing acidosis, may contribute to the genesis of the arrhythmia and may respond to the use of buffers. A solitary precordial thump may be employed.

Epinephrine, the catecholamine of choice for cardiac arrest, has vasoconstrictor alpha-adrenergic properties that make it superior to other alpha-adrenergic agents (methoxamine and phenylephrine), with comparable cardiac effects, but failure to increase central nervous system blood flow was much as phenylephrine. The dose of epinephrine is 0.5–1 mg given at least every 5 min. During cardiac arrest, higher rather than lower doses may be more efficacious. If systole is refractory to the above measures, emergency cardiopulmonary bypass should be considered.

Electromechanical Dissociation. Electromechanical dissociation (also called pulseless electrical activity, PEA) is a condition that is almost uniformly fatal unless the underlying cause can be identified and immediately treated. General treatment includes the use of epinephrine (1 mg every 5 min). Bicarbonate may be considered. If PEA is refractory, emergency cardiopulmonary bypass may be considered.

Underlying causes of PEA include:

- Hypovolemia, especially resulting from bleeding
- Pericardial tamponade, especially in patients with acute infarction, recent cardiac biopsy, recent endocardial pacer insertion, or uremia; if tamponade is suspected, blind pericardiocentesis is warranted
- Enhance vagal tone in patients with ischemic heart disease. Consider this whenever the heart rate is inappropriate for the degree of hypotension. Atropine is required
- Massive pulmonary embolism
- Tension pneumothorax, especially in patients on ventilators or in patients with central venous access above the

diaphragm. Fluoroscopy may be helpful if it is available immediately. If there is any suspicion of tension pneumothorax, the operator should carefully insert a needle attached to a glass syringe into the pleural space. If a tension pneumothorax is present, air under pressure will push the plunger out of the syringe.

Pulmonary Edema

Pulmonary edema during high-risk PCI may be the result of ischemia-induced depression of myocardial function and/or volume overload due to intravenous fluid infusion and high-osmolarity contrast. Initial measures may include administration of furosemide 20–40 mg intravenously (which initially acts as a venodilator well before its diuretic actions become significant) and/or intravenous nitroglycerin (which also acts primarily via venodilation). Inotropic support can be considered utilizing dobutamine (2.5–10 μg/kg/min). Patients refractory to these measures, particularly those with severely compromised left ventricular function, may require intubation, IABP support, or intravenous afterload reduction.

Anaphylactoid Reactions

Anaphylactoid reactions to contrast agents may include hypotension and shock. Although such reactions are exceedingly rare during PCI procedures, the operator should understand the treatment of this life-threatening condition. Recommendations by the Society for Cardiac Angiography and Intervention (SCAI) for treatment include: intravenous epinephrine with large volumes of normal saline, diphenhydramine, hydrocortisone, and, if unresponsive to therapy, an H_2 blocker and dopamine. Specific dosing regimens are given in Box 9-6.

Abrupt Vessel Closure

Dissection. Abrupt vessel closure is usually due to dissection in association with intracoronary thrombosis and has become an uncommon event. Abrupt vessel closure is much less an issue with the availability of coronary stents. Coronary stent implantation is the treatment of choice for dissection and has been responsible for a dramatic decrease in the need for emergency CABG reported in large registries over the last decade.

Box 9-6

Treatment of Severe Anaphylactoid Reactions

Recommendations from the Society of Cardiac Angiography and Interventions

Initial pharmacological therapy

- Epinephrine 10 μg/min IV until desired blood pressure response, then 1-4 μg/min to maintain desired blood pressure, given simultaneously with large volumes of normal saline
- Diphenhydramine 50–100 mg IV
- Hydrocortisone 400 mg IV

If unresponsive to initial therapy

- H_2-blocker therapy
  —Cimetidine 300 mg in 20 ml normal saline administered IV over 15 min
  —Ranitidine 50 mg in 20 ml normal saline administered IV over 15 min
- Dopamine 2–15 μg/kg/min IV infusion

Thrombosis. Although multiple studies have demonstrated that initiation of platelet IIb/IIIa inhibitor therapy at the time of PCI decreases ischemic complications, there is no data on the degree of benefit from the "bailout" use of platelet IIb/IIIa inhibitors once a complication such as abrupt vessel closure occurs. Nevertheless, the pathophysiology of abrupt vessel closure supports initiating platelet IIb/IIIa therapy. The biggest caveat to this recommendation is that, in patients who are highly likely to require emergency CABG, the cardiothoracic surgeon should be consulted when possible before administration of such agents.

Thrombus treatment regimes include adequate anticoagulation. ACT should be checked in patients being treated with either unfractionated heparin or bivalirudin and, if it is low, the heparin dose should be repeated. The ACT does not reflect the degree of anticoagulation in patients who have been treated with enoxaparin. Patients who have received a subcutaneous dose of enoxaparin (1 mg/kg) within the previous 8 hours usually have therapeutic levels of anti-factor-Xa activity. Those who have received their last dose 8–12 hours prior to intervention may benefit from an additional dose (0.3 mg/kg IV,

if they have not already received this "booster" dose at the time of PCI).

The data on administration of intracoronary thrombolytic therapy has been conflicting. This therapy is rarely used in current practice, particularly given the availability of platelet IIb/IIIa inhibitors.

There is little actual data to support the use of a thrombectomy device, such as AngioJet®, in the setting of abrupt vessel closure with thrombosis. However, this setting would certainly be a reasonable one in which to consider thrombectomy.

Slow Flow and No Reflow

Slow flow refers to the phenomenon in which blood flow (as assessed by contrast dye flow) in a treated and nonoccluded artery decreases from TIMI 3 to TIMI 1–2 after coronary intervention. No reflow refers to the phenomenon in which an occluded artery (usually in the setting of acute myocardial infarction) is successfully recannulated with PCI although actual blood flow in the artery is minimal. The occurrence of slow flow during PCI is usually accompanied by severe chest pain, ST segment elevations, and sometimes hemodynamic and/or electrophysiological deterioration. The mechanisms for no/slow reflow are only partially understood but appear to involve a variable combination of:

- Microcirculatory vasoconstriction
- Plugging from fibrin, platelets, thrombus, and leukocytes
- Microcirculatory structural damage or edema.

Treatment recommendations for slow or no reflow are as follows.

GP IIb/IIIa Blockers. Given the contribution of platelets and thrombus to slow flow (and no-reflow), "rescue" or emergent administration of a platelet IIb/IIIa inhibitor is prudent (despite few if any data).

Heparin. The ACT should be checked. Administer additional boluses of antithrombin therapy if the ACT is subtherapeutic. However, there are again no data that this benefits slow flow or no-reflow.

Vasodilator Therapy. Directed at the coronary microcirculation, one or more of the following agents are given intracoronarily:

- Verapamil (125–250 μg boluses)

- Nitroglycerin (100–200 μg boluses)
- Nitroprusside (50–100 μg boluses)
- Adenosine (18–40 μg boluses).

Preparation and administration of these medications is given in Box 9-7.

Atheroemboli

Atherosclerotic emboli appear to play a major role in the slow-flow phenomenon during treatment of degenerated saphenous vein grafts. Distal protection devices can decrease the incidence of complications during saphenous vein graft PCI. These devices should be considered for prophylactic use when possible. However, there is no role for these devices once atheroembolization has already occurred.

Box 9-7

Preparation and Administration Guidelines for Vasodilators Used for "Slow Flow" and "No Reflow"

Verapamil
- Usually comes in preparation of 5 mg/2 ml
- Take 1 ml of verapamil (2.5 mg) and mix in 19 ml = 2.5 mg/20 ml = 125 μg/ml
- Administer 250 μg – 2 ml

Adenosine
- Usually comes in preparation of 6 mg/2 ml
- Take 1 ml (3 mg) of adenosine and mix in 500 ml D5W = 3 mg/500 ml = 6 μg/ml
- Administer 24 μg (4 ml) boluses

Nitroglycerin
- Usually comes in concentration of 5 mg/ml
- Take 0.2 ml (1 mg) and mix in 10 ml = 100 μg/ml
- Administer 200 μg (2 ml)

Nitroprusside
- Usually comes in preparation of 50 mg/2 ml (25 mg/ml)
- Take 2 ml (50 mg) and mix in 500 ml bag = 100 μg/ml
- Administer 100 μg (1 ml)

Perforation

Coronary perforation becomes of major clinical significance when it leads to cardiac tamponade. Treatment of the artery itself can include:

- Inflation of a PTCA balloon proximal to the perforation, minimizing further efflux of blood into the pericardium
- Use of a perfusion balloon at the site of the perforation, preventing further efflux of blood while still maintaining blood flow to the myocardium distal to the perforation
- Placement of a coronary stent graft (such as the JOMED Jostent coronary stent graft) to "seal" the perforation.

Several perfusion balloons and several stent grafts should always be close at hand in the catheterization laboratory (not in a remote storage room or other distant location). It is the opinion of some that perforations should not be treated with implantation of an uncovered stent, since this may serve only to stretch the artery and keep the perforation patent.

Treatment of cardiac tamponade requires pericardiocentesis. Reversal of anticoagulation may facilitate hemostasis at the site of perforation and efflux of blood into the pericardium, but this benefit must be balanced against the risk of thrombosis occurring in the coronary artery being treated.

Major Bleeding

Decisions as to whether to "reverse" anticoagulation and antiplatelet therapy in patients with major bleeding complications must balance the risk of further bleeding with the risk of coronary arterial thrombosis. The anticoagulant effects of unfractionated heparin can be reversed with protamine sulfate (1 units protamine per 100 mg heparin). Protamine should be used with caution in patients who have received NPH insulin or are post-vasectomy. Protamine partially reverses the anticoagulant effects of low-molecular-weight heparin. Patients who have been treated with subcutaneous enoxaparin can be treated with 1 mg protamine per 1 mg enoxaparin when the last enoxaparin dose is within 0–8 hours of PCI, and 0.5 mg protamine per 1 mg enoxaparin when the last enoxaparin dose was 8–12 hours prior to PCI. Protamine is not effective in patients treated with a direct thrombin inhibitor (such as bivalirudin).

Both aspirin and clopidogrel are nonreversible platelet inhibitors whose antiaggregatory effects can only be reversed with platelet transfusion. As abciximab is an antibody fragment that binds tightly to the IIb/IIIa receptor, and as less free abciximab molecules are in circulation than molecules of tirofiban or eptifibatide, the effects of abciximab on platelet aggregation are reversed to a greater degree with platelet transfusion than those of either tirofiban or eptifibatide.

SUMMARY

Careful evaluation of angiographic, patient-related, and clinical factors can identify the high-risk PCI patient. Use of pharmacological and mechanical therapies may serve to decrease the risks of major complications during high-risk PCI. Adequate planning and laboratory preparation is essential to quickly address complications when they occur. Familiarity with both pharmacotherapy and mechanical therapy is essential when attempting high-risk PCI.

Suggested Reading

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10

NONANGIOGRAPHIC CORONARY LESION ASSESSMENT

William F. Fearon, Morton J. Kern, Z. Jacob Litwinczuk, and John McB. Hodgson

Although coronary angiography has revolutionized the cardiologist's ability to diagnose coronary artery disease, it does have certain limitations. For example, determining the clinical importance of an intermediate coronary lesion (diameter stenosis between 40% and 70%) based on angiography remains a challenge. Numerous studies have shown that, using angiography alone, physicians predict poorly which intermediate coronary lesion will prove to be ischemia-producing on a noninvasive stress imaging study. Angiography is also limited in its ability to detect an optimal immediate result after a percutaneous coronary intervention (PCI) and to predict a successful long-term outcome, prompting research for more sensitive means to assess and optimize PCI results. Finally, the resolution of standard angiography limits its ability to determine the presence and degree of collateral circulation.

Recent advances in technology have now made it possible to more accurately assess the functional significance of intermediate coronary lesions, the adequacy of angioplasty results, and the presence of collaterals by using sensor-guidewire-based methods of measuring coronary pressure and flow. Anatomic correlates to these physiologic measures have been identified using intravascular ultrasound (IVUS) which has been studied in many clinical scenarios as well.

The three most common technologies employed for nonangiographic coronary lesion assessment are:

- Intravascular ultrasound
- Coronary pressure wire

- Doppler coronary flow wire

Catheter-based anatomic and physiologic criteria associated with clinical outcomes are shown in Table 10-1.

INTRAVASCULAR ULTRASOUND

Background

Intravascular ultrasound is inherently different from physiologic measures in that it provides anatomic information, including plaque characteristics, lesion length, and lumen dimensions. It is complementary to angiography, allowing a more thorough investigation of the disease process occurring within the vessel wall. With advances in IVUS technology, interventionalists have been able to identify lesion-specific criteria for PCI, as well as criteria defining an optimal PCI. By better determining plaque characteristics, IVUS also is useful in guiding selection of interventional equipment, such as the need for plaque debulking. AHA/ACC/SCAI recommendations for coronary IVUS are shown in Table 10-2.

Two types of IVUS system exist, both utilizing 20–40 MHz silicon piezoelectric crystals: a mechanical system that relies on a rotating internal cable and a solid-state system externally mounted on a monorail-style catheter and controlled electronically. With the mechanical system, the imaging core

Table 10-1

Catheter-Based Anatomic and Physiologic Criteria Associated with Clinical Outcomes

Application	IVUS	CVR	rCVR	FFR
Ischemia detection	<3–4 mm^2	<2.0	<0.8	<0.75
Deferred angioplasty	–	>2.0	–	>0.75
Endpoint of angioplasty	–	>2.0–2.5 With <35% DS	–	>0.90
Endpoint of stenting	>9 mm^2 >80% reference area Full apposition	–	–	>0.94

CVR, coronary vasodilatory reserve; DS, diameter stenosis; FFR, fractional flow reserve; IVUS, intravascular ultrasound; rCVR, relative CVR; FFR, fractional flow reserve.

(From Kern MJ. Coronary physiology revisited: practical insights from the cardiac catheterization laboratory. *Circulation* 2000;101:1344–1351.)

Table 10-2

Recommendations for Coronary Intravascular Ultrasound

	Level of Evidence
Class IIA	
1. Evaluation of lesion severity at a location difficult to image by angiography in a patient with a positive functional study and a suspected flow-limiting stenosis	C
2. Assessment of a suboptimal angiographic result after coronary intervention	C
3. Diagnostic and management of coronary disease after cardiac transplantation	C
4. Assessment of the adequacy of deployment of the Palmaz–Schatz coronary stent, including the extent of stent apposition and determination of the minimal luminal diameter within the stent	B
Class IIB	
1. Determination of plaque location and circumferential distribution for guidance of directional coronary atherectomy	C
2. Further evaluation of patients with characteristic anginal symptoms and a positive functional study with no focal stenoses or mild CAD on angiography	C
3. Determination of the mechanism of stent restenosis (inadequate expansion versus neointimal proliferation) and to enable selection of appropriate therapy (plaque ablation versus repeat balloon expansion)	C
4. Preinterventional assessment of lesional characteristics as a means to select an optimal revascularization device	C
Class III	
When angiographic diagnosis is clear and no interventional treatment is planned	

(From Scanlon PJ, Faxon DP, Audet AM, *et al.* ACC/AHA guidelines for coronary angiography: executive summary and recommendations. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (Committee on Coronary Angiography) developed in collaboration with the Society for Cardiac Angiography and Interventions. *Circulation* 1999;99:2345–2357, with permission from the American Heart Association.)

rotates via a flexible drive shaft in order to sweep the transducer continuously through a 360° arc in the vessel. The rotation rate is 1800 revolutions per minute, generating 30 images per second. An example of this system is manufactured by Boston Scientific Corporation, Natick, MA.

The solid-state electronic system has 64 ultrasound transducers arranged circumferentially around the catheter tip and sequentially activated to produce a 360° image. An example of this design is manufactured by Volcano, Rancho Cordova, CA. IVUS catheters range in size from 2.9 to 3.5 French and can fit through a 6 French guide catheter. The IVUS catheter connects to a console, which displays and records the images to a videotape. Images also can be saved digitally, archived to CD-ROM in DICOM format and, in some cases, transferred directly to an image-archival network.

Several clinical applications for IVUS are summarized in Box 10-1.

Technique

After administration of heparin and positioning of a standard angioplasty guidewire in the distal coronary vessel, intracoronary nitroglycerin is given to avoid vasospasm. The IVUS catheter is then passed along the angioplasty wire, using a monorail technique, until the transducer is beyond the region of interest. The catheter can then be pulled back either manually or using an automated pullback device. The latter is necessary to determine lesion length and for volumetric analyses.

The mechanical system requires catheter flushing to remove air microbubbles and optimize imaging. Several other artifacts of images may occur. *Non-uniform rotational distortion* can occur

Box 10-1

Common Clinical Applications for Intravascular Ultrasound

- Assessment of lesion calcium
- Vessel dimensions
- Confirmation of atherosclerotic plaque
- Stent deployment
- Endothelial function research

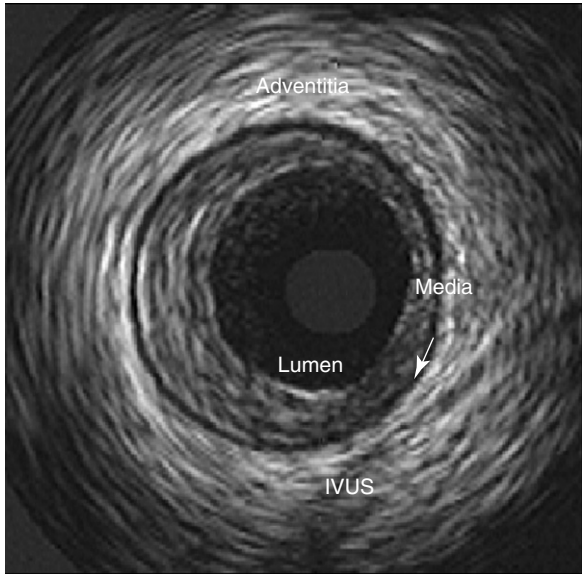
with a mechanical system due to an uneven drag on the catheter drive shaft leading to changes in the rotational speed. This artifact most commonly occurs in tortuous vessels and manifests as a smearing of one side of the image. A *ring-down artifact*, seen with the solid-state system, results in white circles surrounding the ultrasound catheter and precluding near-field imaging and is due to acoustic oscillations in the transducer resulting in high amplitude signals. Adjustments can now be made on the newer solid-state systems to minimize this problem.

Images from both systems are displayed in a tomographic, real-time video format. Currently, IVUS has a resolution of approximately 100–150 μm. A black or gray circle of approximately 1 mm representing the IVUS catheter is seen in the center of the image. It is surrounded by the lumen, a dark and echolucent zone with occasional faint speckling created by blood elements. Surrounding the lumen is the vessel wall and its three layers: the intima, media and adventitia. The intima in a normal vessel is too thin to be seen, and only a thin echogenic layer surrounding the lumen, which represents the internal elastic lamina, can be visualized. However, in diseased vessels the intima appears as a thicker echogenic layer surrounding part or all of the dark, echolucent lumen. The media is a relatively echolucent area between the internal elastic lamina and the external elastic lamina, which is often seen as an echodense layer at the media–adventitia interface. The adventitia is the most echodense layer in normal arteries and surrounds the media. A number of perivascular structures, such as veins and the pericardium, can be identified within the adventitia and aid in both axial and spatial orientation (Fig. 10-1).

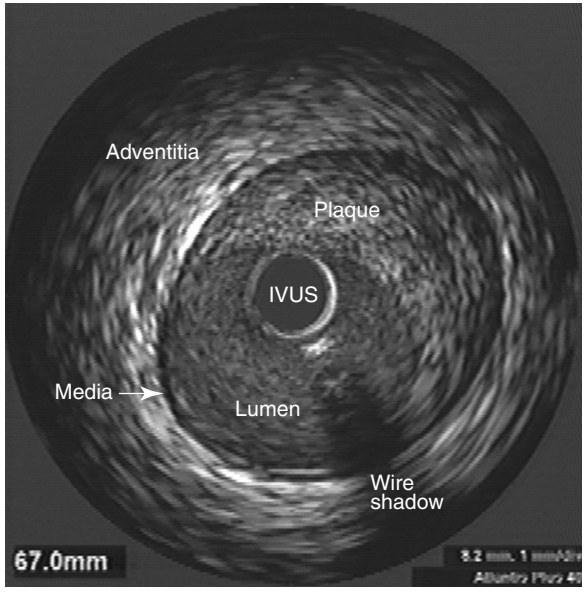
Setup

Integration of IVUS into the laboratory is critical for best use of the technology. Although the systems are portable, moving them into and out of the catheterization suite can be a frustrating experience. To minimize the problems associated with this process, it is important for several members of the support staff to take specialized training and assume responsibility for the equipment. We have found that the following preparations make use of IVUS most efficient:

- Specialized support staff familiar with operation of the equipment and image interpretation



A



B

Fig. 10-1 Intravascular ultrasound image, demonstrating: **A,** the three normal layers of the coronary artery—the adventitia is the outermost, separated by a dark echolucent line between the media and the lumen; **B,** an abnormal artery with eccentric plaque from 11 o'clock to 4 o'clock.

- A hard-wire ancillary video monitor on the angiographic monitor boom for display of the IVUS images to the physician operator
- Hard-wired fluoroscopic image output to the IVUS machine
- An ample supply of super VHS videotapes and IVUS report worksheets, kept with the machine
- A lapel microphone to be attached to the physician operator, allowing "voiceover" on the IVUS images to be recorded (critical to later interpretation). Alternatively, the existing in-room intercom microphone can be slaved to the IVUS console.
- Maintenance of a time log during the procedure that is keyed to the IVUS time code (facilitates later video review)
- Use of an automated pullback device, which standardizes the procedure to prevent too rapid scanning and eliminate much of the physician operator effect on image quality
- A system of maintenance for IVUS-related records, videotapes, and CD-ROMs.
- An image review station that is separate from the IVUS machine itself. Direct transfer of the DICOM images to an image-archival network allows review at many stations.

Image Features

Regardless of the imaging system used, the basic image features are described below from the center outward.

1. **Dead zone.** The black circular ring in the middle of the image is caused by the space occupied by the catheter.
2. **Catheter artifact.** A "halo" artifact around the catheter usually encroaches onto lumen areas and therefore may affect analysis. It may also encroach onto the signals transmitted from the vessel wall. These artifacts are related to either the imaging sheath or a property of ultrasonic imaging termed "ringdown" (disorganized near-field echo signals).
3. **Lumen.** The dark, echolucent area surrounding the catheter artifact signal is the lumen. With some higher-frequency scanners or under conditions of slow blood velocity, a fine speckle pattern may be seen in the lumen.
4. **Inner layer.** In a normal artery, the intima is often too thin to be seen reliably. The thin inner echogenic layer surrounding the lumen usually represents the internal elastic lamina. In a diseased coronary artery, the atheromatous

intima is seen as a thick echogenic layer surrounding the lumen. In vessels with mild to moderate atherosclerosis, a thin echodense layer at the intima–media interface can be seen, correlating histologically to the internal elastic lamina. This may be obscured in severely diseased atherosclerotic arteries.

5. **Middle hypoechoic layer.** The media, packed with smooth muscle cells and a few elastin fibers, appears as a relatively echolucent area. The external elastic lamina may sometimes be seen as an echodense layer at the media–adventitia interface.
6. **Outer echogenic layer.** The adventitia is seen as an echodense layer surrounding the hypoechoic media. The adventitia shows increased echodensity due to both the inhomogeneous histologic structures and the high elastin and collagen content. This structure has the most intense echoes in normal arteries. Echoes that are more intense than the adventitia are therefore abnormal. In this region, perivascular structures may also be observed (i.e., veins and pericardium).

Intravascular Ultrasound Plaque Morphology

In general, plaque may be classified as "soft" or "hard" based on whether the echodensity is less than or similar to the adventitia (Fig. 10-2).

Soft Plaque. More than 80% of the plaque area in an integrated pullback throughout the lesion is composed of thickened intimal echoes with homogeneous echo density less than that seen in adventitia.

Fibrous Plaque. More than 80% of plaque in an integrated pullback throughout the lesion is composed of thick and dense echoes involving the intimal leading edge, with homogeneous echo density greater than or equal to that seen for adventitia.

Calcified Plaque. Bright echoes within a plaque demonstrate acoustic shadowing and occupy more than 90% of the vessel wall circumference in at least one cross-sectional image of the lesion. The extent of calcification, defined as the presence of any hyperechogenic structure that shadows underlying

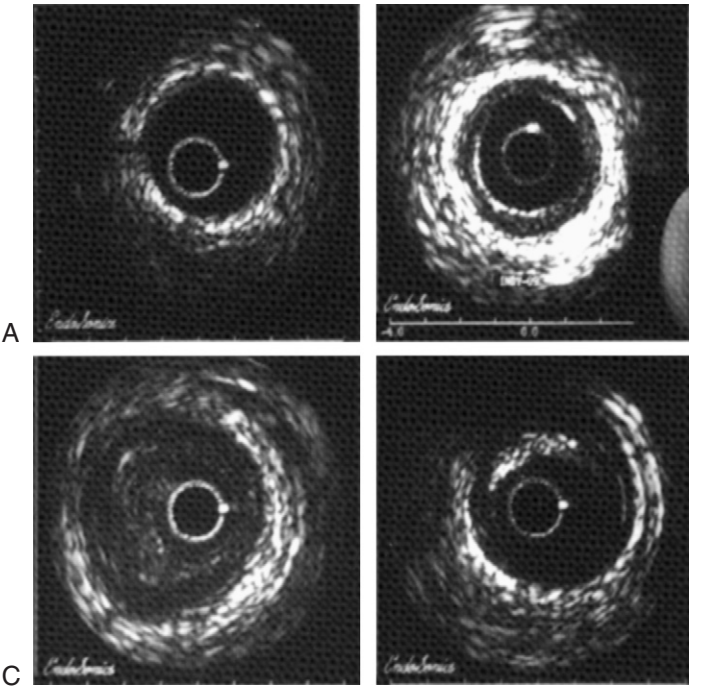


Fig. 10-2 Intravascular ultrasound images for various types of coronary arterial disease. **A,** Normal vessel. **B,** Mild atherosclerosis. **C,** Soft atheromata. **D,** Calcified atheromata. (Courtesy of John McB. Hodgson, MD.)

ultrasound anatomy, is reported as the degree of circumference in which shadowing is present. Calcium is also classified as deep or superficial (Fig. 10-3). Detection of calcium using IVUS can guide appropriate device selection, such as the need for high-speed rotational atherectomy.

Mixed Plaque. Bright echoes with acoustic shadowing encompass less than 90% of the vessel wall circumference, or a mixture of soft and fibrous plaque is seen with each component occupying less than 80% of the plaque area in an integrated pullback through the lesion.

Subintimal Thickening. Subintimal thickening involving reference vessel segments is defined as a concentric prominent

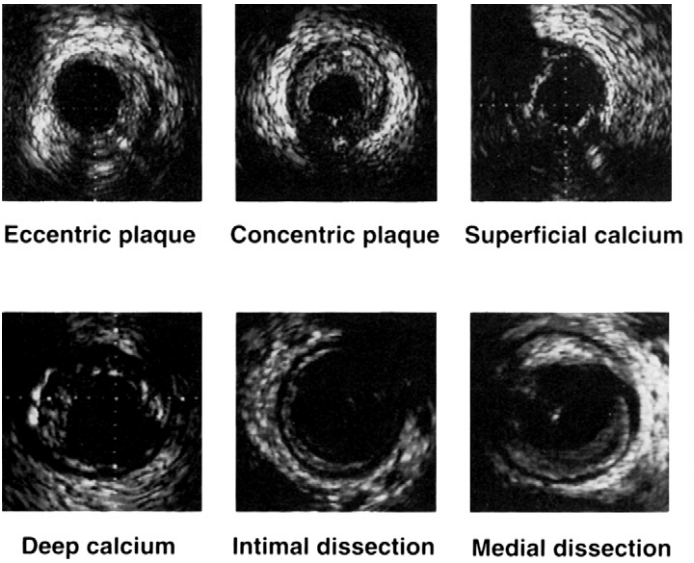


Fig. 10-3 Intravascular imaging illustrating different types of plaque morphology.

leading edge echo and a widened subintimal echolucent zone with a combined thickness of more than 500 μm.

Additional Plaque Features

Plaque Location. Plaque may be described as concentric or eccentric, with or without ulceration. In describing a noncon-centric plaque, its location is noted in relation to a clock, i.e., "plaque is present, extending from the 8 o'clock position to the 11 o'clock position, with calcium deposits seen at 9 o'clock."

Intimal Flap/Dissection. This is seen as a linear structure with or without a free edge. True and false channels can also be visualized. This characteristic motion of the intimal flap may also be seen within the lumen. Radiographic contrast injection can assist in defining the lumen and indicating whether there is communication of the lumen with an echo-free area below a flap. In some systems, blood flow can be colorized and may assist in defining dissections.

Thrombus. Fresh thrombus is a low to moderately echogenic or granular mass that occupies part of the lumen

and adjoins the adjacent wall; often it is mobile and has an irregular border. Edge definition is possible with contrast injection.

Aneurysm. Aneurysmal areas are expanded, thin-walled structures adjoining the lumen. They can be mistaken for branches, which have a similar appearance.

Side Branches. Side branches appear as "buds" with a loss of the intimal border. The location of the lesion in relation to branch vessels and, in particular, in relation to the coronary ostium, can be well visualized with IVUS, which can aid decisions regarding stent placement.

"Vulnerable Plaque" or Atherosclerotic Lesions at High Risk for Rupture Resulting in an Acute Coronary Syndrome. IVUS studies suggest that eccentric lesions and the presence of echolucent zones within the plaque representing lipid pools are major determinants of plaque vulnerability and increased propensity for rupture. Many unstable lesions demonstrate ulceration or thin mobile dissection flaps by IVUS. In addition, the presence of "positive remodeling," or compensatory enlargement of the vessel to accommodate plaque and maintain lumen, has also been found more commonly in unstable than in stable coronary lesions.

Dimensional Measurements

One of the major advantages of IVUS is its ability to prove precise measurements (Fig. 10-4). Several studies have analyzed the accuracy of ultrasound images for measuring lumen size and wall thickness. Correlations with histologic measurements have been uniformly high, although measurements of the dimensions of the layers and overall wall thickness have been reported to be less accurate than lumen area determinations. The lumen–intima and media–adventitia interfaces are generally accurate using ultrasound scanning; both interfaces show a relatively large increase in acoustic impedance as the beam passes through the layers. The intima–media interface may also provide a significant change in impedance, particularly in the presence of prominent internal elastic lamina. At this interface, however, there is a

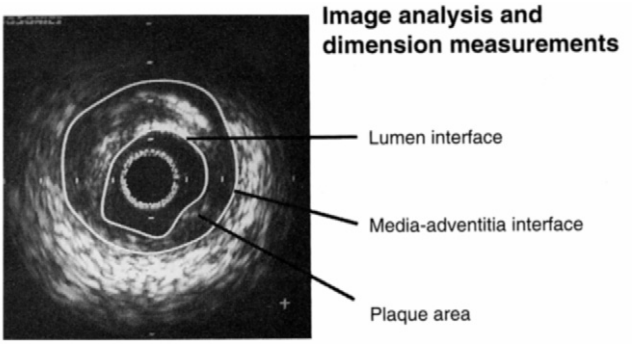


Fig. 10-4 Intravascular ultrasound images showing how ultrasound-derived measurements are obtained from planimetry of the lumen and media–adventitia interfaces. (Courtesy of John McB. Hodgson, MD.)

"trailing-edge" effect that can result in the spreading or blooming of the intimal image. The net result is that the transition is obscured, the intima appears thicker than by histologic determination, and the media appears correspondingly thinner. However, wall thickness using the combined intima and media corresponds closely to the histological dimensions.

All ultrasound measurements are performed on end-diastolic images, unless specified otherwise. Artery lumen dimensions are quantified from images of proximal, distal, or reference vessel segments and within the target lesion(s) or stent. The flowing measurements are routinely obtained.

- **Lumen and vessel diameters.** Minimal, maximal, and mean diameters may be obtained
- **Percentage diameter or area stenosis** is the lumen diameter or area within the lesion segment divided by the lumen diameter or area within the reference segment. This is similar to the measures made by angiography.
- **Total vessel area** is integrated area central to the medial adventitia border. The vessel cross-sectional area is the area confined within the external elastic lamina or the media–adventitia interface
- **Lumen area** is the integrated area central to the leading-edge echo. The area is confined within the lumen-intima interface. If the catheter is tangential, the lumen area is slightly overestimated

- **Wall area (intima and media)** equals total area minus lumen area. In abnormal vessels, this is the plaque area (also called plaque plus media area)
- **Percentage plaque area** (also called plaque burden or percentage cross-sectional narrowing [%CSN]) equals total vessel area minus lumen area divided by total vessel area:

  Percentage plaque area = (total area – lumen area/total area) × 100
- **Indices of Eccentricity.**
  - —A **lesion eccentricity index** (L_{ECC}) is calculated by lumen dimensions:

    L_{ECC} = maximum diameter/minimum diameter
- **Plaque Distribution** is classified into three categories:
  - —*Concentric Plaque.* Maximum plaque thickness (leading-edge plus sonolucent zone) <1.3 times minimum plaque thickness
  - —*Moderately Eccentric Plaque.* Maximum plaque thickness (leading edge plus sonolucent zone) 1.3–1.7 times minimum plaque thickness
  - —*Severely Eccentric Plaque.* Maximum plaque thickness (leading edge plus sonolucent zone) >1.7 times minimum plaque thickness.

Assessing Intermediate Coronary Lesions

Intravascular ultrasound parameters for predicting the clinical significance of intermediate coronary lesions have been identified and closely studied. For example, in one study comparing a number of IVUS measurements in 70 patients to the results of nuclear perfusion imaging, investigators found that a minimum lumen area of more than 4 mm^2 had a sensitivity of 88% and specificity of 90% for predicting ischemia on the noninvasive test. Furthermore, in a large retrospective study of patients with intermediate lesions in whom PCI was deferred, a minimum lumen area of more than 4 mm^2 based on IVUS was a useful predictor of freedom from adverse events.

Intravascular ultrasound findings in patients with intermediate lesions have also been compared to fractional flow reserve (FFR) and coronary flow velocity reserve (CFVR) findings. There is a strong correlation between the minimum lumen area based on IVUS and the FFR result. In one study, a

minimum lumen area of less than 3.0 mm^2 had a sensitivity of 83% and a specificity of 92% for predicting an FFR of less than 0.75. There was also a strong correlation between the IVUS-derived percentage area stenosis—defined as (reference lumen area – minimum lumen area)/reference lumen area)—and FFR; in this case the sensitivity and specificity were 92% and 88% respectively.

Intravascular-ultrasound-derived minimum lumen area also correlates to CFVR findings in patients with intermediate coronary lesions. The diagnostic accuracy of a minimum lumen area of less than 4.0 mm^2 was 89% for predicting a CFVR of less than 2.0 in one study. Again, other anatomic parameters, such as lesion length, were also found to be important predictors of an abnormal CFVR.

Interrogating intermediate left main coronary lesions is another area where IVUS is commonly employed. Unfortunately, there is no universal agreement regarding IVUS criteria for a significant left main coronary lesion; however, a lesion with an absolute minimum lumen area of less than 6 mm^2 and/or a percentage area stenosis of more than 60% is generally considered a significant left main stenosis. One study showed an increased 1-year event rate in patients with left main diameters less than 3 mm, especially in patients with diabetes.

In summary, the IVUS parameters for predicting ischemia have less clear absolute cutoff values, and a combination of IVUS measurements, including minimum lumen area, area stenosis and lesion length, is often necessary to improve the correlation between physiologic measures and IVUS when evaluating intermediate coronary lesions.

Assessing Percutaneous Coronary Interventions

Intravascular ultrasound has been studied extensively in the setting of assessing and optimizing PCI. It is a valuable tool for determining optimal lumen expansion after angioplasty and for ensuring ideal stent expansion and apposition after stenting. IVUS allows a more thorough evaluation of potential residual dissections, particularly involving the stent edges. After angioplasty, the residual plaque burden as assessed by IVUS is a strong predictor of restenosis, independent of angiographic findings. After stenting, use of IVUS universally leads to improved stent expansion and larger final lumen dimensions.

In many studies, this has translated into lower restenosis rates and better long-term outcome. Both absolute and relative criteria have been put forth for gauging an optimal stent result. Complete stent apposition to the vessel wall, a minimum stent area of more than 90% of the average reference area, or 100% of the smallest reference area, and symmetric stent expansion with the minimum/maximum lumen diameter more than 0.7 are commonly cited relative criteria. An absolute minimum stent area that is more than 7 mm^2 is a useful absolute criterion.

There are data to suggest that an IVUS-guided PCI results in a lower target vessel revascularization rate during followup. This has been attributed to the improved stent expansion achieved with IVUS guidance compared to angiography alone. Whether routine IVUS is a cost-effective approach to PCI is still under investigation, although one randomized study documented cost savings over 2 years of followup. With the advent of drug-eluting stents, in which maximal expansion may not be as critical, IVUS guidance may appear less valuable, although ensuring complete lesion coverage by accurate length measures, selection of the appropriate diameter stent, evaluating for calcium that may impair expansion or delivery, documenting appropriate apposition, and ensuring the absence of peri-stent dissection or hematoma should continue to be important (Fig. 10-5).

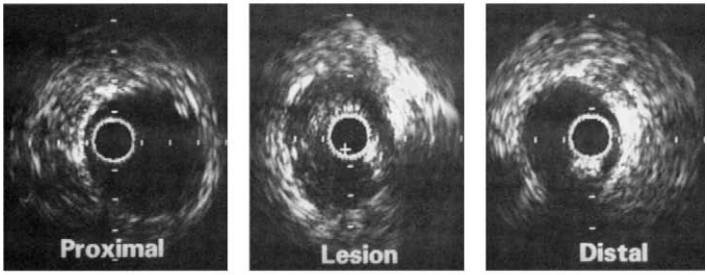
Assessing Complications

Following coronary interventions, vessel stretching, plaque redistribution or shifting, plaque removal, plaque fissuring, and dissections can be clearly outlined by IVUS. The morphologic characteristics, as well as specific dissection patterns following intervention, have shown that dissections are dependent on differential plaque types, usually occurring at the edge of calcified segments.

Diagnosis of Allograft Vasculopathy

Intravascular ultrasound has been an excellent means of diagnosing and quantifying cardiac transplant vasculopathy. Routine annual angiographic studies often reveal "normal" vessels in the transplant patient, whereas IVUS studies of the same cohort reveal diffuse intimal hyperplasia.

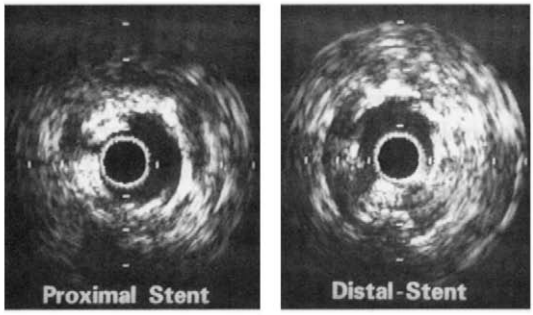
Arterial segments



LA: 9.36 mm^2	2.32 mm^2	4.69 mm^2
MLD: 3.45 mm	1.72 mm	2.44 mm

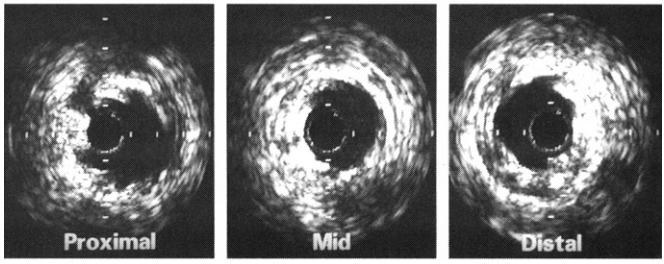
A **3.0/2.5 tapered balloon was selected**

Initial stent assessment



	LA: 5.47 mm	LA: 5.03 mm
B	(90% target: 8.4 mm^2)	(90% target: 4.22 mm^2)

Final stent assessment



LA: 8.41 mm^2	6.33 mm^2	7.03 mm^2
MLD: 3.27 mm	2.84 mm	2.99 mm

C **Criteria met for adequate stent deployment**

Progression and Regression of Coronary Atherosclerosis

Because of the limitations of angiography in defining wall structure and pathology, IVUS use for quantifying and qualitatively appraising extent and progression of atherosclerosis may be especially helpful for regression trials with primary or secondary intervention. Several studies to assess atheroma progression and regression after randomization to a lipid-lowering regimen (diet and/or medication, exercise, and stress reduction) or "regular care" are now under way. All patients will have angiography and IVUS evaluation at baseline and will be reevaluated at 6–12 months. Initial results have already documented slowing of lesion progression and enhanced echogenicity, possibly indicating reduced lipid content. Serial assessments of the progression of intimal proliferation in cardiac transplant patients with angiography and IVUS have documented accelerated vasculopathy, occurring most actively within the first year following transplant.

Evaluation of Vasomotor Tone

Intracoronary ultrasound yields a beat-to-beat analysis of vascular compliance (systolic-to-diastolic lumen area ratio). Additionally, the effects of vasoactive substances can be monitored directly and continuously during IVUS imaging. These unique advantages allow study of the early effects of atherosclerosis and/or intervention on vessel compliance and also allow evaluation of endothelial function in patients with varying degrees of atherosclerosis.

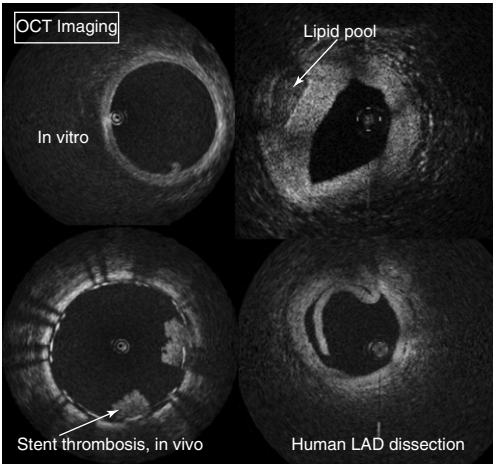
THE FUTURE: OPTICAL COHERENCE TOMOGRAPHY

In the future fiber optic technology will provide topographical, real-time images of the coronary artery in a manner similar to IVUS but with much finer resolution. The glass fibers that

Fig. 10-5 **A,** Initial intravascular ultrasound evaluation showing proximal, lesion and distal segments. The lumen area (LA) and minimal lumen diameter (MLD) is shown for each. Because of the tapering nature of this vessel, a tapered balloon was selected for stent deployment. **B,** After initial inflation at 12 atm, the lumen areas within the stent do not meet the criteria for end of procedure (90% of referring segment). **C,** After repeat dilatation at 16 atm, the lumen areas and minimal luminal diameters are enlarged and the implantation of the stent is completed. (Courtesy of John McB. Hodgson, MD.)

transmit the light for imaging constitute a fiberoptic array with a distal lens that serves to focus the transmitted light. An optical coherence tomography (OCT) imaging catheter will permit studies with tissue characterization to the 10–20 μm level resolution.

The optical catheter is introduced into the artery (over a guidewire if needed), and a small, compliant balloon is inflated to block antegrade blood flow. A continuous flush system of warm saline replaces blood to clear the viewing field. This technique has allowed direct visual identification of thrombus, arterial dissection, and plaque surface characteristics (Fig. 10-6).



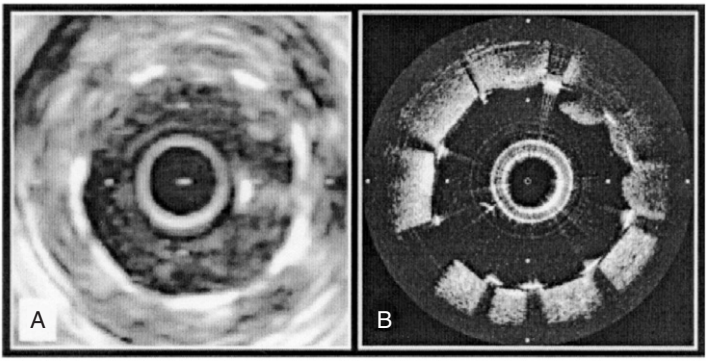


Fig. 10-6 Optical coherence tomographic imaging of coronary arteries: images from *(upper) in vitro* and *(lower) in vivo* models.

CORONARY PRESSURE WIRE

Background

The coronary pressure wire is similar to a standard 0.014 inch. angioplasty guidewire with a high-fidelity pressure transducer mounted 3 cm from the tip of the wire, at the junction of the radio-opaque and radiolucent segments. Pijls and De Bruyne developed and validated an index for determining the physiologic impact of coronary stenoses, called the myocardial fractional flow reserve. FFR is defined as the mean distal coronary pressure, measured with the pressure wire, divided by the mean proximal coronary or aortic pressure, measured with the guide catheter, during maximal hyperemia.

Rationale of Coronary Reserve Derived from Pressure Measurements

As demonstrated by Pijls and De Bruyne, measuring the resting gradient across a stenosis does not accurately predict the presence of ischemia on a noninvasive stress test; it is critical to maximize flow across the stenosis, mimicking exercise, and resulting in the peak transstenotic gradient.

Pressure measurements during hyperemia are another means to determine coronary flow reserve. When blood flows from the proximal to the distal part of the normal epicardial coronary artery, virtually no energy is lost and, therefore, the pressure remains constant throughout the conduit. In the case of epicardial coronary narrowing, potential energy is transformed into kinetic energy and heat when blood traverses the lesion. The resultant pressure drop reflects the total loss of energy. To maintain resting myocardial perfusion at a constant level, a decrease in myocardial resistance compensates for the pressure loss due to the epicardial narrowing. Arteriolar resistance decreases to increase the flow. The decrease in myocardial resistance reserve is proportional to the transstenotic pressure gradient and hence the latter represents an index of the physiologic consequences of a given coronary narrowing on the myocardium.

Concept of Fractional Flow Reserve

The relationship between pressure gradient and myocardial blood flow during maximal arteriolar vasodilation represents

the fractional flow reserve, which is defined at the ratio of hyperemic flow in the presence of an epicardial coronary stenosis to normal (maximal) hyperemic flow in the same artery without the stenosis. The maximal blood flow in the presence of a stenosis is expressed as a fraction of its normal expected value if there was no lesion. A fractional flow reserve can be derived (Box 10-2) separately for the myocardium, the epicardial coronary artery, and the collaterals, based on several assumptions regarding translesional pressure measured during maximal hyperemia. Figure 10-7 illustrates the data to derive FFR_{myo}.

The advantages of FFR are that it has an absolute normal value of 1.0; it is not affected by hemodynamic perturbations because all measurements are made during maximal coronary and microcirculatory vasodilation; and it is an index that is specific for epicardial coronary artery disease, which can be applied in patients with multivessel disease or prior myocardial infarction. In a patient with prior myocardial infarction, where chronic microcirculatory abnormalities result in impaired vasodilatory capacity, a moderate stenosis may

Box 10-2

Calculations of Fraction Flow Reserve from Pressure Measurements Taken During Maximal Arterial Vasodilation

Myocardial fraction flow reserve (FFR_{myo}):

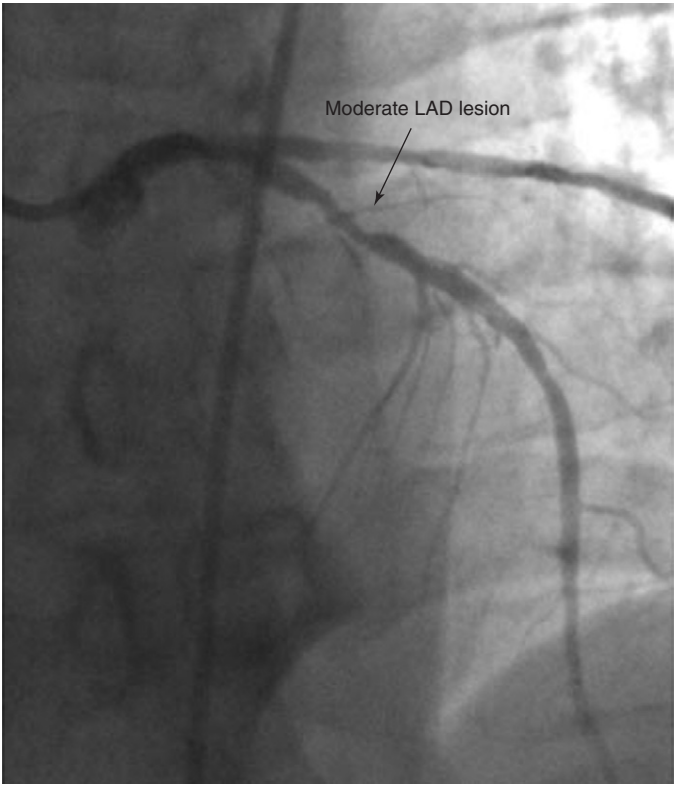
$$FFR_{myo} = 1 - \Delta P/P_a - P_v$$
$$= P_c - P_v/P_a - P_v$$
$$= P_c/P_a$$

Coronary fractional flow reserve (FFR_{cor}): $FFR_{cor} = 1 - \Delta P(P_a - P_w)$

Collateral fractional flow reserve (FFR_{coll}): $FFR_{coll} = FFR_{myo} - FFR_{cor}$

Note: All measurements are made during hyperemia except P_w: P_a, Mean aortic pressure; P_c, distal coronary pressure; ΔP, mean translesional pressure gradient; P_v, mean right atrial pressure; P_w, mean coronary wedge pressure or distal coronary pressure during balloon inflation.

From: Pijls NHJ, van Som AM, Kirkeeide RL, *et al.* Experimental basis of determining maximum coronary, myocardial, and collateral blood flow by pressure measurements for assessing functional stenosis severity before and after percutaneous transluminal coronary angioplasty. *Circulation* 1993;87:1354–1367.



A

Fig. 10-7 Calculation of fractional flow reserve (FFR). **A,** An example of FFR measured to assess the intermediate left anterior descending coronary lesion(s) is shown above.

Continued

result in a higher FFR than in a patient with a similar stenosis but a healthy microcirculation that permits a significant increase in flow across the stenosis, generation of a larger gradient, and hence a lower FFR. FFR remains valid in the setting of chronic myocardial infarction, however, because it still describes to what extent removing an epicardial stenosis will improve flow to the myocardium during stress. FFR values of more than 0.8 in patients more than 6 days after acute myocardial infarction correlate with perfusion imaging for viability.

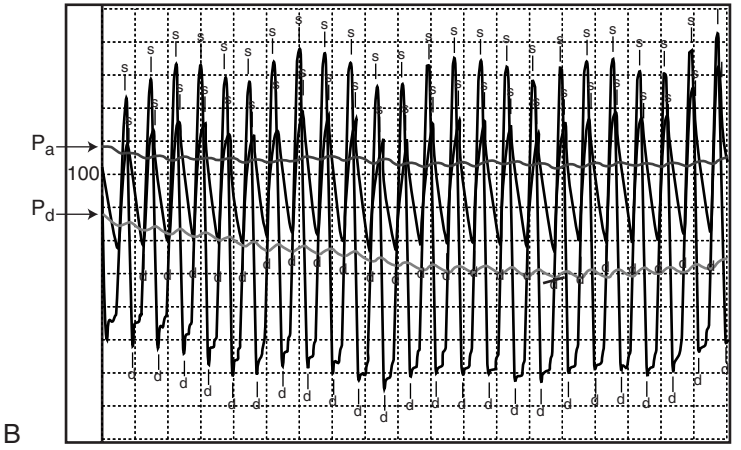


Fig. 10-7, cont'd **B**, P_a = 105, P_d = 70, FFR was 0.65.

Continued

Technique of FFR

Fraction flow reserve can be measured easily using as small as a 5 or 6 French guide catheter and either of two available pressure wire systems (Radi Medical System, Uppsala, Sweden or Volcano, Rancho Cordova, CA). The pressure wire is connected to the system's pressure analyzer and calibrated outside the body. Heparin and intracoronary nitroglycerin are administered. The wire is then advanced so that the pressure transducer is positioned at the ostium of the coronary artery. Pressure readings from the pressure wire are equalized to the guide catheter reading before crossing the stenosis.

The wire is then advanced distal to the coronary lesion. Maximal hyperemia is induced using intracoronary adenosine (> 20 μg for the right coronary artery, >30 μg for the left coronary artery), intravenous adenosine (140 μg/kg/min), or intracoronary papaverine (10–20 mg). The ratio of the mean distal pressure to mean proximal pressure during maximal hyperemia is calculated as the FFR. Intravenous adenosine, the longer acting hyperemic stimulus, allows the performance of a slow pullback of the pressure wire, which can be helpful in identifying the exact location of the pressure dropoff and determining the presence of diffuse disease. If a PCI is deemed

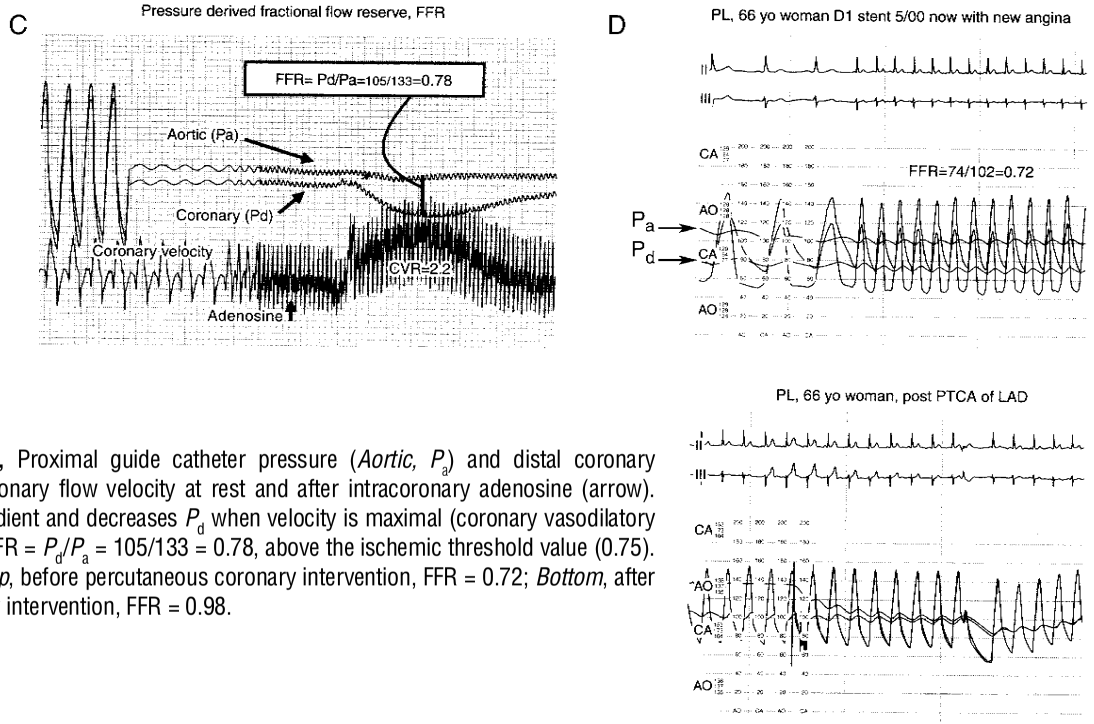


Fig. 10-7, cont'd **C,** Proximal guide catheter pressure (*Aortic, P_a*) and distal coronary pressure (P_d) and coronary flow velocity at rest and after intracoronary adenosine (arrow). Hyperemia widens gradient and decreases P_d when velocity is maximal (coronary vasodilatory reserve [CVR] = 2.2, FFR = P_d/P_a = 105/133 = 0.78, above the ischemic threshold value (0.75). **D,** Patient example: *Top*, before percutaneous coronary intervention, FFR = 0.72; *Bottom*, after percutaneous coronary intervention, FFR = 0.98.

necessary, it can be performed using the pressure wire as the angioplasty guidewire. After the procedure, FFR can be remeasured to assess the adequacy of the intervention. Finally, at the end of the procedure, the pressure wire should be pulled back so that the transducer is positioned at the coronary ostium to confirm equal pressure readings, or the lack of pressure drift, from the pressure wire.

Controversy regarding use of intravenous versus intracoronary adenosine was addressed by Jeremias *et al.*, who examined differences in FFR between intracoronary (15–20 μg in the right, and 18–24 μg in the left coronary artery) and intravenous adenosine (140 μg/kg/min) in 52 patients with 60 lesions. Mean percentage stenosis was 56 ± 24% (range 0–95%). The mean FFR was 0.78 ± 0.15 with a range of 0.41–0.98. There was a strong and linear relationship between intracoronary and intravenous adenosine (r = 0.978 and $p < 0.001$). The mean measurement difference for FFR was –0.004 ± 0.03. A small random scatter in both directions of FFR was noted in 8.3% of stenosis, where intracoronary adenosine FFR was more than 0.05 compared with intravenous FFR, suggesting a suboptimal intracoronary hyperemic response. Changes in heart rate and blood pressure were significantly greater with intravenous adenosine. Two patients with intravenous, but none with intracoronary adenosine, had side effects of bronchospasm and nausea. These data indicated that intracoronary adenosine is equivalent to intravenous infusion for determination of FFR in large majority of patients. However, in a small percentage of cases, coronary hyperemia was suspected to be suboptimal with intracoronary adenosine, suggesting that a repeated higher intracoronary adenosine dose may be helpful (some investigators use up to 48 μg for the right coronary and up to 96 μg for the left coronary).

The major pitfall associated with measuring FFR is the potential for incomplete hyperemia. If maximal flow down the vessel does not occur, the maximal pressure gradient will not be detected and the full impact of the coronary disease will be underappreciated (i.e., FFR will be overestimated). If this is suspected, care should be taken to assure intracoronary delivery of the hyperemic agent, if using intracoronary adenosine or papaverine. Alternatively, intravenous adenosine should be administered. Occasionally, larger guide catheters

can become partially occlusive of the coronary ostium as hyperemia is induced and impair maximal flow; removing the guide catheter from the coronary ostium after giving the hyperemic agent will help to avoid this pitfall.

Assessing Intermediate Coronary Lesions

A number of groups have confirmed Pijls and De Bruyne's initial findings that an FFR of less than 0.75 correlates with ischemia on an exercise test, nuclear perfusion imaging study, or stress echocardiogram. The sensitivity of FFR for predicting ischemia is in the high 80% range, with specificity in the high 90% range. Moreover, a randomized trial has demonstrated the safety of deferring PCI in patients with an FFR of more than 0.75. In this study, patients referred for PCI and found to have intermediate coronary lesions had their FFR measured. If the FFR was more than 0.75, the patients were randomized to performance of PCI as planned, or to deferral of PCI. After 2 years, the event-free survival rate was not significantly higher in the deferral group compared to the performance group, 89% vs 83%.

Fraction flow reserve has also been compared directly to CFVR and relative CFVR (rCFVR). As one might expect, the two indices that are epicardium-specific, FFR and rCFVR, correlate best with each other, while CFVR often does not correlate with either, particularly in the presence of microvascular disease. For this reason, FFR and rCFVR may be more appropriate measures in patients with known microvascular disease (or after PCI when transient microvascular dysfunction is common) if one is interested solely in the status of the epicardial artery.

Assessing Percutaneous Coronary Interventions

Measuring FFR after PCI to assess the adequacy of the intervention has also been studied extensively. A combination of an FFR of more than 0.90 and a residual diameter stenosis of less than 35% in patients after angioplasty alone was shown to predict a significantly higher event free survival rate compared to those in whom these parameters were not achieved. After stenting, an FFR $\geq$0.94 if using intravenous adenosine, and $\geq$0.96 if using intracoronary adenosine, correlated with IVUS determinants of optimal stent deployment, such as minimum stent area.

Significance of Abnormal Physiology after PCI

A normal FFR after PTCA is associated with stent-like late clinical outcomes (Bech *et al.*). In 43% of patients with optimal quantitative coronary angiography, a residual diameter stenosis of less than 35%, and good functional (FFR >0.90) results (26 of 60 single vessel angioplasty) had event-free survival rates that were significantly better at 6 months (92% vs 72%, $p = 0.047$), 12 months (92% vs 69%, $p = 0.028$), and 24 months (88% vs 59%, $p = 0.014$) compared to those patients with an FFR of less than 0.90. No improvement in clinical outcome was gained by additional stenting.

Coronary pressure measurements after stenting also predict adverse cardiac events at followup. Pijls *et al.* examined 750 patients with postprocedural FFR and related these findings to major adverse cardiac events at 6 months. In 76 patients, 10.2%, one adverse event occurred. Five patients died, 19 experienced myocardial infarction, and 52 underwent at least one repeat target vessel revascularization. Fractional flow reserve immediately after stenting was an independent variable related to all types of events. In 36% of patients, FFR normalized (>0.95) with an event rate of 5%. In 32% of patients with post-procedure FFR between 0.90 and 0.95, event rate was 6%. In the remaining 32% with FFR less than 0.90, event rates were 20%. In 6% of patients with FFR less than 0.80, the event rate was 30%. The authors concluded that FFR after stenting is a strong predictor of outcome at 6 months. These data suggests that both edge stent subnormalization and diffuse disease are associated with a worse long-term outcome.

Assessing Collateral Flow

The coronary pressure wire can be used to assess the presence and degree of collateral circulation in a fashion that is more sensitive than angiography alone. The pressure-derived fractional collateral flow is defined as the mean coronary wedge pressure (distal coronary pressure during balloon occlusion) divided by the mean aortic pressure (if the central venous pressure is abnormal, then it should be subtracted from both the wedge and aortic pressures). In general, a pressure-derived fractional collateral flow of 0.25 or more suggests sufficient collaterals to prevent ischemia during PCI. Furthermore, these patients have a significantly lower adverse event rate during

followup compared to those with insufficient collaterals at the time of PCI (pressure-derived collateral flow <0.25). Pressure-derived collateral flow has also been studied in patients with acute myocardial infarction and has been shown to be the major determinant of left ventricular recovery after primary PCI. Unfortunately, this technique for assessing collaterals is limited by the requirement for coronary artery occlusion.

Coronary Thermodilution Blood Flow Measurements Using the Coronary Pressure Wire

Recently, software has been developed that allows simultaneous measurement of FFR and coronary flow reserve (CFR) using the pressure wire. CFR is measured using a novel coronary thermodilution technique, whereby the pressure transducer serves as a distal thermistor and the shaft of the pressure wire as a proximal thermistor. In this manner, the resting mean transit time of room-temperature saline injected down the coronary artery can be measured with the pressure wire and compared to the hyperemic mean transit time. The ratio of resting to hyperemic mean transit times serves as an estimate of CFR. FFR using the standard technique can be measured simultaneously (Fig. 10-8).

Thermodilution CFR (CFR_{thermo}) is calculated as follows. CFR is defined as the ratio of hyperemic divided by resting coronary flow (F).

$$\text{CFR} = \frac{F \text{ at hyperemia}}{F \text{ at rest}}$$

Flow is the ratio of the volume (V) divided by T_{mn}. Thus, CFR can be expressed as:

$$\text{CFR} = \frac{V/T_{mn} \text{ at hyperemia}}{V/T_{mn} \text{ at rest}}$$

Assuming that the epicardial volume (V) remains unchanged, CFR can be calculated as:

$$\text{CFR} = \frac{T_{mn} \text{ at rest}}{T_{mn} \text{ at hyperemia}}$$

Coronary thermodilution CFR was initially validated in an *in vitro* model and *in vivo* animal model. Subsequently, it has been shown to correlate with CFVR measured with a Doppler

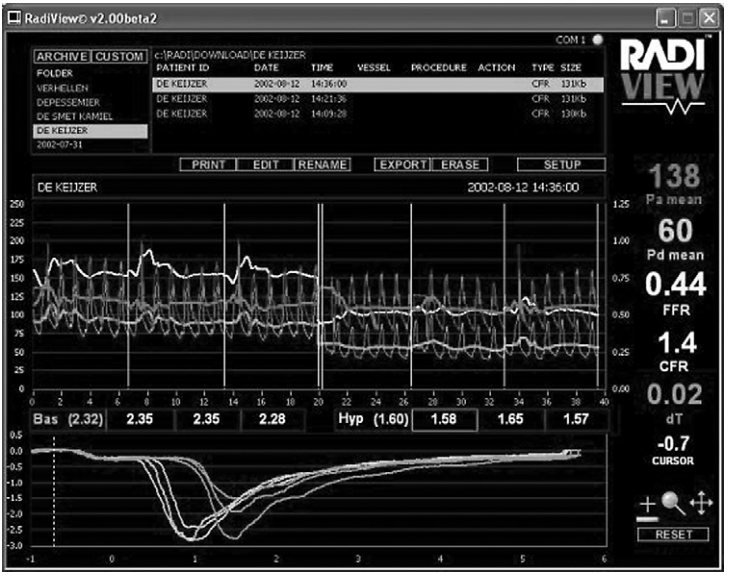


Fig. 10-8 Example of simultaneous pressure and temperature tracings. The top tracings represent central aortic pressure (Pa) and distal coronary pressure (Pd), and FFR (Pd/Pa). The lower tracings are temperature tracings recorded by the proximal (shaft) and distal sensors. The half-time of injection was derived from the proximal thermodilution curve. Coronary flow velocity is calculated from the distal thermodilution. (Courtesy of Radi Medical, Uppsala, Sweden.)

wire in humans. A similar wire system that measures pressure-derived FFR and Doppler-derived CFVR is undergoing preliminary testing. Because of the coexistence of epicardial and microcirculatory disease in many patients, the ability to reliably and easily distinguish one from the other may be an important addition to the interventional cardiologist's armamentarium.

CORONARY DOPPLER FLOW

Technical Aspects

Intracoronary Doppler flow velocity provides an objective, physiologic measurement of coronary blood flow.

The Doppler angioplasty guidewire (Volcano) is a flexible, steerable guidewire 175 cm long and 0.014 inch in diameter with a piezoelectric ultrasound transducer integrated into the

tip. The forward-directed ultrasound beam diverges in a 27° arc from the long axis (measured to the –6dB roundtrip points of the ultrasound beam pattern). A pulse repetition frequency of more than 40 kHz, pulse duration of +0.83 msec, and sampling delay of 6.5 msec are standard for clinical usage. The system is coupled to a real-time spectrum analyzer, a videocassette recorder, and a video page printer. The quadrature/Doppler audio signals are processed by the spectrum analyzer using on-line fast-Fourier transformation to provide a scrolling gray-scale spectral display. The frequency response of the system calculates approximately 90 spectra/sec. Simultaneous electrocardiographic and arterial pressure is also input to the video display.

Doppler guidewire velocity demonstrated excellent correlation with electromagnetic flow velocity and volumetric flow in straight and curved tube models as well as in *in vivo* testing using a circumflex canine coronary artery. The Doppler guidewire measures phasic flow velocity patterns and tracks linearly with flow rates in most small, straight coronary arteries.

The timing of the sending and receiving velocity allows the flow wire to measure blood flow velocities from moving red cells in a sample area 5 mm (×2 mm) from the tip of the wire, a distance far enough away for the blood velocity not to be affected by the wake of the wire. The returning signal is transmitted, in real time, to the display console and is seen on a gray-scale spectral scrolling display of all velocities of the red blood cells within the sample volume. The key parameters are derived from the automatically tracked peak blood velocities within the sample area, making the parameter values less position-sensitive. As long as the sample area accurately tracks the peak velocity in the center of the artery, the key parameters will remain positionally insensitive and reliable. The fundamentals and artifacts of flow velocity measurements have been described in detail elsewhere (see Suggested Readings).

Coronary Flow Velocity Signal Analysis

The velocity of red blood cells flowing past the ultrasound emitter/receiver on the end of a guidewire can be determined from the frequency shift, defined as the difference between the transmitted and returning frequency, where:

$$V = (F_1 - F_0) \times (C/2F_0) \times \text{Cos}\text{Ø},$$

where V = velocity of blood flow, F_0 = transmitting (transducer) frequency, F_1 = returning frequency, C = constant for the speed of sound in blood, Ø = angle of incidence. Volumetric flow is the product of the vessel area (cm^2) and the flow velocity (cm/sec), yielding a value in cm^3/sec.

Flow velocity data are printed on an integrated video page printer, which provides computerized parameters of intracoronary flow velocity, including maximal peak velocity (MPV) and mean or average peak velocity (APV) diastolic and systolic velocities, diastolic and systolic velocity integrals (DVI; obtained by planimetry of total area under the peak instantaneous velocity profile), mean total velocities, and the total velocity integral (Fig. 10-9). These automatic parameters were validated using a custom software program and manual tracing of the spectral peak Doppler velocity signal on digital computer.

The CFR, or the CFVR, is the ratio of the average peak hyperemic and resting coronary velocities. Other measurements, such as the diastolic–systolic flow ratio and the proximal–distal flow ratio can provide useful information regarding coronary stenosis severity, but CFVR is the most common index measured (Fig. 10-10).

Coronary flow velocity reserve measures the summed response of both the epicardial artery and the microcirculation. For this reason, a patient without epicardial disease but with abnormal microcirculatory function can have an abnormal CFVR, limiting use of CFVR for assessment of intermediate epicardial stenoses in patients with microvascular disease. For this reason, the relative CFVR index is a way of isolating the contribution of the epicardial disease to the abnormal CFVR. rCFVR is defined as the CFVR in the artery with epicardial disease divided by the CFVR in an adjacent vessel without epicardial disease. Because most microvascular dysfunction (e.g., due to diabetes or left ventricular hypertrophy) occurs diffusely throughout the myocardium, CFVR should be reduced equally in two coronary arteries. Any further reduction in CFVR in one artery over the other could then be attributed to epicardial disease. By definition, rCFVR cannot be applied in patients with three-vessel disease, and it requires placing a coronary wire down two separate arteries.

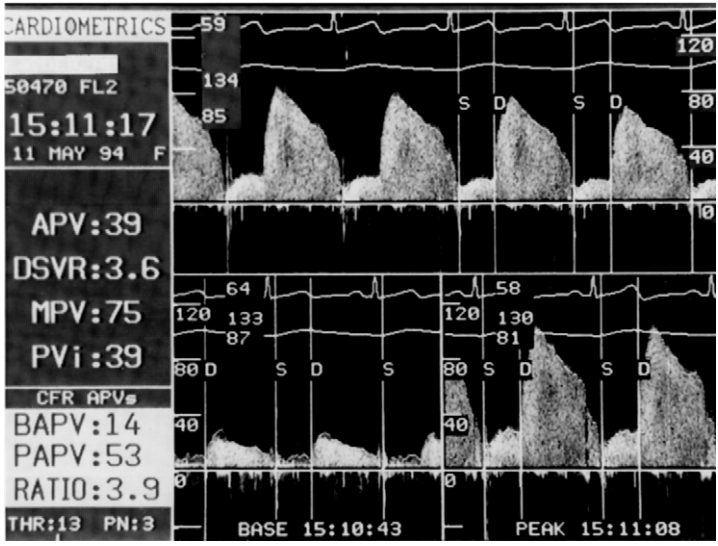
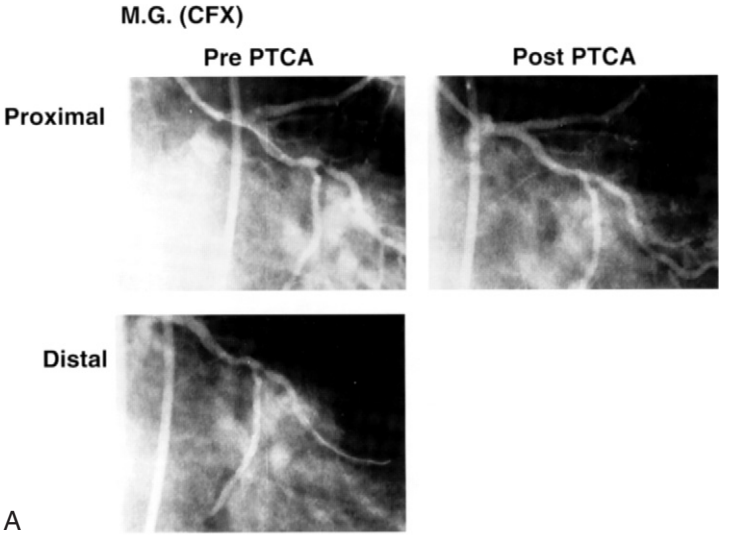


Fig. 10-9 Panel of flow velocity signals for measurement of coronary vasodilatory flow reserve. Flow panel was divided into upper and lower parts. The upper panel is a continuous display in real time of the flow spectra. The normal phasic pattern is seen shortly after hyperemia. The electrocardiogram (ECG) and aortic pressure is displayed on top of the flow signals. The numbers in the upper left corner box are the heart rate, systolic pressure, and diastolic pressure. The lower panel is divided into left and right for storage of baseline and hyperemic signals respectively. The codes at the left are the values for the flow parameters of the top panel (far right). APV, average peak velocity, DSVR, diastolic-systolic velocity ratio, MPV, maximal peak velocity, PVI, peak velocity interval, Ratio, coronary flow reserve; BAPV, PAPV, base and hyperemic APV respectively.

Technique of Coronary Doppler Measurements

Prior to passing the wire down the coronary vessel, heparin and intracoronary nitroglycerin (200 μg) are administered. Nitroglycerin avoids changes in epicardial lumen dimension during the measurements, which alter the flow velocity down the vessel. Using standard angioplasty equipment, the wire is introduced through the guide catheter to the coronary artery. The tip of the wire should be positioned at least 5–10 artery-diameter lengths distal to a stenosis. Attempts should be made to position the wire in the middle of a major vessel to maximize



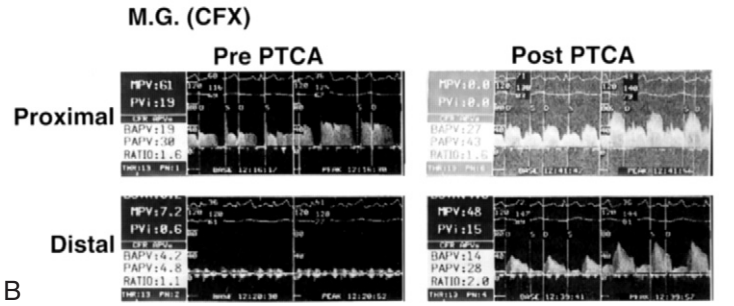


Fig. 10-10 **A,** Cineangiograms from an angioplasty of circumflex marginal branch with measurements of flow velocity. The position of the FloWire is shown proximal (*top left*) and distal (*bottom left*) to the stenosis. *Top right*, Angiographic result after percutaneous transluminal coronary angioplasty (PTCA). **B,** *Left panels,* Flow velocity recordings before PTCA. Proximal flow reserve is impaired, at 1.6, with abnormal phasic pattern. Flow is more disturbed in the poststenotic distal region, with nearly no flow. *Right panels,* After PTCA, both distal and proximal flow improve, with distal coronary flow velocity 2.0 and return of normal phasic pattern.

the velocity signal. The wire should not be moved between the resting velocity measurement and the hyperemic velocity measurement. Hyperemia is generally induced with intracoronary adenosine (>20 μg for the right coronary artery and >30 μg for the left) or intravenous adenosine (140 μg/kg/min) (see further discussions on dosing under the FFR section above). Changes in cardiovascular hemodynamics (heart rate, contractility, blood pressure) can impact the CFVR, a limitation when performing a followup or serial CFVR measurements.

Although earlier studies report a coronary vasodilatory reserve ratio of 3.5–5 in normal patients, lower values are more commonly observed in patients with chest pain and angiographically normal arteries (normal 2.7 ± 0.6). In transplanted hearts with angiographically normal arteries, coronary vasodilatory reserve ratios are usually higher (3.1 ± 0.6).

Flow Velocity Criteria

Normal Flow Criteria. A hierarchy of flow velocity findings describing normal flow characteristics is identified below.

- Poststenotic coronary vasodilatory reserve >2.0
- Diastolic–systolic velocity ratio (DSVR) >1.5
- Proximal–distal ratio <1.7.

Hemodynamically Significant Lesion Flow Criteria

Criteria of lesion significance for flow velocity finding distal to severe coronary stenoses are illustrated in Figure 10-10. The four criteria are:

- Decrease in mean velocity, usually <20 cm/sec.
- Mean proximal–distal flow velocity ratio >1.7
- Impaired phasic pattern of coronary flow (DSVR <1.5). A normal DSVR is usually over 1.8 for the left coronary artery. This value may vary normally among vessels, but a DSVR of less than 1.4 is common in severe lesions
- Impaired distal coronary hyperemia, coronary flow reserve <2.0.

Flow Velocity Criteria for Successful Angioplasty

Criteria of flow velocity for successful angioplasty are:

- Distal mean velocity is increased (usually >20–30 cm/sec)
- Phasic pattern of flow is normalized (DSVR >1.5)
- Coronary flow reserve is improved, <2.0. An inadequate lumen or microvascular impairment may yield lower

coronary flow reserve. Use a reference vessel coronary flow reserve to compare.

The comparison of a post-stenotic flow reserve of more than 2.0 with myocardial perfusion stress imaging has demonstrated a high correlation with normal perfusion scintigraphy. High sensitivity, specificity, and predictive accuracy are reported for both perfusion sestamibi and thallium-201 imaging with distal CFR.

Methodological Difficulties

Doppler coronary velocity only measures relative changes in velocity. To measure absolute blood flow, the following assumptions must be made:

- The cross-sectional area of the vessel being studied remains fixed during hyperemia
- The velocity profile across the vessel is not distorted by arterial disease
- The angle between the crystal and sample volume remains constant and less than 30° from the horizontal flow stream.

Assessing Intermediate Coronary Lesions

In patients with truly normal epicardial and microcirculatory coronary systems, maximal flow (or velocity) should increase three- to fivefold and the CFR (or CFVR) should be 3–5. Unfortunately, there is no inherent normal cutoff applicable to all patients. In general, investigators have found that a CFVR below 2.0 is abnormal and correlates with ischemia on noninvasive stress testing. Initial studies comparing CFVR with noninvasive stress testing demonstrated a sensitivity and specificity of CFVR in the low 90% range. Subsequent larger followup studies have shown a diagnostic accuracy in the low 80% range. Presumably the presence of occult microcirculatory disease in some patients in the larger studies is responsible for the slight decrease in diagnostic characteristics (Table 10-3).

The clinical outcome of the prospective deferment of angioplasty of intermediate stenoses based on normal flow criteria is excellent. Of 88 patients with 100 lesions with 40–70% diameter stenosis over a mean of 10 ± 5 months, the cardiac event rates were 4, 6, 0, and 2 patients requiring repeat

Table 10-3

Comparison of Stress (Ischemia) Testing and Directly Measured Coronary Blood Flow Physiology

Author	*n*	Ischemic Test	Physiologic Threshold	Sens	Spec	PV^+	PV^-	Accuracy
Poststenotic CVR/rCVR								
Miller	33	Adeno/dipy MIBI	<2.0	82	100	100	77	89
Joye	30	Exercise thallium	<2.0	94	95	94	95	94
Deychak	17	Exercise thallium	<1.8	94	100	91	95	
Heller	100	Exercise thallium	<1.8	89	92	96	89	92
Danzi	30	Dipy echo	<2.0	91	84	–	–	87
Schulman	35	Exercise ECC	<2.0	95	71	–	86	
Donahue	50	Exercise/pharm thallium	<2.0	98	75	88	88	–
Duffy	43	Stress echo	<2.0	80	93	–	–	88
			rCVR <0.75	100	76	–	–	81
Chamuleau	127	Dipy MIBI	CVR <0.20	–	–	–	–	69
			rCVR <0.75	–	–	–	–	75
El Shafei	53	Exercise/pharm thallium	CVR <0.20,	71	83	81	74	–
			rCVR <0.75	63	88	83	70	–
FFR								
Pijls	45	Four test standard*	<0.75	88	100	100	88	93
De Bruyne	60	Exercise ECC	<0.72	100	87	–	–	–
Bartunek	37	Dobu/exercise echo	<0.58	95	90	–	–	–
Chamuleau	127	Dipy MIBI	<0.75	–	–	–	–	75
Caymaz	30	Exercise thallium	<0.75	–	–	91	100	
			<0.75	90	100	–	–	95

* Four tests were electrocardiography, echocardiography, pacing, nuclear stress tests.

Adeno/dipy MIBI, adenosine or dipyridamole sestamibi scan; CFR, coronary vasodilatory reserve; dobu, dobutamine; ECG, electrocardiography; echo, echocardiography; pharm, pharmacologic; PV^+/PV^-, predictive value positive/negative; Sens, sensitivity; Spec, specificity.

angioplasty, requiring coronary artery bypass surgery, experiencing myocardial infarction, or dying, respectively. Of the 10 patients having angioplasty or coronary artery bypass surgery, only 6 had lesion progression, with 4 being new stenoses requiring intervention for nontarget arteries. In a comparative angioplasty patient cohort undergoing similar Doppler flow measurements over the same period, 26% of the patients required angioplasty or coronary artery bypass surgery, compared to 12% of the deferred group. PCI should be performed based on objective evidence of flow limitation responsible for the clinical syndrome.

Relative CFVR does have an absolute normal value of 1.0. The cutoff value used to define an ischemia-producing lesion varies between 0.80 and 0.65; it has been less well studied compared to CFVR. One study using a cutoff value of 0.65 found that rCFVR correlates better with nuclear perfusion imaging than does CFVR.

Doppler measurements, including the translesional velocity ratio, but in particular the CFVR do not only correlate with noninvasive tests for ischemia, but also identify lesions in which it is safe to defer PCI. For example, in a study of 70 patients with chest pain and or ischemia on a noninvasive stress test who were referred for PCI, 22 patients had a CFVR of more than 2.0 and PCI was deferred. The patients were followed for over 1 year and the major adverse cardiac event rate was significantly lower in the patients in whom PCI was deferred, 9% vs 33%.

Assessing Percutaneous Coronary Interventions

The Doppler wire has been studied extensively in the setting of assessing optimal angioplasty and PCI. For example, the combination of a postangioplasty CFVR of more than 2.5 and a residual diameter stenosis of less than 35% identified lesions with a significantly lower 6-month restenosis rate, 16% vs 41%. Measurement of CFVR after angioplasty as a means of determining the need for provisional stenting has been investigated in three large randomized trials. All three studies found that the group randomized to provisional stenting, only if the CFVR and angiographic result were deemed inadequate, had short- and long-term outcomes similar to those of the group that underwent routine stenting. However, with advances in stent technology limiting stent-related complications

and the advent of drug-eluting stents limiting in-stent restenosis, coupled with operator preferences, this approach may be limited.

Assessing Collateral Flow

When measuring coronary flow velocity with a Doppler flow wire in a stenosed coronary artery without collateral circulation, balloon occlusion of the stenosed vessel results in loss of the Doppler flow signal. If, however, "bridging" or ipsilateral collaterals are present, then balloon occlusion may result in a persistent antegrade flow signal. Alternatively, contralateral collaterals will manifest as a retrograde flow signal during balloon occlusion. In this manner, the Doppler wire can be used to assess the presence of collateral circulation during PCI.

In order to quantify the presence and degree of collaterals, a Doppler collateral flow index (CFI) has been described. CFI is defined as the amount of flow via collaterals to a vascular region, divided by the amount of flow to the same region via the normally patent vessel. It is determined by summing the integral of systolic and diastolic flow velocities during balloon occlusion. In the case of temporally shifted bidirectional flow velocity signals, the antegrade and retrograde velocity integrals are added. The total velocity integral during balloon occlusion is then divided by the velocity integral after successful PCI, in order to calculate the CFI. A Doppler CFI of more than 0.30 has been shown to accurately predict collateral circulation adequate enough to prevent myocardial ischemia during PCI. Moreover, the Doppler CFI is a more sensitive determinant of collateral flow than is angiographically visible collateral circulation. In another study, patients undergoing PCI who had a Doppler CFI of 0.25 or more had a fourfold decrease in the major adverse cardiac event rate at approximately 2 years compared to those with a CFI below 0.25. The obvious limitation of this technique is that it requires performance of PCI (Fig. 10-11).

Safety of Intracoronary Sensor-Wire Measurements

Qian *et al.* examined the safety of intracoronary Doppler wire measurements in 906 patients. Fifteen patients (1.7%) had severe transient bradycardia after intracoronary adenosine, 14 in the right coronary artery and one in the left coronary artery. Nine patients (1%) had coronary spasm during passage of the Doppler guide wire (5 in the right coronary and 4 in the left

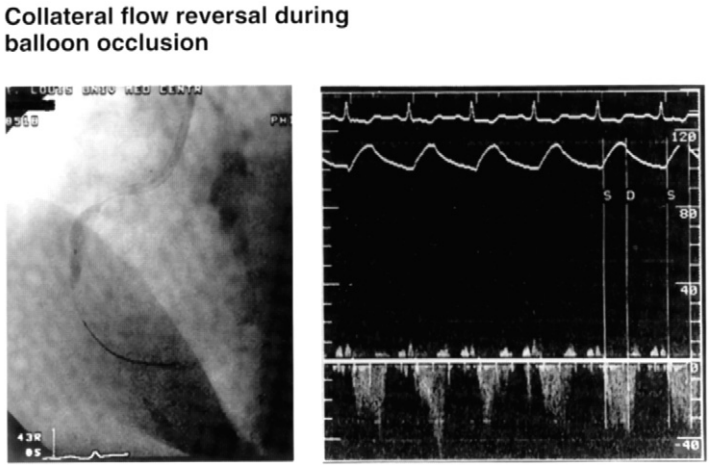


Fig. 10-11 Collateral flow velocity reversal can be observed during balloon occlusion. *Left panel,* Percutaneous transluminal coronary angioplasty (PTCA) balloon occluding artery with FloWire in distal segment. *Right panel,* Inverted flow velocity signal indicates retrograde flow coming towards the tip of the FloWire. These signals disappear after successful PTCA.

Table 10-4

Characteristics of Coronary Lesion Assessment Modalities

	CFVR	rCFVR	FFR	IVUS
Normal value	>2	1.0	1.0	Range
Epicardial-artery-specific	No	Yes	Yes	Yes
Assesses microcirculation	Yes	No	No	No
Ischemic threshold	<2	<0.65–0.80	<0.75	MLA <3.0–4.0 mm^2
Optimal PCI threshold stenosis >90%	>2	Unknown	>0.90	MSA >7.0 mm^2 % area
Assesses collaterals	Yes	No	Yes	No
Use in multivessel disease	Yes	No	Yes	Yes
Hemodynamic independence	No	Yes	Yes	Yes

% area stenosis, signifies (MSA / average reference lumen area) × 100%.
CVR, coronary vasodilatory reserve; rCVR, relative CVR; FFR, fraction flow reserve; IVUS, intravascular ultrasound; MLA, minimum lumen area; MSA, minimum stent area.

Table 10-5

Recommendations for Intracoronary Physiologic Measurements

	Level of Evidence
Class IIA	
1. Assessment of the physiologic effects of intermediate coronary stenosis (30–70% luminal narrowing) in patients with anginal symptoms. Coronary pressure of Doppler velocimetry may also be useful as an alternative to performing noninvasive functional testing (e.g., when the functional study is absent or ambiguous) to determine whether an intervention is warranted	B
Class IIb	
1. Evaluation of the success of percutaneous coronary revascularization in restoring flow reserve and to predict the risk of restenosis	C
2. Evaluation of patients with anginal symptoms without an apparent angiographic culprit lesion	C
Class III	
1. Routine assessment of the severity of angiographic disease in patients with a positive, unequivocal noninvasive function study	C

(From Smith SC Jr, Dove JT, Jacobs AK, *et al.* ACC/AHA guidelines of percutaneous coronary interventions (revision of the 1993 PTCA guidelines)—executive summary. A report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (committee to revise the 1993 guidelines for percutaneous transluminal coronary angioplasty). *J Am Coll Cardiol* 2001;37:2215–2239.)

anterior descending). Two patients (0.2%) had ventricular fibrillation during the procedure. Hypotension with bradycardia and ventricular asystole occurred in one patient. Transplant recipients had more of these complications than either diagnostic or interventional procedures. All complications could be managed medically. These data support the safety of clinical practice using sensor wire measurements with intracoronary adenosine.

A summary of the characteristics of coronary vasodilatory reserve (CVR), relative CVR (rCVR), FFR and IVUS is provided in Table 10-4.

Recommendations for intracoronary Doppler ultrasound and coronary pressure measurements are shown in Table 10-5.

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11

PERIPHERAL VASCULAR INTERVENTION

Jose Antonio Silva and Christopher J. White

Atherosclerosis is a systemic disease that often affects several vascular territories. The age-adjusted prevalence of peripheral atherosclerotic disease is 12%; however, in patients with established coronary artery disease this prevalence is substantially higher. Although relatively less attention has been given to this disease when it affects the peripheral circulation compared to coronary atherosclerosis, peripheral arterial-occlusive disease has a natural history, progression pattern, and susceptibility for developing vulnerable and complex plaques comparable to that of the coronary circulation. Furthermore, peripheral atherosclerotic disease is known to be a strong marker for cardiovascular events and mortality.

Despite this, peripheral atherosclerotic disease remains poorly recognized. Results of the Partners Program (Peripheral Arterial Disease Awareness, Risk and Treatment: New Resources for Survival), a US national survey of almost 7000 patients seen in 320 primary care clinics, has shown that only 45% of the patients with peripheral vascular disease had been diagnosed with this condition prior to the Partners Program. Its detection and treatment is of utmost importance because it may have a profound impact on health-related quality of life.

Percutaneous, catheter-based revascularization techniques have profoundly changed the management of obstructive atherosclerotic disease in the coronary vessels and in the peripheral circulation and are at present accepted as alternatives to surgery in selected patients. Endovascular therapy offers several distinct advantages over surgical revascularization.

It may be performed under local anesthesia, enabling the treatment of patients who are at high risk for general anesthesia. The morbidity and mortality from endovascular therapy is very low when compared to surgical revascularization. Problems secondary to angioplasty are generally related to vascular access. Following endovascular therapy, patients are usually ambulatory on the day of treatment and, unlike vascular surgery patients, can often return to normal activity within 24–48 hours of an uncomplicated procedure. Finally, endovascular therapies may be repeated if necessary, generally without increased difficulty or increased patient risk compared to the first procedure, and prior angioplasty does not preclude surgery if required at a later date.

VASCULAR ACCESS

The first step to ensure a successful procedure is to plan the appropriate vascular access (Table 11-1). The majority of peripheral vascular interventions can be performed from several access sites (i.e., brachial, ipsilateral femoral, contralateral femoral)—the choice is often left to the preference of the physician. However, some cases require a specific access in order to achieve a successful result (Table 11-2). Consequently, familiarity with a variety of vascular access sites and techniques is one of the most important components of the basic skills in peripheral vascular intervention.

Retrograde Common Femoral Access

The retrograde approach to the common femoral artery (CFA) is the most frequently used vascular access. The inguinal skin crease is highly variable in relation to the CFA and may be

Table 11-1

Arterial Access for Different Vascular Territories

Vascular Access	Artery(ies) to Revascularize
Retrograde CFA	Aortic arch vessels, renal, and mesenteric arteries
Contralateral CFA	Contralateral iliacs, CFA, PFA, SFA, popliteal arteries
Antegrade CFA	Mid to distal femoral, popliteal, and infrapopliteal arteries
Brachial/radial artery	Renal (caudal takeoff), mesenteric, and iliac arteries
Retrograde popliteal artery	SFA and iliac artery

CFA, common femoral artery; PFA, profunda femoris artery; SFA, superficial femoral artery.

Table 11-2

Most Useful Angiographic Views for Different Vascular Territories

Artery or Vascular Territory	Angiographic View
Aortic arch	30–60° LAO
Brachiocephalic vessels (origin)	30–60° LAO
Subclavian	AP, ipsilateral oblique with caudal angulation
Vertebral origin	AP, ipsilateral oblique with cranial angulation
Carotid extracranial	Lateral, AP, ipsilateral oblique
Renal arteries (origin)	AP, 5–10° ipsilateral oblique
Mesenteric arteries (origin)	Lateral
Iliac artery	Contralateral 20° oblique and 20° caudal
CFA, SFA and PFA arteries	Ipsilateral 30–60° oblique
Femoropopliteal	AP
Infrapopliteal trifurcation and runoff	AP

AP, anteroposterior; CFA, common femoral artery; LAO, left anterior oblique; PFA, profunda femoris artery; SFA, superficial femoral artery.

caudal to the CFA bifurcation in up to 75% of the patients. Identifying the femoral head with fluoroscopy is helpful (Fig. 11-1), because it has been shown that puncturing the CFA at this level ensures access below the inguinal ligament and above the level of the CFA bifurcation in most patients. Retrograde CFA access is the most commonly used vascular access site for performing angiography and intervention of the aortic arch and the subclavian, carotid, vertebral, renal, mesenteric, ipsilateral iliac and contralateral lower extremity arteries.

Antegrade Common Femoral Access

Antegrade CFA access is useful for performing intervention of the mid to distal superficial femoral (SFA), popliteal, and infrapopliteal (anterior tibial, posterior tibial and peroneal) arteries. It is more technically demanding than retrograde CFA access, particularly in obese patients. Antegrade CFA access may carry a higher complication rate than retrograde CFA access, such as retroperitoneal hematoma if the access is "too

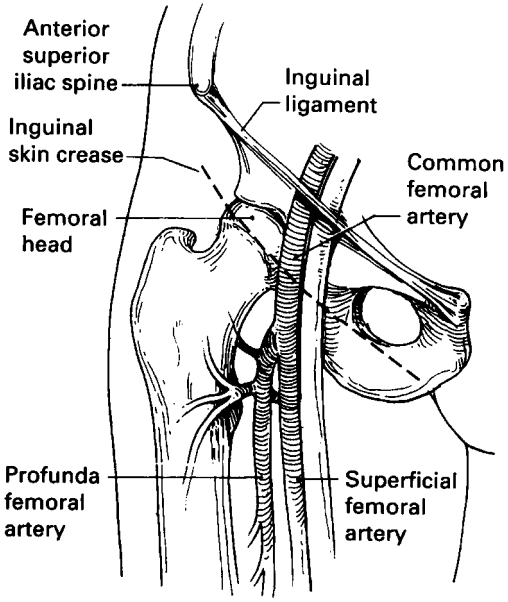


Fig. 11-1 Schematic drawing of the right femoral area.

high," i.e., cephalad to the femoral head or hematoma, or pseudoaneurysm and arteriovenous fistulas if the access is too caudal to the CFA.

When entering the CFA in an antegrade fashion, it is helpful to identify the femoral head under fluoroscopy. Depending on the amount of subcutaneous tissue, a skin incision is made 1–2 cm cephalad to the middle of the femoral head. After the CFA pulse is located at the middle of the femoral head, the percutaneous needle is introduced through the skin incision and directed obliquely and caudally toward the center of the femoral head. Once the CFA has been entered, a steerable guidewire (Wholey, Malinckrodt, St Louis, MO) is advanced under fluoroscopic guidance toward the superficial femoral artery (SFA) which runs medial to the profunda femoris artery (PFA). It is important to emphasize that, at their origin, the SFA and PFA overlap in the antero-posterior fluoroscopic view. In order to separate them a lateral oblique view (20–40°) is used.

Relative contraindications for the use of this vascular access site include atherosclerotic disease of the CFA or proximal SFA, and extreme obesity.

Brachial Artery Access

This is an important alternative vascular access for percutaneous intervention of the iliac and femoral arteries. This vascular access is also preferred for renal, celiac, and mesenteric artery intervention when these vessels have cephalic orientation from the aorta.

Brachial access is performed using the Seldinger technique at the level of the antecubital fossa. The right or left brachial artery may be chosen. Some operators prefer the left brachial artery, in order to have more direct access to the descending aorta and to avoid potential embolization into the carotid arteries. Useable catheter length may become an issue for distal iliac or femoral artery interventions; often extra-long catheters (150 cm) are required.

Contralateral Lower Extremity Artery Access

Retrograde CFA access permits selective angiography and intervention of the contralateral pelvic and lower extremity vessels. After gaining retrograde access to the CFA, the contralateral iliofemoral system is reached by placing a diagnostic catheter with an acute bend at the tip (usually an internal mammary artery or Simmons catheter) at the aortic bifurcation. The catheter is manipulated so that the tip "engages" the ostium of the contralateral common iliac artery. A stiff, angled Glidewire™ (Terumo Medical Corp, Somerset, NJ) is then carefully steered to the femoral artery and the diagnostic catheter is advanced over the Glidewire™ into the CFA. The Glidewire™ is then exchanged through the diagnostic catheter for a stiff guidewire (Amplatz extra-stiff, Cook, Bloomington, IN) which is advanced into the distal femoral artery. The diagnostic catheter is then removed, leaving the extra-stiff wire in place. A crossover sheath (6–8F) may then be advanced over the stiff guidewire and positioned in the contralateral CFA. This allows contrast injection during lesion dilation and backup support for crossing lesions.

In patients with an acute angle between the origin of the common iliac arteries or in patients after aortofemoral bypass grafts, this access may be difficult (Fig. 11-2).

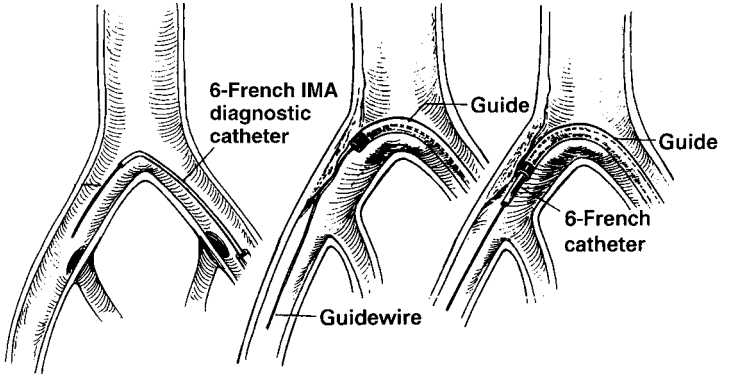


Fig. 11-2 Schematic drawing of contralateral femoral access. IMA, internal mammary artery.

Retrograde Popliteal Arterial Access

This is an infrequently used vascular access, but it is useful when trying to cross an occluded SFA, after antegrade attempts to cross the occlusion have failed. It is important to be aware of the anatomical relationship between the popliteal artery and vein. At the level of the joint space, the artery courses anterior to the vein, whereas, at approximately 6 cm cephalad to the joint space, the artery is medial to the vein (Fig. 11-3).

For popliteal arterial puncture the vessel should be free of significant disease and larger than 4 mm in diameter. Prior angiography and/or color-flow duplex imaging may provide useful information regarding puncture of this vessel.

The first step is to gain contralateral CFA access to provide contrast injections to help visualize the target popliteal artery. The CFA sheath is sutured to secure it to the skin, a sterile dressing is applied, and the patient is turned to the prone position. The skin is infiltrated with local anesthetic 3–4 cm cephalad to the popliteal joint space. An assistant performs a hand-injection of contrast through the contralateral CFA sheath, which allows fluoroscopic visualization of the popliteal artery. The puncture needle is directed obliquely from medial to lateral while visualizing the popliteal artery on fluoroscopy, so that the artery is entered approximately 6 cm above the joint space. A 0.035 inch floppy guidewire is then

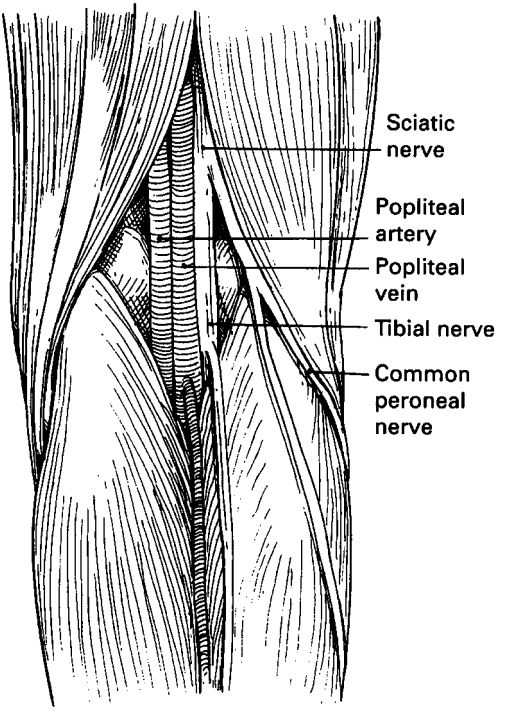


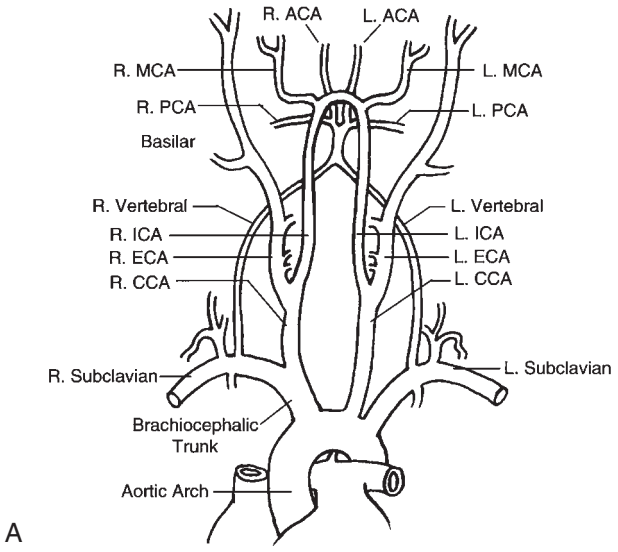
Fig. 11-3 Schematic drawing of the popliteal fossa.

advanced into the popliteal artery, and a 4–6 French arterial sheath is then placed over the wire.

BRACHIOCEPHALIC INTERVENTION

The innominate, left common carotid, and left subclavian arteries originate in the transverse thoracic aorta (Fig. 11-4). The innominate artery divides into the right common carotid artery and the right subclavian artery. The left common carotid and left subclavian artery usually originate separately from the aortic arch; however, a common variant, a "bovine arch" in which the left common carotid artery arises from the innominate, is present in 10% of patients.

The common carotid arteries bifurcate into the external and internal carotid arteries at the level of the fourth cervical vertebra. The extracranial internal carotid artery has no



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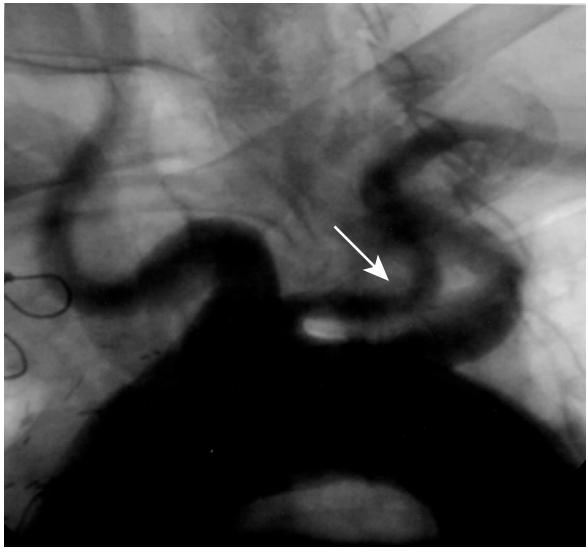


B

Fig. 11-4 **A,** Normal aortic arch vessels. ACA, anterior carotid artery; CCA, common carotid artery; ECA, external carotid artery; ICA, internal carotid artery, MCA, middle cerebral artery; PCA, posterior cerebral artery. **B,** Bovine arch angiogram demonstrating the left common carotid arising from the innominate artery (*arrow*).

branches. Upon entering the skull, the internal carotid artery takes a tortuous path known as the carotid siphon.

Subclavian Artery Intervention

The prevalence of brachiocephalic or subclavian artery stenosis ranges between 12% and 15% of patients undergoing angiography for cerebrovascular symptoms. Stenoses are most commonly located in the proximal segment of the vessel, prior to the origin of the vertebral and internal mammary artery. The left subclavian artery is affected three to four times more frequently than the right, which is important in protecting patency of the commonly used internal mammary artery bypass graft.

Although atherosclerotic disease is the most common cause of subclavian artery stenosis, conditions such as Takayasu's arteritis, fibromuscular dysplasia, giant cell arteritis, radiation-induced occlusive disease, and the thoracic outlet syndrome may cause significant stenosis.

The clinical manifestations of subclavian artery stenosis include a blood pressure difference between the arms, the subclavian steal syndrome, and arm claudication. The subclavian steal syndrome occurs as a result of flow reversal in the vertebral artery, leading to symptoms of vertebrobasilar insufficiency with upper extremity activity. In the coronary–subclavian steal syndrome, there is reversal of flow in an internal mammary bypass graft as a result of a proximal subclavian stenosis, which may cause myocardial ischemia.

Procedure. Patients are pretreated with aspirin 325 mg daily. The most commonly used vascular access is retrograde CFA access. Occasionally, the ipsilateral brachial access may be necessary, particularly when the subclavian artery is totally occluded at or close to its origin from the aorta. Heparin anticoagulation is administered after vascular access has been obtained.

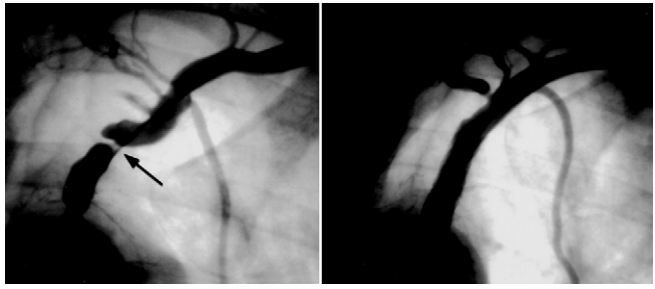
After CFA access is obtained, the area of stenosis and runoff vessels are imaged with diagnostic angiography. A steerable angioplasty guidewire may be advanced across the lesion and the diagnostic catheter used to measure a translesional pressure gradient. The guidewire is left across the lesion and the diagnostic catheter is exchanged for either a 6 French sheath or an 8 French guiding catheter (multipurpose or

hockey-stick) positioned proximal to the subclavian artery stenosis. Using the diagnostic catheter or an external scaling object, we measure the reference vessel diameter (RVD) using online quantitative angiography.

The lesion is predilated with a balloon sized 1:1 with the RVD, using the lowest pressure that will fully expand the balloon. The angiographic results are assessed with hand injection of contrast through the guiding catheter or sheath. Provisional stenting, stent placement for suboptimal (≥30% residual stenosis or translesional gradient ≥5 mm Hg) results, is advocated by some authors, although most experts believe that primary stenting (stent placement regardless of the balloon dilation result) is appropriate for subclavian lesions to minimize restenosis. Balloon-expandable stents are preferred when precise stent placement is required (aorto-ostial lesion, or when trying to avoid "jailing" a branch artery) and self-expanding stents are used when precision is not a critical factor and the vessel tapers in diameter.

When primary stent placement with a balloon expandable stent is planned, the sheath or guiding catheter is carefully advanced across the lesion while the predilation balloon is deflating. The balloon catheter is removed. A balloon-expandable stent is positioned at the lesion site, while the sheath or the guiding catheter is still across it. This maneuver helps to prevent the undeployed stent from catching on the lesion, with the risk of embolization or dislodgment of the stent. When the stent is at the lesion site (but still within the sheath or guiding catheter), the sheath or guiding catheter is withdrawn and contrast is injected to confirm the correct position of the stent. The stent is then deployed with balloon inflation to ensure adequate stent expansion and apposition of the struts to the vessel wall. If doubts regarding adequate stent expansion remain after post-stent angiography, the operator may repeat balloon inflation using a higher inflation pressure, or use a bigger balloon.

Clinical Results. Percutaneous intervention has largely replaced surgical revascularization for treating symptomatic subclavian artery stenosis (Fig. 11-5). Several surgical techniques have been used, including carotid–subclavian bypass and axillo-axillary bypass. However all of them carry significant



A B

Fig. 11-5 **A,** Baseline angiogram of discrete (arrow) left subclavian artery stenosis. **B,** Angiogram following balloon-expandable stent placement with resolution of stenosis and no pressure gradient. Note that antegrade vertebral flow is now present.

morbidity and mortality. Hadjipetrou *et al.* recently reviewed the outcomes of 52 surgical studies with 2496 patients. The technical success was 96% (range 75–100%) and the complication rate was 16 ± 11% (range 0–43%), with a mortality rate of 2 ± 2% (range 0–11%) and a stroke rate of 3 ± 4% (range 0–14%). At a mean followup of 51 ± 25 months, the recurrence of symptoms occurred in 16 ± 14%.

Endovascular procedures can be carried out with a high technical success, and are preferred over surgery. Hadjipetrou and coworkers reported a series of patients treated with stents. Of 108 patients treated with these devices, technical success was obtained in 97 ± 4%. Adverse events were reported in 6 ± 5%. In a multicenter registry involving eight centers, stenting of the subclavian artery was successful in 98.5% of patients and a transient ischemic attack (TIA) occurred in only one patient (0.5%).

Vertebral Artery Intervention

It has been difficult to correlate the severity of stenoses affecting the vertebrobasilar circulation and symptoms of vertebrobasilar insufficiency. In some patients the stenosis may be severe, yet the patient may experience minimal or no symptoms. On the other hand, some patients with moderate vertebrobasilar arterial disease may suffer disabling symptoms. The lack of correlation between anatomical stenoses and clinical manifestations is due

to the fact that the posterior fossa receives blood supply from the contralateral vertebral artery and also receives collateral blood flow from the carotid artery system through the posterior communicating arteries. In addition, stenosis of the subclavian artery may also lead to symptoms of vertebrobasilar insufficiency.

The assessment of a patient with a significant stenosis of a vertebral artery should include angiography of the contralateral vertebral artery and an angiographic assessment of the contribution to flow from the circle of Willis. We also recommend a consultation with a neurologist to help select appropriate candidates for intervention.

The indications for revascularization of the vertebral arteries include symptoms of vertebrobasilar insufficiency (dizziness, visual disturbances, and confusion or coma). When the diagnosis of vertebrobasilar insufficiency has been established, revascularization should be considered since these patients carry a significant risk for posterior circulation stroke. Atherosclerotic disease of the vertebral arteries most commonly affects the ostium or the very proximal portion of this vessel, making percutaneous revascularization an attractive alternative to surgery.

Procedure. The vertebral artery is the first branch of the subclavian artery, arising from the superior and posterior surface of the vessel. It angles backward to the transverse process of the sixth cervical vertebra and runs superiorly through the foramina in the transverse processes of the upper cervical vertebrae to the inferior surface of the skull. It then courses posteromedially through the foramen magnum. Just after entering the skull, it gives rise to the posterior inferior cerebellar artery (PICA) and subsequently joins the contralateral vertebral artery to form the basilar artery (Fig. 11-6).

Femoral access is the preferred access site for percutaneous revascularization of the vertebral arteries, although occasionally the ipsilateral brachial arterial access may be used. All patients are pretreated with aspirin (325 mg daily) and clopidogrel (75 mg daily) or ticlopidine (250 mg twice a day). After access is obtained, heparin anticoagulation is obtained.

The target vertebral artery is engaged with a diagnostic catheter (4, 5, or 6 French Judkins right-4, Berenstein, Vitek, internal mammary artery, or multipurpose diagnostic coronary catheter). Quantitative angiography is used to measure the

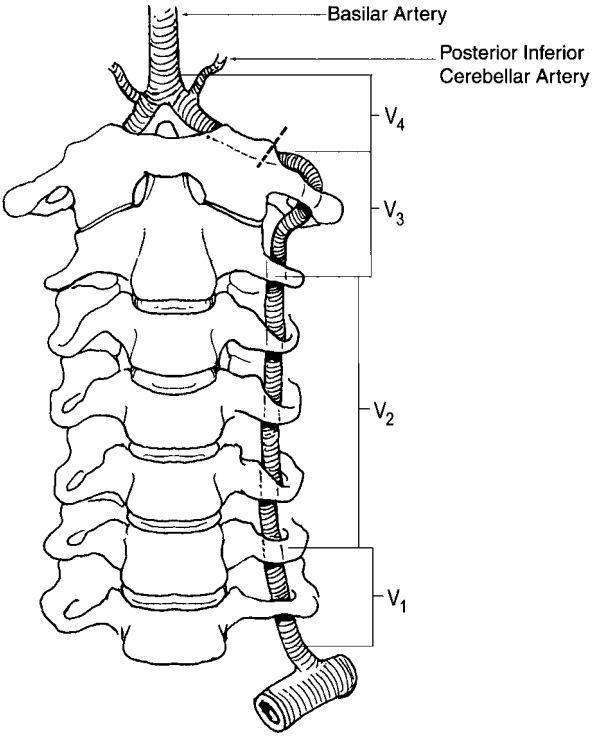


Fig. 11-6 Schematic drawing of the vertebral artery, showing branches and vessel segments.

reference vessel diameter (RVD) of the vertebral artery. A steerable 0.014 inch angioplasty guidewire is used to cross the stenosis. The diagnostic catheter is then exchanged over the guidewire for a guiding catheter, placing it just proximal to the ostium of the vertebral artery. The stenosis is then predilated using a balloon with a diameter ratio of 1:1 or less to the RVD, using the minimum inflation pressure necessary to completely expand the balloon. For proximal or ostial lesions, a balloon-expandable stent with a diameter that matches the RVD is chosen. After stent deployment, angiography of the stent lesion is performed, to include the intracranial posterior circulation (Fig. 11-7).

Clinical Results. Before the development of percutaneous catheter-based revascularization techniques, surgery was the only

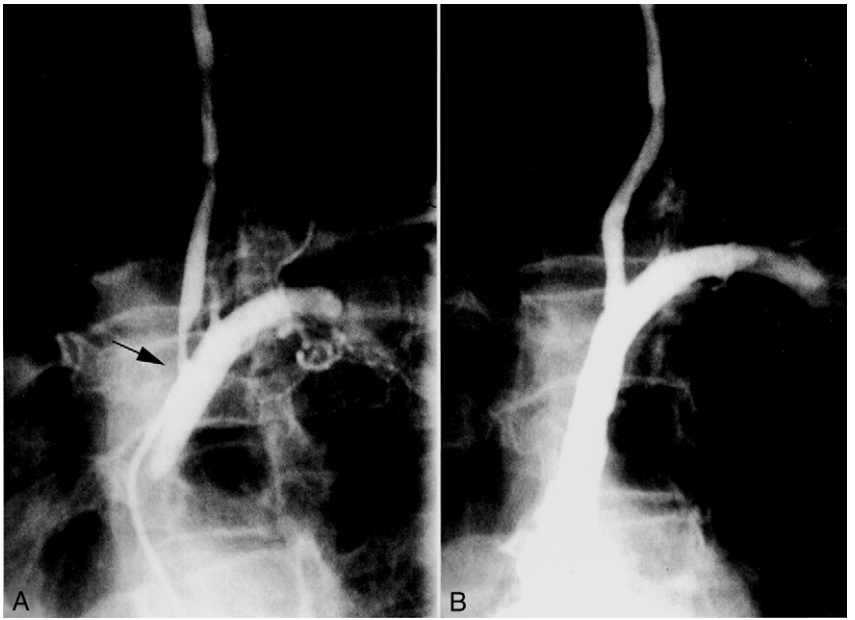


Fig. 11-7 **A,** Baseline angiogram of left vertebral ostial stenosis (*arrow*). **B,** Angiogram following balloon-expandable stent placement.

treatment option to revascularize symptomatic patients with vertebral artery stenosis. Surgical options included transplantation of the vertebral arteries onto the carotid artery, or a bypass graft from the subclavian to the vertebral artery. However, the surgical procedures were associated with significant morbidity. In one series of 174 patients undergoing vertebral artery reconstruction, complications included recurrent laryngeal palsy in 2%, Horner's syndrome in 15%, lymphocele in 4%, chylothorax in 4%, and immediate thrombosis in 0.5%.

Balloon angioplasty with or without stenting has been shown to be an attractive and feasible alternative to surgery. As is the case for other aorto-ostial lesions (saphenous vein grafts, right coronary arteries, or renal arteries), a strategy of primary stenting should be considered for stenoses of the ostium, since stents scaffold the lesions and minimize the elastic recoil. Lesions located more distally may be treated with balloon angioplasty with provisional stenting, depending upon the angiographic results and the tortuosity of the vessel. Distal vertebral artery lesions are more difficult to access and more prone to dissection.

In a prospective study from our institution, 38 vertebral arteries of 32 patients (87% ostial, 3% in the V1 segment, and 10% in the V2 segment of the vertebral artery) were treated with 42 stents. The indications for the procedure were: diplopia ($n = 4$), blurred vision ($n = 4$), dizziness ($n = 23$), TIA of the vertebrobasilar system ($n = 4$), gait disturbance ($n = 1$), drop attack ($n = 1$), headaches ($n = 2$), and critical stenosis ($n = 1$). Procedural success (<20% residual diameter stenosis and freedom from in-hospital TIA, stroke, or death) was achieved in 100%. One patient (3%) had a TIA 1 hour post-procedure, which resolved within 5 min. Repeated angiography in this patient revealed a widely patent stent with a patent posterior cerebral circulation. At a mean followup of 10.6 months, all patients were alive. One patient (3%) had recurrent symptoms at 3.5 months due to in-stent restenosis, which was successfully treated with balloon angioplasty. The other 31 patients remained asymptomatic.

Patients with symptomatic vertebral artery stenosis who have failed medical treatment should be considered for percutaneous revascularization. Percutaneous revascularization procedures with balloon angioplasty and stent placement offer

a less invasive alternative to surgery with durable results, excellent clinical success, and low complication rates.

Carotid Artery Intervention

In the USA alone, there are approximately 500,000 cases of stroke a year, of which 150,000 are fatal, making stroke the third leading cause of death. Cerebral infarction is the most common cause of stroke, with obstructive extracranial carotid artery disease ranking second. Randomized controlled trials have clearly demonstrated that carotid endarterectomy is superior to medical treatment for preventing strokes in selected patients with extracranial carotid artery atherosclerosis.

Evidence is mounting to support percutaneous intervention of carotid artery disease as a viable alternative to carotid endarterectomy in selected patients, and it appears to be the treatment of choice in patients who are high-risk for surgery (Table 11-3, Box 11-1).

Procedure. It is important to emphasize that carotid stent placement should ideally be performed by a multidisciplinary team with the necessary skills to provide optimal patient care. The team should include an experienced vascular interventionist to perform the procedure and a neurologist or vascular internist to screen patients for appropriate indications and to independently assess outcomes. In addition, it is essential that the capability exists to perform an emergency neurovascular rescue procedure.

Antiplatelet therapy preceding carotid stenting is of the utmost importance. Aspirin 325 mg daily is started at least

Table 11-3

Thirty-Day Complications of Carotid Stent Placement

Author	Arteries (*n*)	All Stroke & Death (%)	Major Stroke & Death	Death (%)
Yadav	126	7.9	2.4	0.8
Wholey	2048	5.8	2.7	1.4
Henry	174	2.8	1.7	0
Waigand	53	1.9	1.9	1.9
White	481	2.8	2.8	0.6
Total	3050	4.9	2.5	1.1

Box 11-1

SAPPHIRE Trial: Patients at High-Risk for Surgical Revascularization (see SAPPHIRE Investigators)

- Older than 80 years of age
- Presence of congestive heart failure NYHA III/IV, left ventricular ejection fraction ≤30%
- Open heart surgery needed within 6 weeks
- Recent myocardial infarction (>24 h, <4 weeks)
- Unstable angina (CCS III/IV)
- Severe pulmonary disease
- Contralateral carotid occlusion
- Severe tandem carotid lesions
- Contralateral laryngeal nerve palsy
- Restenosis after previous endarterectomy
- Previous radiation therapy in the neck
- Previous radical neck surgery
- High cervical or low intrathoracic lesion location

CCS, Canadian Cardiovascular Society; NYHA, New York Heart Association.

24 hours prior to the procedure. An ADP platelet-inhibitor, such as clopidogrel (75 mg daily) is begun 3–5 days before carotid stenting. If necessary, patients may be pretreated with aspirin 325 mg and a loading dose of clopidogrel 375 mg at least 6 hours before the procedure, since this dose of clopidogrel produces similar platelet inhibition to a similar dose of aspirin and 75 mg of clopidogrel given for 5 days or more. Because clopidogrel has been shown to have at least equivalent antiplatelet efficacy to ticlopidine, significantly fewer side effects, and a more rapid onset of action when used with a loading dose regimen, clopidogrel and aspirin are used as the standard carotid stent antiplatelet regimen.

Routine pre-procedural sedation should be minimal in carotid stent patients to allow frequent neurological assessment during the procedure. If the preprocedure angiogram shows evidence of intraluminal thrombus, the elective intervention is postponed. Under these circumstances it is our practice to anticoagulate these patients for several weeks in an attempt to allow thrombus resolution and minimize the risk of distal embolization during the intervention.

Internal and Extrathoracic Common Carotid Artery. From the femoral approach, arterial access is obtained and either a long, 6 French introducer sheath (Shuttle sheath, Cook, Bloomington, IN) or, if a multipurpose shape coronary guide catheter is to be used, a short, 8 French sheath is inserted. The patient is anticoagulated with heparin. A 125 cm long, 5 French diagnostic catheter (JR-4, IMA, Berenstein, Simmons, Headhunter [H5N] or Vitek) is placed through the 6 French sheath or 8 French coronary guide catheter and functions as an introducer. Either the innominate or the left common carotid artery is engaged using the 5 French diagnostic catheter. A 0.035 inch stiff angled Glidewire™ is advanced into the external carotid artery and the diagnostic catheter is advanced (over the wire) several centimeters into the common carotid artery. For gentle curves, the softer wires are often sufficient to support the sheath or coronary guide catheter in entering the common carotid artery over the 5 French diagnostic catheter. However, for more acute angles or tortuous arteries, the soft steerable wires are exchanged for an extra-stiff 0.035 inch Amplatz wire to provide more support. The diagnostic catheter is then used as an introducer to assist the atraumatic advancement of the sheath or guiding catheter into the common carotid artery. The 0.035 inch guidewire and 5 French diagnostic catheter are removed, leaving the sheath or coronary guide catheter in the common carotid artery. It is useful to confirm that the activated clotting time (ACT) is greater than 250 sec (some believe ≥300 sec) at this time, and to give additional heparin if necessary prior to crossing the lesion with a guidewire.

Baseline carotid angiography with digital subtraction is performed and intracranial views are obtained. Quantitative angiographic measurements of the internal carotid artery distal to the lesion and the common carotid artery are obtained to assist in balloon and stent sizing. The lesion is crossed either with an exchange length, extra-support coronary guidewire (0.014 inch or 0.018 inch) or with a soft-tipped steerable wire that is then exchanged for an extra-support wire. Care is taken to control the distal portion of the guidewire to avoid intimal damage or spasm in the intracranial portion of the carotid artery.

Predilation of the carotid lesion is often performed with undersized balloons to avoid the "Dotter" effect of advancing the bulky stent delivery catheter across the lesion. For lesions involving the carotid bifurcation, some investigators prophylactically place a temporary right ventricular pacemaker to treat bradycardia that may occur with balloon inflation. Others premedicate the patient with 0.5 mg of atropine prior to balloon inflation and some operators only use atropine if bradycardia occurs (Table 11-4).

Self-expanding stents are used for lesions not protected by the axial skeleton or skull from external compression (Fig. 11-8). Self-expanding stents are sized at least 1 mm larger than the reference diameter. For lesions at the carotid bifurcation, the

Table 11-4

Pharmacological Management of Elective Carotid Stenting

Pre-Procedure Antiplatelet Agents	Intra-Procedure Anticoagulation	Postprocedure Antiplatelet Agents
Aspirin 325 mg PO at least 24 h before Clopidogrel 75 mg PO for 5 days or 375 mg PO 6 h prior to the procedure	Heparin 70–100 u/kg to keep ACT ≥300 sec Heparin 70 u/kg and keep ACT between 200 and 250 sec if abciximab used Abciximab (optional) 0.25 mg/kg bolus then 0.125 μg/kg/min	Aspirin 325 mg indefinitely Clopidogrel 75 mg PO for at least 4 weeks
No sedation	**Bradycardia**	**Hypotension**
	Atropine 0.6–1 mg IV up to 2 mg IV	Neo-Synephrine infusion: titrate to keep SBP ≥130 mm Hg
	Hypotension	**Persistent Hypotension**
	Normal saline intravenously Neo-Synephrine 100 μg IV boluses to keep SBP ≥130 mm Hg	Midodrine hydrochloride 5.0 mg 2–3 times daily

(Modified from Silva JA, White CJ. Adjunctive pharmacologic treatment for elective stenting of the extracranial carotic arteries. *Int J Cardiovasc Interv* 2001;4:141–144.)

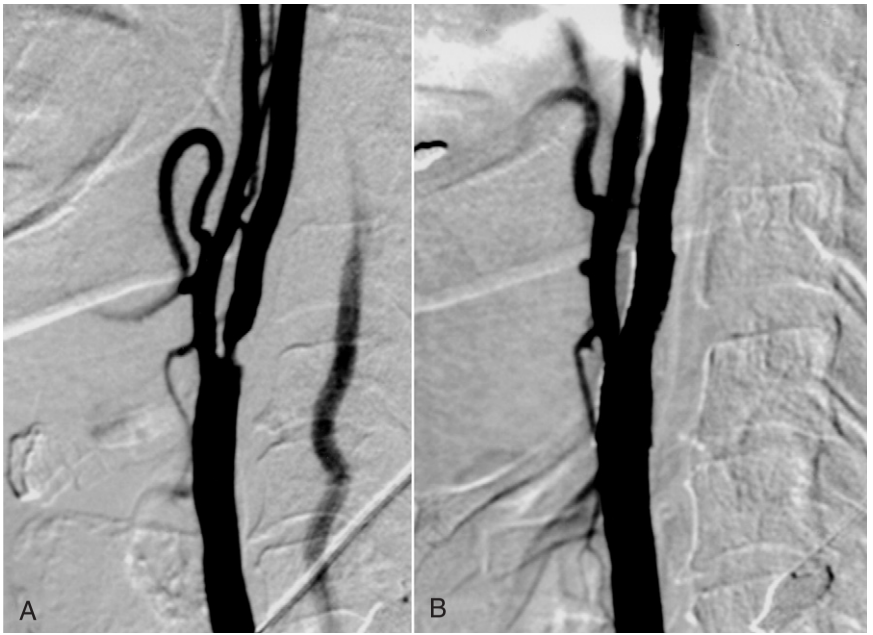


Fig. 11-8 **A,** Baseline angiogram of right internal carotid artery stenosis. **B,** Angiogram after self-expanding stent placement.

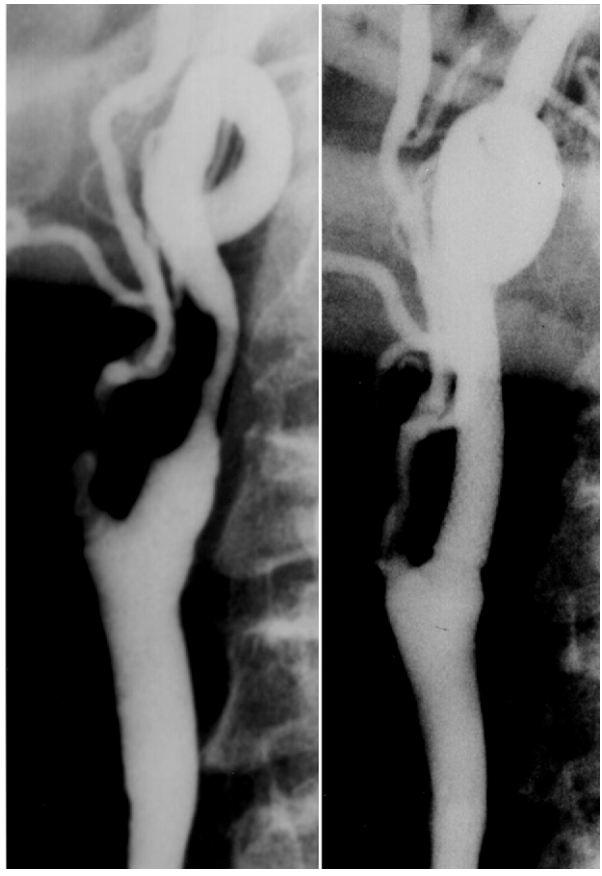
stent is sized to be larger than the common carotid artery. There does not appear to be any disadvantage in placing self-expanding stents across the origin of the external carotid artery. Many operators routinely use self-expanding stents that are 8 mm or 10 mm in diameter stent and either 2 cm or 4 cm in length for all internal carotid lesions.

After stent deployment, a final balloon inflation is performed to help expand the stent. The balloon size is determined by quantitative measurement of the internal carotid artery distal reference segment. Experienced operators emphasize the importance of conservative balloon sizing (≤1:1) for postdeployment balloon dilations and accept a residual diameter stenosis of ≤50% as a good final result. This conservative approach avoids the risk of vessel rupture, minimizes distal dissections, minimizes barotrauma to the carotid body, and potentially decreases the risk of distal embolization that may occur with high-pressure inflations (Fig. 11-9).

Following postdeployment balloon dilation, final digital subtraction angiograms of the carotid lesion, with intracerebral views, are performed. It is important to confirm that there are no missing intracerebral branches and there are no distal internal carotid artery dissections secondary to guidewire manipulation. Before the catheters are withdrawn, a neurologic examination is performed to insure that the patient is neurologically intact. If a deficit is discovered, the angiograms are reviewed to look for a culprit lesion and, if one is discovered, attempts to relieve the ischemia are made (neurologic rescue). The ability to perform a neurologic rescue procedure is an essential element to ensuring the safety of the carotid stent procedure.

Aorto-Ostial and Intrathoracic Common Carotid. These vessels are protected by the thorax from external compression, and require precision placement of the stent at the ostial portion of the vessel. In these ostial lesions, we favor the use of balloon expandable stents.

Femoral access is obtained with either an 8 or 9 French sheath depending on the diameter of balloon and stent to be deployed. Anticoagulation with heparin is obtained. A 5 French diagnostic catheter (JR-4, IMA, Berenstein, Headhunter, or Vitek) is advanced through the 8 French multipurpose guiding



A B

Fig. 11-9 **A,** Baseline angiogram of right internal carotid artery stenosis. **B,** Angiogram after self-expanding stent placement.

catheter to the aortic arch and the ostium of the common carotid artery is gently engaged. The lesion is crossed with an 0.035 inch exchange length Wholey guidewire (Malinckrodt, St Louis, MO). The coronary guiding catheter (multipurpose or hockey-stick) is advanced over the diagnostic catheter to the ostium of the target vessel and baseline angiography of the lesion and intracranial angiography are performed.

The ostial lesion is then predilated with a balloon sized 1:1 with the common carotid artery reference diameter. Upon deflation of the balloon, the guiding catheter is gently advanced over the balloon into the target vessel. This allows the guide catheter to act as a sheath and protect the stent during delivery to the lesion. The balloon catheter is removed and a balloon-expandable stent is then advanced to the ostial carotid lesion within the guiding catheter. The guiding catheter is withdrawn and final positioning of the stent is performed with contrast injections from the guiding catheter. It is preferable to have the stent protruding slightly into the aorta to ensure that the ostium of the target vessel is covered by the stent struts. The stent is deployed with balloon inflation, usually 10–12 atm.

After the final inflation, the guiding catheter is once again advanced over the deflating balloon to selectively intubate the stent segment. This allows easy access to the stent if a second stent is required. Final angiography is performed, including intracranial views, and a neurological assessment is performed on the table.

Post-Procedural Care. Patients who have had an uneventful procedure are observed in a postangioplasty recovery area, with frequent neurologic exams over the next several hours. Arterial sheaths are removed when the ACT falls to less than 170 sec. Alternatively, femoral closure devices may be used to assist hemostasis.

Troubleshooting. Patients who are moderately hypertensive (systolic ≤180 mm Hg), should be observed, as their blood pressure will generally fall to an acceptable range over several hours. Hypotension may lead to relative hypoperfusion of the brain and neurologic symptoms due to a delay in the cerebrovascular autoregulatory vasomotor response.

If a patient becomes hypotensive due to carotid body stimulation, it is important to pharmacologically support their blood pressure to maintain brain perfusion and to vigorously administer fluids to counteract the reflex vasodilation that may occur. Vasoconstrictor medications (Neo-Synephrine) may be titrated intravenously to achieve a target systolic blood pressure of 140 mm Hg or more. Most patients will normalize their blood pressure over several hours, but in severe cases hypotension may persist for 24–36 hours. It is also important to look for potential bleeding sources in any patient who becomes hypotensive during or following an invasive vascular procedure.

Some patients may remain hypotensive for several hours after completion of the procedure, requiring a continuous infusion and titration of a vasoconstrictor to maintain adequate blood pressure and brain perfusion. When this is the case, we prescribe oral midodrine hydrochloride 2.5–5.0 mg, two to three times a day. In most patients, the Neo-Synephrine infusion can be successfully terminated and patients can start ambulation in preparation for discharge within 24 hours. Midodrine hydrochloride is gradually tapered and discontinued and the patient's antihypertensive medications are gradually resumed as necessary.

Carotid stent patients should have a complete neurological examination within 24 hours of the procedure. It is preferable to have this examination done by an unbiased observer (neurologist or vascular internist) who is not a member of the interventional team. Patients are usually discharged from the hospital the morning after the procedure. Aspirin (325 mg per day) is prescribed indefinitely if there are no contraindications to its use. Clopidogrel (75 mg per day) is given for a minimum of 30 days following the procedure. A followup clinical examination and carotid duplex ultrasound are scheduled for 1 month.

Clinical Results. Several nonrandomized studies using endovascular stenting as revascularization strategy have shown very favorable results (Table 11-2). The results of CAVATAS (the Carotid and Vertebral Artery Transluminal Angioplasty Study), a randomized trial of carotid angioplasty versus surgery, have been favorable. A total of 504 patients (>90% symptomatic) were randomized to either carotid endarterectomy (n = 253) or

carotid angioplasty (n = 251). Only 26% of the angioplasty patients received a carotid stent for a failed angioplasty result; the remainder were treated with balloon angioplasty alone.

The 30-day endpoint of disabling stroke or death showed no difference between the angioplasty arm (10%) or the surgical arm (9.9%). The 95% confidence intervals (CI) for the surgical event rate (9.9%, 95% CI 6.2–13.6%) overlapped with the complication rates of both the European Carotid Surgery Trial (7.0%, 95% CI 5.8–8.1%) and the North American Symptomatic Carotid Endarterectomy Trial (6.5%, 95% CI 5.2–7.8%). Complications of cranial nerve injury and myocardial ischemia were only seen in the surgical arm. Long-term followup has shown no difference in neurological events between the groups. The authors concluded that angioplasty and surgery were equivalent for safety and efficacy but the angioplasty group experienced less procedural morbidity.

The results of a landmark trial (Stenting and Angioplasty with Protection in Patients at High Risk for Endarterectomy, SAPPHIRE) compared carotid stenting with distal protection to endarterectomy in high-risk surgery patients. A total of 723 patients were enrolled in the trial, with 156 randomized to carotid stenting with distal protection, 151 randomized to endarterectomy, and 416 patients treated in a nonrandomized registry (409 stent, 7 surgery).

The 30-day stroke/death/myocardial infarction rate for the randomized patients was significantly lower in the carotid stent group (5.8%) compared to the surgical group (12.6%, $p < 0.05$; Table 11-5). In the nonrandomized registry patients the 30-day stroke, death, or myocardial infarction rate was 7.8% for stents and 14.7% for surgery. The surgical group also

Table 11-5

SAPPHIRE Trial: Carotid Stent with Distal Protection Versus Endarterectomy in High-Risk Surgery Patients*

	Stent (%)	Surgery (%)	*p* value
Symptomatic	4.2	15.4	NS
Asymptomatic	6.7	11.2	NS
All	5.8	12.6	0.047

* 30-day stroke, death, and myocardial infarction.

had an excess of cranial nerve injury (5.3%), which was not seen in the stent group. Long-term followup in this trial is ongoing. This trial provides strong evidence that stent placement with distal protection is the procedure of choice in patients at increased risk for surgery.

Currently there are no FDA-approved carotid embolus protection devices or stents. The distal protection devices are available only under the auspices of a IDE investigation, with the exception of the approved PercuSurge™ (Medtronic, Minneapolis, MN) system (Fig. 11-10). Several large reports suggest that the presence of embolic material is very common; however, there has been no significant reduction in embolic complications demonstrated.

RENAL ARTERY INTERVENTION

Secondary hypertension due to renal artery stenosis occurs in the general population with a prevalence of less than 5%; however, its occurrence is substantially higher in patients with established peripheral and/or coronary vascular disease, patients with hypertension and concomitant renal insufficiency, and diabetic patients. In one study of 196 patients undergoing cardiac catheterization for presumptive coronary artery disease, it was found that the prevalence of significant (>50%) renal artery disease was 18% and among those with confirmed coronary artery disease the prevalence was 22%. Some investigators have reported an incidence of renal artery stenosis as high as 60% in patients with documented peripheral artery disease and concomitant hypertension. Percutaneous revascularization for significant renal artery stenosis has been shown to be a very effective treatment modality for this condition.

Indications

Significant hemodynamic obstruction of the renal flow activates the renin–angiotensin system, leading to production of angiotensin II, which in turn causes systemic hypertension and fluid retention. Patients at high risk for renal artery stenosis should undergo a noninvasive screening test to rule out this condition (Box 11-2). The noninvasive tests of choice for making the diagnosis of renal artery stenosis are renal duplex ultrasound, magnetic resonance angiography, and computed

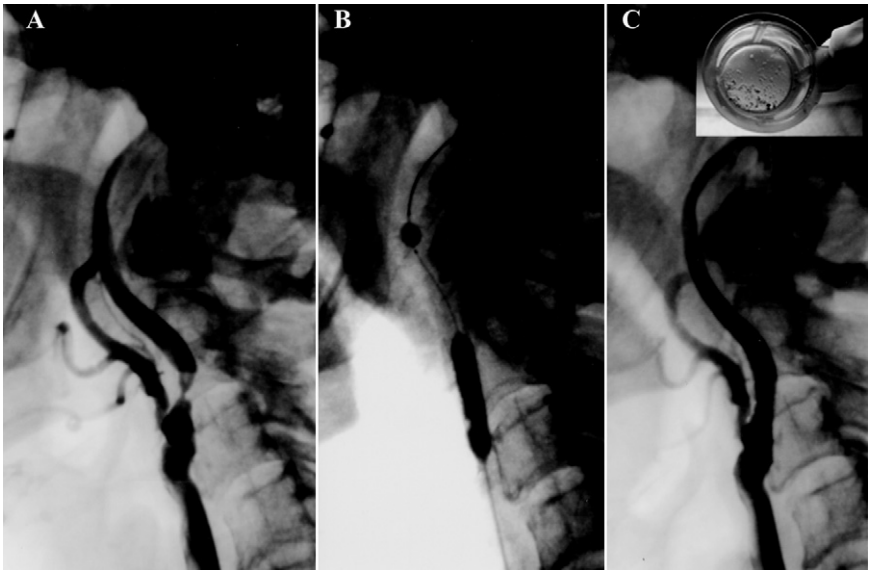


Fig. 11-10 **A,** Baseline angiogram of right internal carotid artery stenosis. **B,** PercuSurge™ occlusion balloon in place during balloon inflation. **C,** Angiogram after self-expanding stent placement. Insert at upper right shows plaque debris washed out from PercuSurge protection system.

Box 11-2

Hypertensive Patients at Increased Risk of Renal Artery Stenosis

- Abdominal bruit (systolic and diastolic)
- Onset of hypertension <30 years or >55 years
- Previous control of hypertension, now uncontrolled
- Malignant hypertension
- Hypertension refractory to medical management
- Unexplained azotemia in an elderly patient with atherosclerosis
- Azotemia with angiotensin converting enzyme inhibitors or angiotensin receptor antagonists
- Atrophic kidney

tomographic angiography (Table 11-6). Captopril renal artery scintigraphy is a relatively specific but insensitive test to demonstrate unilateral renal artery stenosis; however, the incidence of false negatives is substantial. Measurement of plasma renin levels is discouraged, because it is neither a specific nor a sensitive indicator of renovascular hypertension.

One of the most important indications for revascularization of the renal arteries is to improve blood pressure control (Box 11-3). Stenting for renal artery stenosis has been shown to

Table 11-6

Screening Tests for Renal Artery Stenosis

Test	Advantage(s)	Disadvantage(s)
Duplex ultrasound	High sensitivity and specificity	Operator-dependent; difficult in obese patients
Magnetic resonance angiography	Good sensitivity and specificity	Increased false positives. Not useful if metal stent present
Computed tomographic angiography	Good sensitivity and specificity, useful if stents present	Ionizing radiation Radiographic contrast required
Captopril renal artery scintigraphy	Good specificity	Poor sensitivity
Renal vein renin	Lateralizing renin predicts treatment response	Poor sensitivity/specificity; invasive
Renal angiography	High sensitivity and specificity	Invasive

Box 11-3

Indications for Renal Artery Stenting

- Hypertension control
- Preservation and/or improvement of renal dysfunction
- Stabilization and/or improvement of angina pectoris
- Stabilization and/or improvement of heart failure symptoms

have a beneficial immediate and long-term impact for controlling hypertension. In a study from our institution of 100 patients, procedural success was obtained in 99%. Blood pressure significantly decreased from 173 ± 25/88 ± 17 to 140 ± 21/73 ± 10 immediately after stenting to 146 ± 20/77 ± 12 at 6-month followup ($p < 0.01$; Fig. 11-11). In another study of 163 consecutive patients, blood pressure decreased from 166 ± 26/86 ± 14 to 148 ± 22/80 ± 11 ($p < 0.05$) at 4-year followup.

Another indication for revascularization of renal artery stenosis is for preservation of renal function, reversal of end-stage renal failure in selected patients, or decrease in progression of renal failure. Harden *et al.* assessed the renal function of 32 patients with renal artery stenosis before and after treatment with endovascular stents. These investigators demonstrated a significant slowing in the progression of renal failure after stenting compared to before stenting by calculating the mean slope of the reciprocal of serum creatinine.

Renal artery stenting has also been shown to improve functional class in patients with unstable angina and congestive heart failure, probably through a mechanism of better blood pressure control and by favorably affecting the renin–angiotensin system.

Procedural Technique

Aspirin is started at least 1 day prior to the procedure. Although in the majority of the cases the retrograde femoral access is used (when the takeoff of the renal artery is horizontal, caudal, or mildly cephalad), in some cases the brachial access is necessary (when the takeoff of the renal artery is overly cephalad) to ensure a successful procedure.

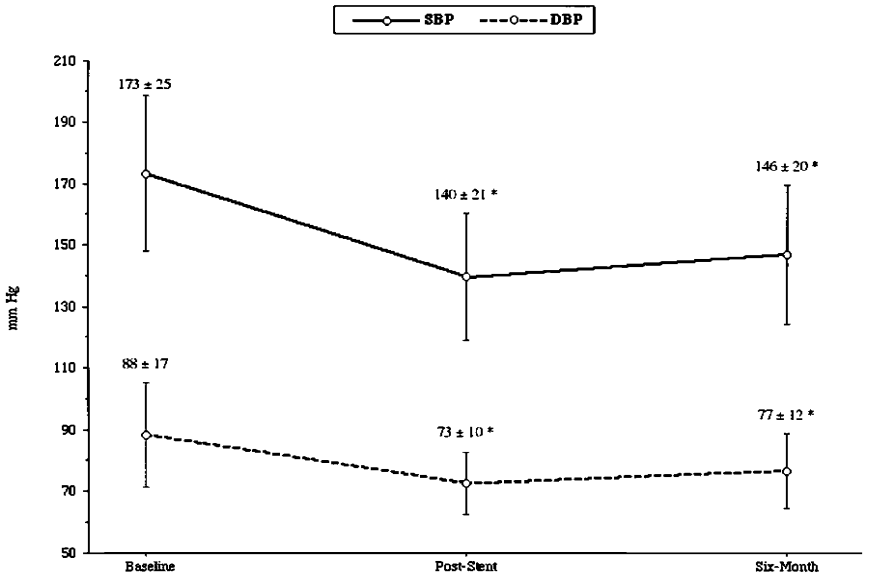


Fig. 11-11 Blood response to renal stents. DBP, diastolic blood pressure; SBP, systolic blood pressure. (Redrawn from White CJ, Ramee SR, Collins TJ, *et al.* Renal artery stent placement: utility in lesions difficult to treat with balloon angioplasty. *J Am Coll Cardiol* 1997;30:1445–1450.)

After vascular access is obtained, intravenous heparin is given.

When the brachial access is used for a cephalad-oriented renal artery, a 6.5 French, 90 cm long vascular sheath (Daig, Minnetonka, MN) is advanced over the guidewire and positioned in the suprarenal abdominal aorta. A 6 French multipurpose diagnostic catheter is then advanced through the long sheath and is used to engage the renal artery. A soft-tip exchanged-length 0.035 inch steerable guidewire (Wholey wire, Malinckrodt, St Louis, MO) is advanced into the renal artery. Keeping the multipurpose diagnostic catheter engaged in the renal artery and the guidewire in a distal branch of the renal artery, the sheath is advanced over the multipurpose catheter and positioned in contact with the ostium of the renal artery. The multipurpose diagnostic catheter is then removed, leaving the guidewire in the renal artery and the sheath in contact with the ostium of the renal artery.

When retrograde CFA access is chosen, a 7 or 8 French sheath is inserted. A 6 French diagnostic catheter (internal mammary, cobra, or Judkins right configuration) is advanced to engage the ostium of the renal artery. A soft-tip exchange-length 0.035 inch guidewire is used to cross the lesion and is positioned in a renal artery branch. The diagnostic catheter is then exchanged over the guidewire for a 7 or 8 French renal angioplasty guiding catheter, which is positioned in contact with the ostium.

We recommend avoiding the use of a Glidewire™, since this may cause inadvertent perforation and/or dissection. Also, we prefer to use 5/6 French diagnostic catheters to locate the ostium of the renal arteries to avoid trauma and potential cholesterol embolization from scraping the aorta that may occur with larger angioplasty guiding catheters.

After the RVD is measured with online quantitative angiography a peripheral angioplasty balloon (4–8 mm in diameter, 2 cm long) is advanced over the guidewire and positioned at the lesion. The lesion is then dilated with a balloon sized 1:1 with the RVD, using the lowest pressure that will fully expand the balloon. If a decision to proceed with stenting is made because of a suboptimal angiographic result, the balloon is inflated again at a low pressure (1–2 atm) and, while the balloon is deflating, the catheter is advanced across the lesion

over the balloon. This maneuver enables the stent to be positioned at the lesion site (within the sheath) without risking its edges catching on the plaque and reducing the risk of stent embolization.

Balloon-expandable stents are used to scaffold the lesion and maximize the angiographic result. The stent is advanced over the guidewire, still within the sheath or guiding catheter, to the lesion site. The sheath or the guiding catheter is withdrawn, uncovering the stent, and, with contrast injections through the sheath or the guiding catheter, the stent is positioned at the lesion site. When treating ostial lesions, it is important to allow approximately 1 mm of the stent to protrude into the aorta to ensure complete coverage of the ostium of the artery. The stent is deployed at 6–8 atm, and then the balloon is withdrawn into the sheath or the guiding catheter. Angiography is then performed and, if inadequate expansion of the stent is observed, the operator should repeat dilation of the stent at a higher inflation pressure or with a larger balloon (Fig. 11-12).

Clinical Outcomes

Percutaneous transluminal renal stenting is the preferred treatment for significant stenosis of the renal arteries. Balloon angioplasty, however, remains the treatment of choice for fibromuscular dysplasia and is an accepted treatment for selected patients with renal artery stenosis causing renovascular hypertension and/or renal insufficiency. However, atherosclerotic aorto-ostial renal artery lesions are particularly difficult to treat with balloon angioplasty alone because they are prone to significant vascular recoil, leading to a restenosis rate of approximately 50% over 6 months. Balloon angioplasty alone may have negligible long-term benefit for controlling blood pressure, probably because of a high restenosis rate.

Endovascular stents have the capacity to scaffold dilated lesions and prevent elastic recoil (Fig. 11-13). Several studies have shown a significantly greater acute gain in luminal diameter and better angiographic results with renal artery stenting than with balloon angioplasty alone. In a study of 76 patients and 92 renal arteries treated with primary stenting, technical success was obtained in 100%, with a restenosis rate at 6 months

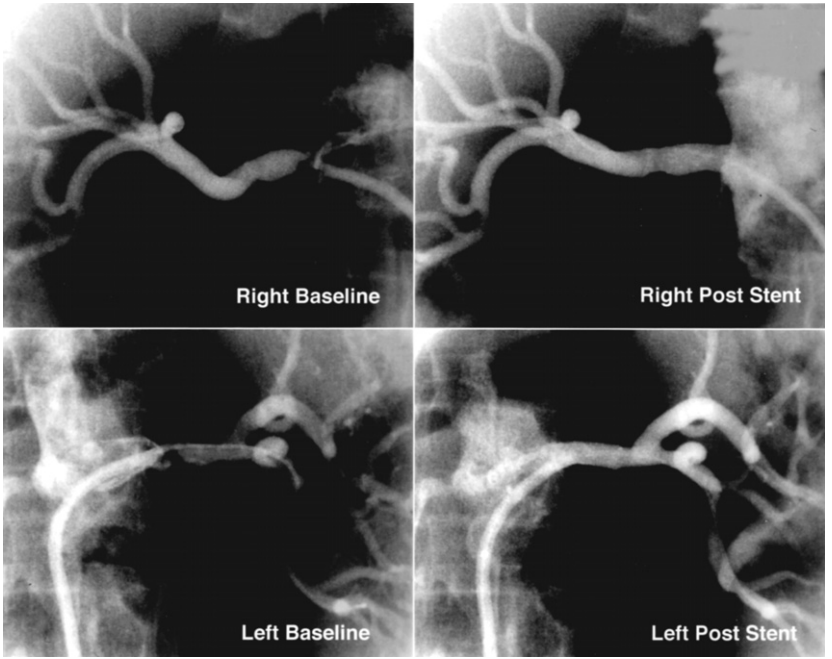


Fig. 11-12 Bilateral renal artery stenosis. *Top left panel,* Baseline angiogram of ostial renal artery stenosis. *Top right panel,* Angiogram after stent placement. *Bottom left panel,* Baseline angiogram of ostial renal artery stenosis. *Bottom right panel,* Angiogram after stent placement.

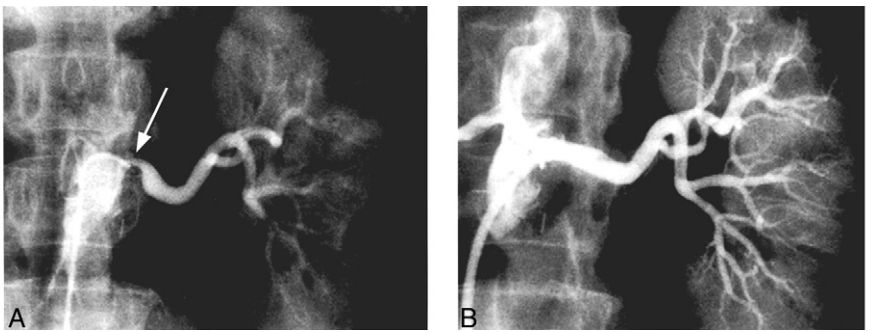


Fig. 11-13 **A,** Baseline right renal artery ostial stenosis. **B,** Angiogram after stent placement.

of 25%. Blum *et al.* treated 74 renal artery stenosis with endovascular stents. Technical success was achieved in 100% of the vessels, and the restenosis rate at 12-month followup was 11%.

In diabetic patients, renal artery stenosis is more prevalent than in the general population. Blood pressure control in this patient population has been shown to have a greater impact than in nondiabetic patients for decreasing cardiovascular events and renal damage. We recently showed that renal artery stenting is equally beneficial for decreasing blood pressure and for attenuating or stabilizing the renal function in diabetic and nondiabetic patients.

Stenting for renal artery stenosis also appears to have a beneficial effect in patients with refractory unstable angina and congestive heart failure. In 48 patients with unstable angina (n = 23) or congestive heart failure (n = 25) who had hypertension refractory to medical therapy and significant unilateral (n = 30) or bilateral (n = 18) renal artery stenosis, stenting significantly improved the blood pressure and functional class at 24-hour and 6-month followup. The dramatic improvement seen in this very sick group of patients was independent of a coronary angioplasty procedure.

In conclusion, the incidence of renal artery stenosis in patients with poorly controlled hypertension and atherosclerotic cardiovascular disease ranges from 20% to 30%. Patients with a high-risk clinical profile for this entity should be screened with a noninvasive test or renal angiography at the time of diagnostic cardiac catheterization. Endovascular stents yield clinical outcomes superior to balloon angioplasty alone for the treatment of atherosclerotic renal artery stenosis. They have a dramatic impact on hypertension control and appear to have a beneficial effect in the treatment of refractory unstable angina and congestive heart failure. Considering the treatment alternatives for atherosclerotic renal artery stenosis causing medically refractory hypertension and/or renal insufficiency, stent placement is the current treatment of choice

ILIAC ARTERY INTERVENTION

Because the retrograde CFA access is the most frequently used access for percutaneous angiography and intervention for both coronary and noncoronary vessels, interventionists should

have the necessary skills to treat these vessels to insure vascular access and control complications.

Indications

The indication to perform an intervention of the iliac arteries includes vascular access and symptomatic lower extremity ischemia. Iliac intervention may also be appropriate in patients with severe stenosis or occlusion of the femoropopliteal or infrapopliteal arteries and concomitant moderate iliac artery disease, in whom revascularizing the moderately stenosed iliac artery may improve the arterial inflow and lead to symptomatic improvement or salvage of the limb. Another indication for iliac artery percutaneous revascularization in asymptomatic iliac lesions is to allow intra-aortic balloon pump placement in patients with cardiogenic shock or resting ischemia. Specific indications to revascularize these vessels are based upon lesion and patient characteristics found in the *Guidelines for Peripheral Percutaneous Transluminal Angioplasty of the Abdominal Aorta and Lower Extremity Vessels*. In general terms, longer lesion length, complete occlusions, heavy calcification, presence of diabetes, and more severe symptoms correlate with lower success rates and poorer long-term patency rates.

Technique

Patients are started on aspirin before the procedure. The most commonly used vascular access for revascularization of the common and external iliac artery is retrograde ipsilateral CFA access. Occasionally, the contralateral or brachial access may be necessary when the very distal portion of the external iliac/CFA arteries is involved.

After gaining vascular access, heparin is given and a baseline angiogram is obtained. After the target lesion has been identified, the RVD is determined, since balloon or stent oversizing may lead to rupture of the external iliac artery. Visual estimation of vessel diameter is discouraged. The authors recommend the use of online quantitative angiography or intravascular ultrasound. Angiography of the lesion is performed by positioning a catheter immediately above the lesion after it has been crossed with a steerable guidewire. When the lesion is located in the distal common iliac or proximal external iliac artery, retrograde hand injections of

contrast through the femoral sheath may also be used. One particular angiographic view, which nicely separates the origin of the internal and external iliac arteries, is the contralateral caudal oblique view (20° lateral oblique and 20° caudal). The soft-tip guidewire is exchanged for an extra-stiff guidewire (0.035-inch Amplatz wire, Cook, Bloomington, IN) to provide support and trackability for stent placement.

The lesion is dilated with a balloon sized 1:1 with the RVD, using the lowest pressure that will fully expand the balloon. The results are assessed by reinserting the pigtail catheter above the lesion or using a hand injection of contrast through the sheath. The practice of provisional stenting requires stent placement only for suboptimal results, although most experts believe that primary stenting is appropriate for iliac lesions.

Balloon expandable stents are preferred when a precise stent placement is required, and self-expanding stents are preferred when precision is not a critical factor and the vessel tapers in size (Fig. 11-14). For balloon-expandable stents we use an arterial sheath long enough to cross the lesion to avoid having the undeployed stent catch on the lesion, risking embolization or dislodgment of the stent. When the stent is at the lesion site but still within the sheath, the sheath is withdrawn and contrast is injected to confirm the correct position of the stent. The stent is then deployed using at least 6–8 atm of pressure to ensure adequate stent expansion and apposition to the vascular wall.

If doubts regarding adequate stent expansion remain after post-stent angiography, the operator may repeat balloon inflation using a higher inflation-pressure or use a bigger balloon. Simultaneous pressures using a catheter proximal to the target lesion and the vascular sheath should be performed to ensure a final gradient of 5 mm Hg or less.

Clinical Results

Traditional surgical therapies for iliac lesions include aortoiliac and aortofemoral bypass. These bypass procedures have a 74–95% 5-year patency rate, which is comparable with but not superior to percutaneous intervention. Ameli and coworkers reported a series of 105 consecutive patients undergoing aortofemoral bypass. The majority (58%) of their patients had mild to moderate clinical symptoms and were

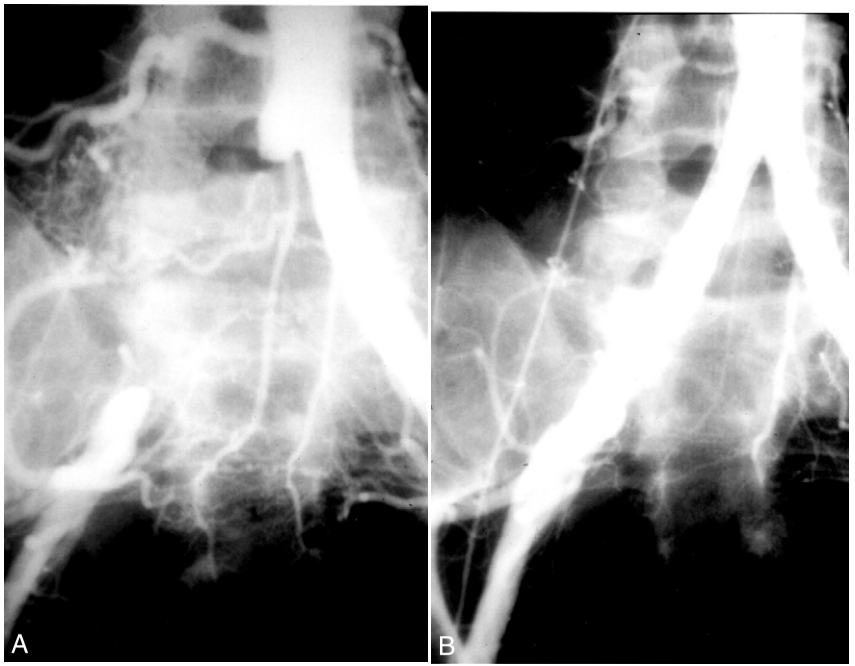


Fig. 11-14 **A,** Baseline angiogram with occlusion of right common iliac artery. **B,** Following stent placement.

treated for claudication. The operative mortality was 5.7%, the early graft failure rate was 5.7%, and the 2-year graft patency rate was 92.8%.

Several studies have suggested that the use of endovascular stents yields a higher procedural success rate and a lower restenosis rate than balloon angioplasty alone. In one meta-analysis comparing six balloon angioplasty studies (1300 patients) with eight stent placement studies (816 patients), the technical success was higher for the stent group (96% vs 91%; $p < 0.05$). The complication and mortality rates were similar for the two groups. The 4-year primary patency rates for restenosis lesions (77% vs 65%) and occlusions (61% vs 54%) in patients with claudication was statistically higher in the stent-treated group. The 4-year primary patency rate for stenoses (67% vs 53%) and occlusions (53% vs 44%) in patients with critical limb-ischemia was also statistically higher in patients treated with stents.

Femoropopliteal Artery Intervention

Atherosclerotic occlusive disease is three to five times more common in the femoropopliteal artery than in the iliac artery. When the femoropopliteal artery is involved in symptomatic lower extremity, occlusions are three times more frequent than stenosis. This distribution is the opposite in the aortoiliac vessels.

Indications

Revascularization of the femoral or popliteal arteries is reserved for patients with lifestyle-limiting claudication, ischemic rest pain, and limb-threatening ischemia. The technical success of endovascular revascularization procedures and the long-term patency rate vary according to the lesion characteristics. Treatment of short (<5 cm) occlusions yields better results than treatment of long (>10 cm) occlusions or stenosis. The presence of patent runoff vessels correlates with long-term benefits, reflected in the improved outcome in patients with milder symptoms. Significant residual stenosis after angioplasty correlates with a poor long-term outcome whereas the absence of diabetes correlates with an improved patency rate.

Provisional stent placement (for suboptimal PTA results) remains the endovascular treatment of choice for femoral

and popliteal artery disease. Stenting of these vessels has not shown to yield patency rates superior to angioplasty alone. We believe stents should only be used in cases of suboptimal results, flow limiting dissection, or abrupt occlusion after balloon angioplasty.

Technique

The most commonly used vascular access for the treatment of femoropopliteal arterial disease is the contralateral CFA access. The antegrade vascular access cannot be used for the treatment of CFA or ostial SFA disease. The brachial and retrograde popliteal access are occasionally used; in particular, the popliteal access may prove useful to recanalize SFA occlusions when antegrade approaches have failed.

Aspirin is started the day before the procedure and heparin is given after arterial access has been gained. Then the vascular sheath or guiding catheter are positioned and the lesion is localized with a baseline angiogram. For total occlusions hydrophilic guidewires (Glidewire™) are very useful, when other guidewires frequently fail to cross. When a hydrophilic guidewire is used to cross a lesion, we prefer to exchange it for a nonhydrophilic wire prior to intervention because tracking can be difficult and wire position can be lost. After the RVD is measured with on-line quantitative angiography, the lesion is dilated with a balloon sized 1:1 with the RVD, using the lowest pressure that will fully expand the balloon.

If the postprocedural angiogram shows a satisfactory angiographic result (a residual diameter stenosis of ≤30%) and no flow-limiting dissection, the procedure is terminated. However, if there is significant residual stenosis, flow limiting dissection, or abrupt occlusion, the operator should proceed with stent placement. In general terms, balloon expandable stents are not used outside the axial skeleton. Finally, when a stent is needed, we add to the pharmacologic regimen clopidogrel 75 mg for 4 weeks, after a loading dose of 300 mg usually given in the catheterization suite.

Clinical Results

Balloon angioplasty can be accomplished with a high procedural success and low complication rates, although the

long-term patency rates are not as favorable as in the larger iliac arteries (Fig. 11-15). In a study of 236 patients who underwent conventional balloon angioplasty, procedural success was obtained in 96%. At 1-month followup, procedural success (determined by an improved clinical grade and noninvasive vascular laboratory measurements) was 89%. The success rates at 1- and 4-year followup significantly decreased, to 62% and 38%. The independent predictors of procedural success in this study on multivariate analysis were adequate distal runoff and lesion stenosis rather than occlusion. In another study it was found that the early and 3-year patency rate was superior in patients treated for claudication (89% and 62%) than in patients treated for limb salvage (77% and 43%).

In contrast to the favorable impact of endovascular stents on patency rate in the aortoiliac system, these devices have not been clearly shown to improve the late patency rate when implanted in the femoropopliteal system. A prospective study showed that conventional femoropopliteal angioplasty has a 1-year primary patency rate (65%) equivalent to the secondary patency rate of femoropopliteal Wallstents (69%). In this study, early clinical significant restenosis was 38% and early thrombosis was 19% in the stent group. On the other hand, the use of Intracoil stents for significant stenoses of the femoropopliteal arteries appears promising. A pilot study of 93 patients with stenosis or occlusion (29%) of these vessels showed a 9-month target vessel revascularization of 82% (Fig. 11-16). Likewise, a recent prospective randomized trial of drug-eluting, self-expanding, coated stents with sirolimus for femoropopliteal stenosis showed significant inhibition of intimal proliferation compared with noncoated stents at 6-month followup.

BELOW-KNEE INTERVENTION

The traditional indications for infrapopliteal angioplasty have been ischemic rest pain and ischemic ulceration or gangrene. However, infrapopliteal angioplasty for severe claudication that prevents ambulation, and for patients with moderate to severe claudication to increase the durability and effectiveness of femoropopliteal PTA, has been advocated by some. It is possible that with the advent of small profile balloons,

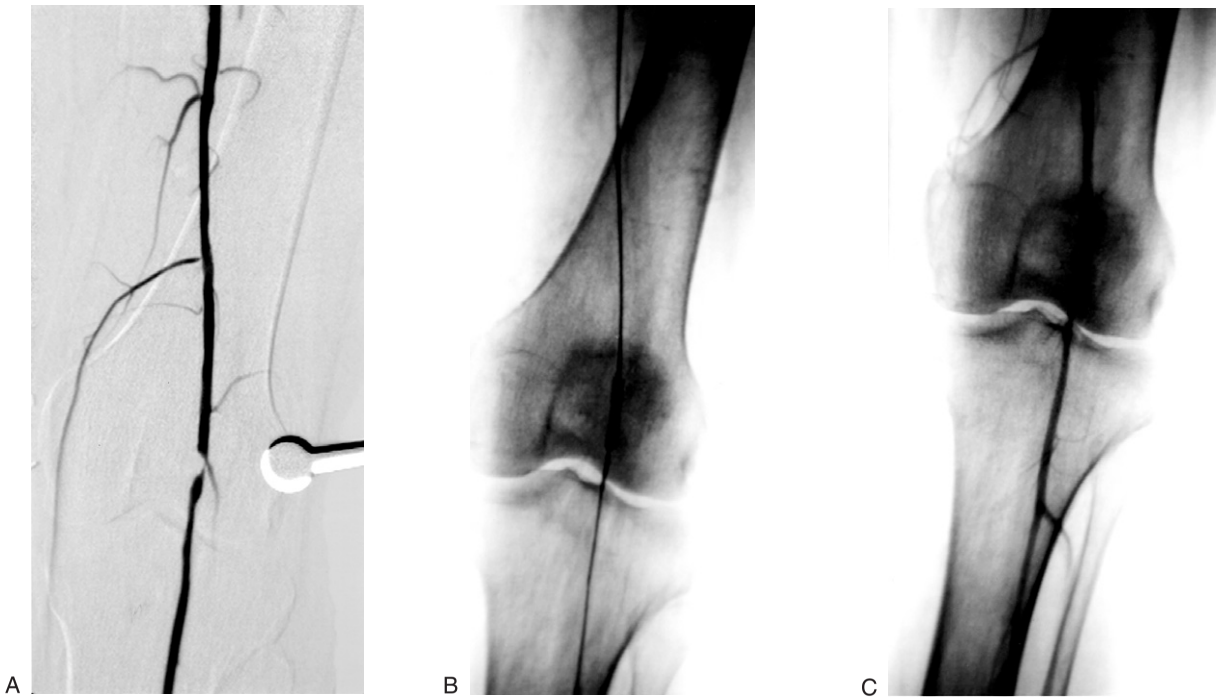


Fig. 11-15 **A,** Baseline angiogram of tight lesion in the popliteal artery. **B,** Balloon inflation. **C,** Postballoon angiogram.

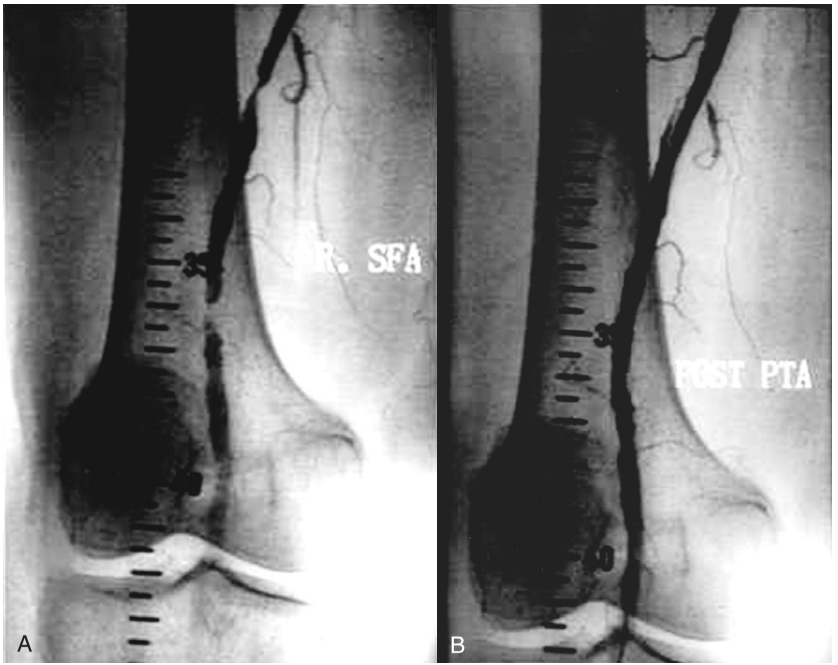


Fig. 11-16 **A,** Right femoral and popliteal stenoses. **B,** Following balloon dilation with dissection present.

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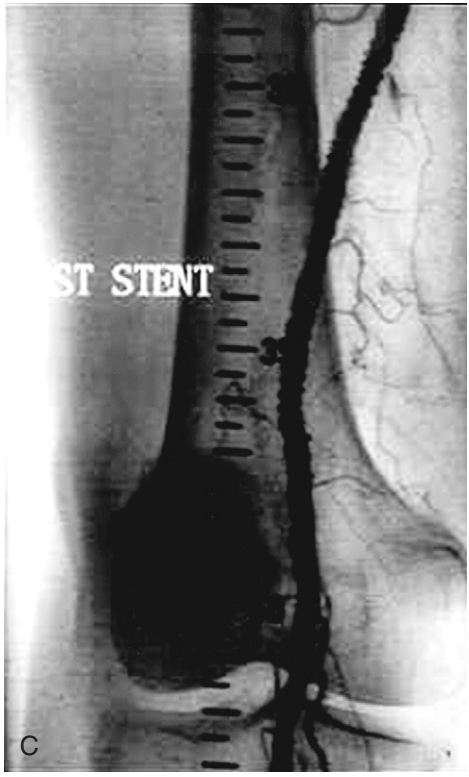

Fig. 11-16, cont'd **C,** Following VascuCoil stent placement.

improvement in technique, and increased operator experience, the use of tibial angioplasty will be not limited to the above-mentioned indications.

Technical Considerations

The preferred vascular access to perform percutaneous intervention of the infrapopliteal vessels is the ipsilateral antegrade CFA access, which enables an almost direct approach to the infrapopliteal vessels. The contralateral CFA access using a crossover approach is also useful, particularly when planning simultaneous revascularization of the iliac arteries, CFA, or proximal SFA. When using the contralateral crossover approach the operator must bear in mind that catheter length

is an issue and that long (150 cm) shafts are usually necessary to reach the infrapopliteal vessels.

All patients are pretreated with aspirin 325 mg from 1 day prior to the procedure. After contralateral CFA vascular access has been obtained, the patient is anticoagulated with 5000–10,000 units of heparin. A soft-tipped 0.035 inch guide wire is advanced to the distal popliteal artery. A 6 French multipurpose guiding catheter is then advanced over the guidewire and positioned at the mid or distal popliteal artery. Baseline angiography of the infrapopliteal vessels is obtained using hand-injection of contrast through the guiding catheter or sheath. After the stenosis has been identified, the lesion is usually crossed with an 0.014-inch guidewire. After the lesion is crossed, online quantitative angiography is obtained for a more accurate measurement of the RVD.

A balloon catheter is chosen for a 1:1 balloon to RVD ratio. The balloon is inflated usually at 6–8 atm of inflation pressure, or more if necessary to allow complete expansion of the balloon. Multiple inflations are performed as necessary to attain a satisfactory angiographic result. In case of suboptimal angiographic result, more than 30% residual stenosis, dissection, or slow flow, it is our practice to place a stent. Post-stenting angiography is obtained and special attention must be paid to rule out dissection or perforation (Fig. 11-17).

When coronary stents are deployed in the infrapopliteal vessels, we medicate these patients with clopidogrel, with a loading dose of 300 mg given at the end of the procedure followed by 75 mg daily for at least 4 weeks. Platelet IIb/IIIa inhibitors have been reported to be of benefit in selected cases; however, there is no evidence at present supporting the routine use of these agents in infrapopliteal intervention.

The post procedural sheath management is similar to revascularization in other vascular territories. It is worth mentioning that a recent study reported that suture closure devices might be used with antegrade CFA access.

Clinical Results

Below-knee angioplasty has led to a dramatic decrease in the amputation rate. Dorros *et al.* reported their results of below-knee angioplasty in 111 patients and 168 tibioperoneal vessels. The indications were claudication (42%), nonhealing

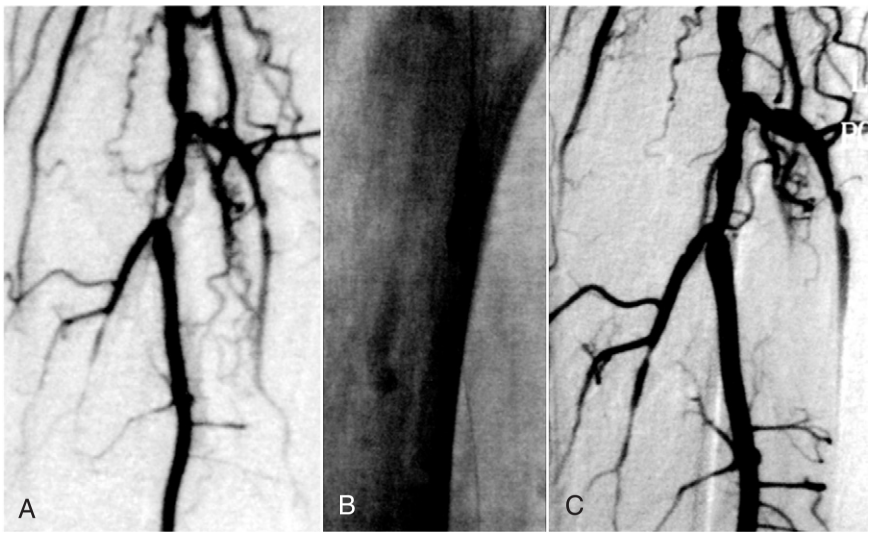

Fig. 11-17 **A,** Baseline below-knee popliteal stenosis. **B,** 3.5 mm coronary balloon inflation. **C,** Postangioplasty result.

ulcer/gangrene (27%), and rest pain (26%). The procedural success was 90% (99% in stenoses and 65% in occlusions). Significant complications (death, emergent bypass surgery, or distal embolization) occurred in 3%. At discharge 95% of the patients were clinically improved. At a mean followup of 9 ± 6 months, 40% needed a second PTA; however, only a third of those who required a second PTA showed lesion recurrence, with the rest showing progression of disease.

Hanna *et al.* reported their results of infrapopliteal PTA for limb salvage in 29 diabetic patients. Technical success (<20% residual stenosis) was achieved in 26 patients (90%), and clinical success (avoidance of amputation and achievement of wound healing) at 12-month followup was obtained in 23 patients (79%). In a prospective series of 284 critically ischemic limbs tibioperoneal angioplasty was successful in 95% of the limbs. Clinical success (relief of rest pain or improvement of lower-extremity blood flow) was successful in 95% of the limbs. At 5-year followup 91% of the limbs were salvaged and 8% required surgery.

Balloon angioplasty of the infrapopliteal vessels is an effective technique for treating patients with distal atherosclerotic occlusive disease. It has been employed mainly in patients with limb-threatening ischemia and multisegment disease. Appropriate anatomic selection is a key factor to maximize the benefit of this technique.

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12

PERCUTANEOUS MITRAL COMMISSUROTOMY AND BALLOON AORTIC VALVULOPLASTY

Ted Feldman and Michael H. Salinger

PERCUTANEOUS MITRAL COMMISSUROTOMY

Inoue reported a single balloon technique for mitral commissurotomy in 1984. Although a number of other techniques have subsequently been described, the Inoue balloon technique and the double balloon technique have been used most commonly. The Inoue technique is the most frequently used in practice internationally and is at present the only approved mitral dilatation balloon in the USA.

Hemodynamic results have been well characterized by the Inoue Multi-Center Registry. On average there is more than 80% increase in mitral valve area. Balloon inflation results in splitting of the fused commissures with reductions in the transmitral pressure gradient, the mean left atrial pressure, and the pulmonary artery pressure. The cardiac output and mitral valve area increase (Table 12-1).

The most important complication of the procedure is mitral regurgitation. Mitral valve replacement is needed during the initial hospitalization in about 2% of patients. An additional 3–4% have resultant 3^+ or greater mitral regurgitation without the need for immediate valve replacement. Other complications are shown in Table 12-2.

The durability of the results is excellent. Figure 12-1 shows the stability of the achieved valve area over a period of years. The 5-year actuarial freedom from death with mitral valve

Table 12-1

Hemodynamic Results of Balloon Mitral Valvotomy

Parameter	Pre	Post	*p*
Left atrium (mm Hg)	24 ± 8	19 ± 12	<0.001
Pulmonary artery (mm Hg)	34 ± 14	29 ± 12	<0.001
Mitral gradient (mm Hg)	13 ± 6	6 ± 3	<0.001
Cardiac output (l/min)	4.1 ± 1.1	4.4 ± 1.3	<0.001
Mitral area (cm^2)	1.0 ± 0.3	1.7 ± 0.6	<0.001

replacement or repeat balloon commissurotomy for the Inoue Registry population was 71%. More than 80% of the patients remained symptomatically improved at 5 years.

Technique

The Inoue balloon. The Inoue device differs substantially from conventional balloons. It is constructed of two layers of latex with a nylon meshed sandwiched in between them. The latex is compliant whereas the nylon mesh limits the maximum inflated diameter of the balloon and gives it a unique shape and three-stage inflation characteristics (Fig. 12-2).

Table 12-2

Complications of Percutaneous Transvenous Mitral Commissurotomy

Complication	%
Hospital mitral valve replacement	1.0
Hospital death	1.4
Transient ischemic attack	0.6
Stroke	0
Cardiac perforation	1.4
Pericardiocentesis	
Myocardial infarction	0.3
DC shock for atrial or ventricular fibrillation	1.0
Vascular repair	0.6
Transfusion	0.3
Temporary pacer	0
Mitral regurgitation 3+ or more (no mitral valve replacement)	3.8
Atrial septal defect > 1.5	3.1
Failure to cross mitral valve	1.7

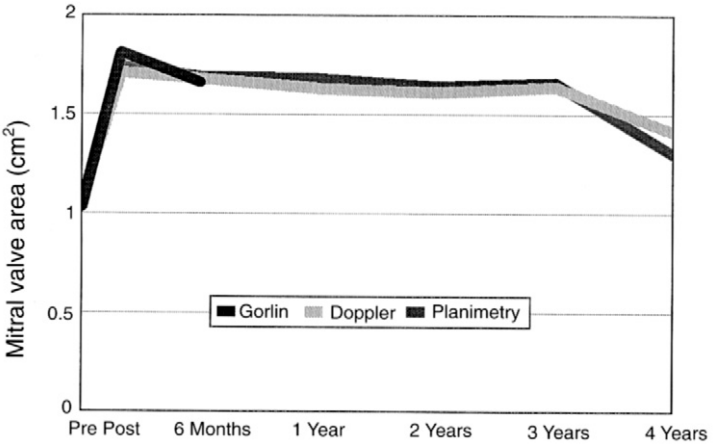

Fig. 12-1 Mitral valve area before percutaneous transvenous mitral commissurotomy (PTMC), immediately after and 4 years following PTMC. Gorlin-calculated valve areas were obtained in 86 patients at repeat catheterization. Doppler and planimetry areas remained relatively constant for 3 years and then fell off slightly in the fourth year.

The front half of the balloon inflates first, giving the appearance of a balloon flotation catheter. The proximal half of the balloon inflates next, creating a dumbbell or hourglass shape. When it is passed across the mitral valve this shape facilitates self-positioning of the balloon device in the valve orifice. Finally, the center portion of the balloon inflates, resulting in splitting of the fused mitral valve leaflet commissures. The distensibility of the latex material allows each balloon to be inflated over a 4 mm range of diameter sizes (i.e., between 26 and 30 mm diameter for the largest available model). A single balloon can thus be used to cause sequential dilatation of the valve by inflating it to serially larger diameters without removing it from the patient. This procedure is thus analogous to coronary angioplasty, during which the result of the balloon inflations is evaluated and additional balloon inflations are performed if necessary.

Patient Evaluation. Evaluation by two-dimensional transthoracic and transesophageal echocardiography is essential before mitral valvotomy. Patients with thin, pliable mitral

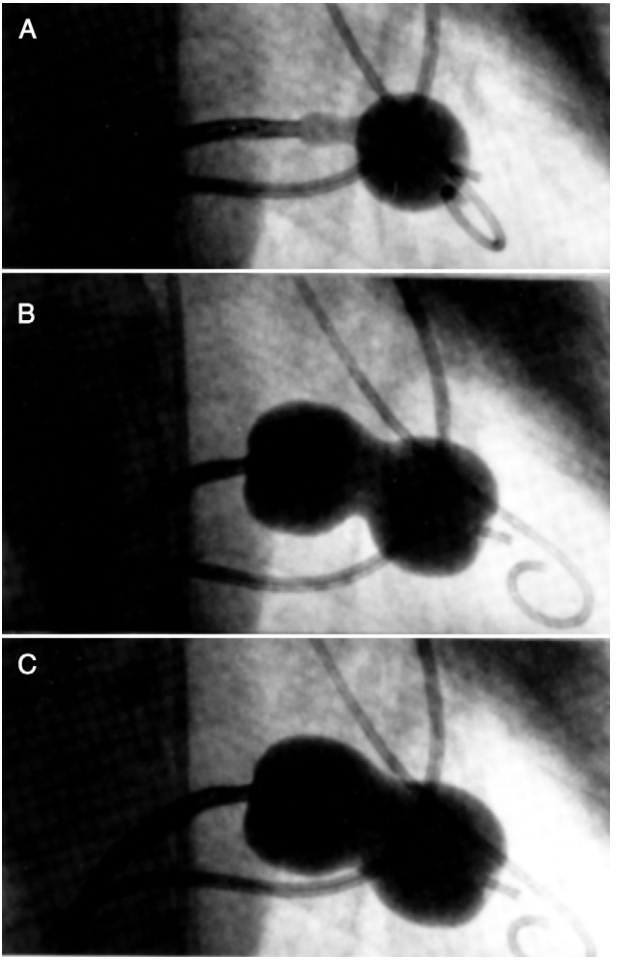

Fig. 12-2 **A,** Front half of balloon inflated and passed across the mitral valve orifice. This is analogous to the manner in which a balloon flotation catheter is maneuvered from the right atrium to the right ventricle during right heart catheterization. The partially inflated balloon is pulled back until it engages the mitral valve. **B,** Front and back portions of balloon inflated, creating a "dog-bone" shape that self-positions the balloon in the mitral orifice. **C,** Almost fully inflated balloon opening the commissures. Note that the inferior indentation in the balloon is more pronounced than the superior indentation, signifying incomplete commissural separation.

leaflets and minimally diseased subvalvular apparatus have the best long-term outcome from surgical commissurotomy (Fig. 12-3). This is no less true when using percutaneous methods to achieve commissurotomy. Although the immediate results of percutaneous transvenous mitral commissurotomy (PTMC) are acceptable in patients with significant valve deformity, the restenosis rate and the need for rate mitral valve replacement remains higher in these patients. The goal of therapy and the long-term prospects for event-free survival must be appropriate for patients with significant valve deformity and echocardiographic scores greater than 10–12.

Transesophageal echocardiography before PTMC is useful for the detection of atrial thrombi. Even when PTMC was performed in patients before the widespread use of transesophageal echocardiography, embolic events were infrequent. Experience since the routine use of transesophageal echo screening has virtually eliminated the chance of this devastating complication.

Atrial thrombi are a strong relative contraindication to the performance of both transseptal puncture and balloon mitral valvotomy. Atrial thrombi are found in 15–25% of patients with mitral stenosis being considered for PTMC, many of whom have been on long-term warfarin anticoagulation therapy even when sinus rhythm is present. In many cases when atrial thrombi are noted it is possible to either institute or intensify anticoagulation for 3–12 months and achieve resolution of thrombi. PTMC may then be undertaken without unnecessary risk. In some cases, small, densely organized thrombi in the atrial appendage may be present. These thrombi are not as likely to contain fresh clots or to be mobile. It is possible to do PTMC in these cases without complications, although this must be done with extreme care and recognition of the serious risk of stroke. Operator experience with the handling characteristics of the Inoue balloon steering stylette is essential in this setting. Some patients in atrial fibrillation without prior anticoagulation therapy are found not to have atrial thrombi upon transesophageal echocardiographic examination. In these cases balloon dilatation may proceed without a prior course of anticoagulation.

Cardiac Catheterization Technique

1. The left femoral arterial and venous sheaths are placed. Because a pigtail catheter will be left in place in the left

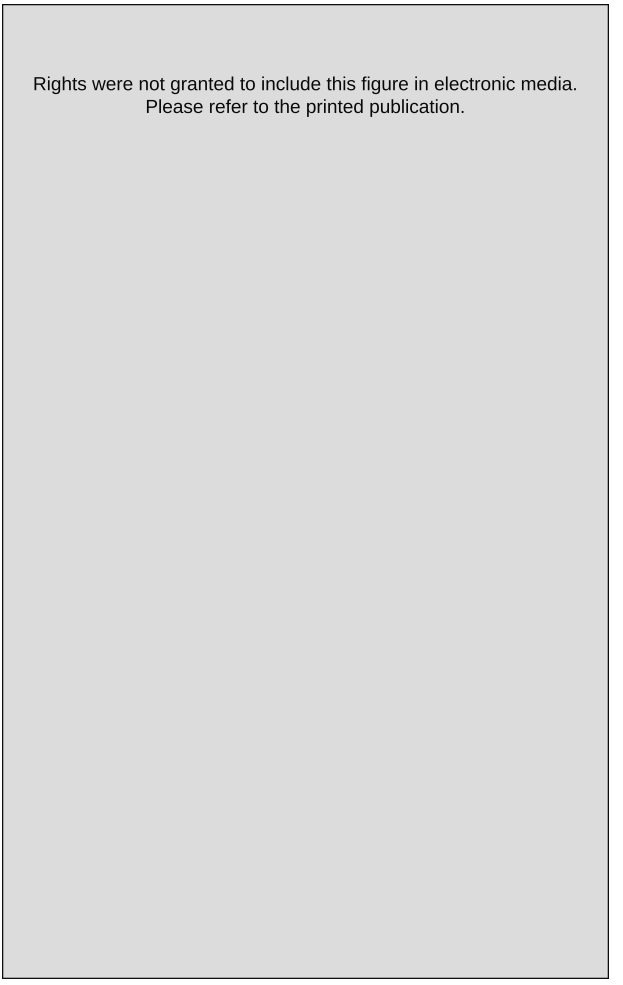

Fig. 12-3 **A,** Long-axis two-dimensional echocardiographic image from an ideal candidate for mitral commissurotomy. The solid arrows show the thin, domed leaflets. The mitral apparatus is not visible in the left ventricle, signifying its freedom from significant thickening. **B,** Typical valve replacement candidate. The solid arrow points at a thickened and calcified anterior mitral leaflet. The open arrows show the thickened submitral apparatus. These patients have echocardiographic scores between 8 and 12 and are reasonable candidates for percutaneous transvenous mitral commissurotomy, although they may have poorer long-term freedom from mitral valve replacement. **C,** Elderly patient, not a likely candidate for surgical therapy of any kind. This patient was an 88-year-old woman. The solid arrow shows a

ventricle for a relatively long period of time, we prefer to use 5 or 6 French arterial catheters.

2. A multilumen pulmonary artery balloon catheter with thermodilution cardiac output capability is used for right heart catheterization. Left femoral access is preferred for these catheters, leaving the right side for insertion of the dilatation balloon catheter. Pulmonary artery catheters with oximetric monitoring greatly simplify the evaluation of venous saturations for the detection of atrial shunting following the procedure, although these catheters are often more difficult to place than more conventional pulmonary artery catheters. When significant right atrial dilatation is present, passage of the pulmonary artery catheters are facilitated by the use of an extra stiff 0.025 inch guidewire.
3. Left ventriculography and coronary arteriography are performed when indicated. The AHA/ACC guidelines for valvular heart disease recommend arteriography for men over age 35 years, or women with risk factors over age 35 years.
4. Right heart pressures and cardiac output are measured.
5. Right femoral venous puncture is performed for placement of an 8 French Mullins sheath. An ipsilateral pulmonary artery catheter does not interfere with the performance of the transseptal catheterization.
6. Following transseptal puncture heparin is administered. When the transseptal catheterization has been completed successfully, the transmitral pressure gradient is measured using the Mullins sheath for the left atrial and the pigtail for left ventricular pressures. If the Mullins sheath can be passed into the left ventricle with a gentle counterclockwise rotation, a transaortic gradient is measured with the Mullins and pigtail to exclude aortic valve disease.

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densely calcified, thickened, and rigid anterior mitral leaflet. The open arrow points at the similarly thickened and calcified mitral apparatus. Balloon dilatation may be accomplished successfully in these patients but with acute results that are not as good as those in patients with less deformed valves. The long-term event-free outcome for these patients is poor. (From Feldman T, Carroll JD. Cardiac catheterization, balloon angioplasty, and percutaneous valvuloplasty. In: Hall JB, Schmidt GA, Wood LDH, eds. *Principles of critical care*. New York: McGraw-Hill, 1992:343–360.)

A simplified procedure with no arterial access and no pulmonary artery catheterization is not recommended. The safety of the procedure and the evaluation of the resultant possible complications mandate continuous arterial pressure monitoring, a full right heart catheterization before and after the procedure, and accurate cardiac output determination.

Selection of Balloon Size. Balloon sizing has not been rigorously defined by any study; rather a combination of experience in the first decade of mitral valvotomy, common sense, and data from a few studies has established the approach to balloon sizing.

The maximum expected inflated balloon diameter may be selected based on the patient's height (Table 12-3). This value provides a guideline for balloon selection with a stepwise technique. A first inflation is always performed at a diameter smaller than the maximum possible for the selected balloon. An initial inflation of 2–4 mm less than the maximum is usually chosen. An alternative method for selecting balloon size is to calculate the ratio of inflated dilating balloon area to the body surface area, called the effective balloon dilating area (EBDA).

This method results in somewhat different values for maximal balloon size in a given patient compared to the recommendations originally made by Inoue based on his empiric observations. When the Inoue balloon is inflated to an EBDA of 4.0 using a single inflation without the stepwise technique, results similar to those reported for the stepwise technique have been achieved. EBDA may not be equally useful in all patient populations. For overweight or obese patients, in particular, a better estimate of largest expected

Table 12-3

Selection of Balloon Size for Percutaneous Transvenous Mitral Commissurotomy

Balloon diameter (range, mm)	Balloon dilating area (cm^2)	Patient height, cm (inches)
26 to 30	7.07	>180 (70.9)
24 to 28	6.16	>160 (62.9)
22 to 26	5.13	<160

balloon inflation balloon diameter may be based on height alone.

For patients with pliable valves, the first balloon inflation can be made with a balloon 2 or 3 mm smaller than the reference size and then increased in increments of 1 mm until either a maximal diminution of gradient has occurred or mitral regurgitation has begun to worsen significantly. For patients with more deformed valves, the first inflation can be performed at 4 mm less than the reference size with increments of 1 mm in size while the balloon is in the shallow or low-pressure portion of the pressure–volume cure. Increments of 0.5 mm may then be used over the last couple of millimeters of balloon diameter when the balloon reaches the high-pressure portion of its pressure–volume curve.

Special Considerations. There are a number of special considerations in balloon size selection. Smaller balloons than initially estimated may be useful in patients with advanced age, patients with subvalvular disease, or those in whom persistence with marked constriction during full inflation indicates that balloon pressure may be insufficient. In this last situation, the use of a smaller balloon inflated to the same diameter as a previous larger balloon will result in a greater inflation pressure.

The Stepwise Technique. The stepwise balloon expansion technique obviates the need for precise determination of maximum inflated balloon diameter. Because the Inoue device may be inflated over a range of sizes, balloon size selection need only be accurate enough to address this range. Although patient and balloon characteristics may be evaluated in a consistent manner, the inhomogeneity of the valve pathology and the limitations of our ability to predict ultimately what balloon sizes and pressures will produce commissural splitting make the stepwise approach more practical than preprocedure predictions of expected balloon size.

Balloon Preparation. Once the diagnosis of mitral stenosis is confirmed after successful transseptal puncture, the balloon catheter can be prepared. The balloon catheter comes packaged with all the components necessary for the dilatation procedure

(Fig. 12-4). These include:
- A balloon-stretching metal tube
- A calibrated inflation syringe specifically matched to each balloon
- A rigid 12–14 French plastic dilator
- A 0.025 inch spring tip exchange guidewire
- A stylet for manipulating the balloon across the mitral valve after it has been placed in the left atrium
- The Inoue balloon catheter
- Calipers for measuring the balloon diameter and confirming its inflated size.

The balloon catheter lumen is flushed with saline. Dilute contrast (saline:contrast 2:1 or 3:1) is injected through the vent lumen to purge air from the inflate/deflate channel to the balloon and the stopcock is closed on that lumen.

The precalibrated balloon inflation syringe is filled to the calibration corresponding to the smallest inflated diameter. After connecting the inflation syringe to the inflation port and checking that all connections are secure, the balloon is slowly inflated over a period of 5 sec so that the nylon mesh may be slowly stretched without risking mesh rupture.

The balloon is allowed to deflate passively in a bath of flush solution. Small bubbles will escape from within the mesh layer of the balloon. The balloon is then inflated rapidly and the inflated diameter is measured using calipers to verify the precalibrated inflation syringe. If the balloon does not inflate to the desired diameter, small amounts of contrast are added or subtracted to achieve proper calibration.

The syringe is then filled to the calibration corresponding to the maximum nominal inflated size. The balloon may be tested to insure that the maximum size calibration is also correct. In practice, this calibration step is often omitted. The next step in balloon preparation is to elongate the balloon catheter along its long axis, causing it to become more slender. A metal tube (balloon-stretching tube) is inserted into the center lumen of the balloon over the guidewire and advanced until it locks into the metal hub at the proximal end of the balloon catheter. The balloon and stretching tube are then advanced into the balloon catheter shaft until they engage the plastic slot on the balloon catheter Luer lock. This leaves the balloon in its elongated, slenderized form to ease not only

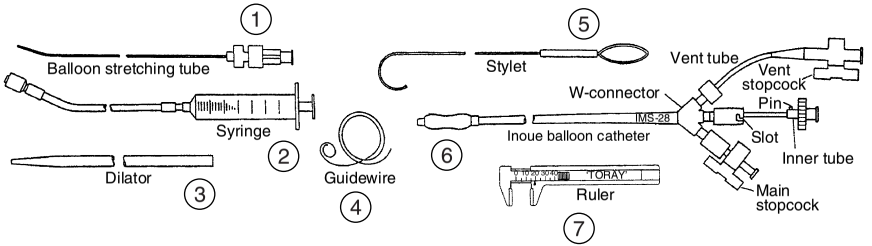

Fig. 12-4 The components of the equipment box supplied by Toray include: (1) a long metal hypotube, the balloon-stretching tube, used to pass through the inner tube of the Inoue balloon catheter to elongate and slenderize the balloon; (2) a calibrated syringe used for inflation of the balloon. The syringe provides calibration marks so that predetermined diameters of the balloon can be achieved driven by the volume rather than pressure; (3) a dilator used to dilate the subcutaneous tissue at the femoral venous puncture site, and to dilate the septum as well; (4) a 0.025 inch stainless steel spring guidewire; (5) a steering stylet that is introduced through the inner tube after the balloon is in the left atrium to help guide it across the mitral valve; (6) the balloon catheter itself, which has a W connector from which arise a vent tube, an inner tube used for introduction of the balloon stretching tube and stylet, and for stretching the balloon, and a main stopcock for balloon inflation via the syringe; (7) a ruler or caliper, which is used to confirm that the graduations on the syringe used to inflate the balloon result in the desired inflation diameters.

percutaneous insertion but also delivery across the interatrial septum.

Balloon Valvotomy. The major steps in valvotomy are illustrated diagrammatically in Figure 12-5. The 0.025 inch spring guidewire is advanced through the Mullins sheath into the left atrium with the fully coiled distal portion out of the sheath and positioned in the roof of the atrium. The Mullins sheath is withdrawn over the guidewire with the guidewire remaining in the left atrium. The dilator is advanced through the skin and then into the atrial septum, where it may be passed through the septal puncture as shown in Figure 12-5. The dilator is left sitting in the septal puncture for several seconds to stretch the septal tissue. The dilator is removed and the balloon catheter is passed over the guidewire directly through the skin and then across the atrial septum. Resistance at the skin is common but may be overcome by twisting the balloon catheter so that the angled tip finds its way through the subcutaneous tissue. Care must be taken to not unlock the metal Luer lock connection while passing the catheter through the skin. A 14 French sheath may be used if direct insertion through the skin is not feasible or too much resistance is encountered.

After the balloon is passed through the atrial septum, it must be allowed to resume its unstretched conformation to prevent the very stiff slenderizing tube from puncturing the roof of the left atrium. In some cases the blunt tip of the balloon will catch on the right atrial side of the septal puncture. Rotating the catheter slowly with gentle probing pressure will allow it to find its way through the septal puncture into the left atrium. After the tip of the balloon has passed across the atrial septum, the stretching metal tube is then disengaged from the catheter metal hub and withdrawn as the balloon catheter is advanced. The tip of the balloon will then begin to track around the coiled spring guidewire. As the balloon reaches the roof of the left atrium, the gold metal Luer lock is disconnected and pulled back, allowing the balloon to shorten. The balloon catheter is then advanced further over the spring-tipped guidewire. The balloon stretching tube and spring guidewire are removed from the patient and cleaned and prepared for later use to remove the balloon. It is important to track the balloon around over the wire until it

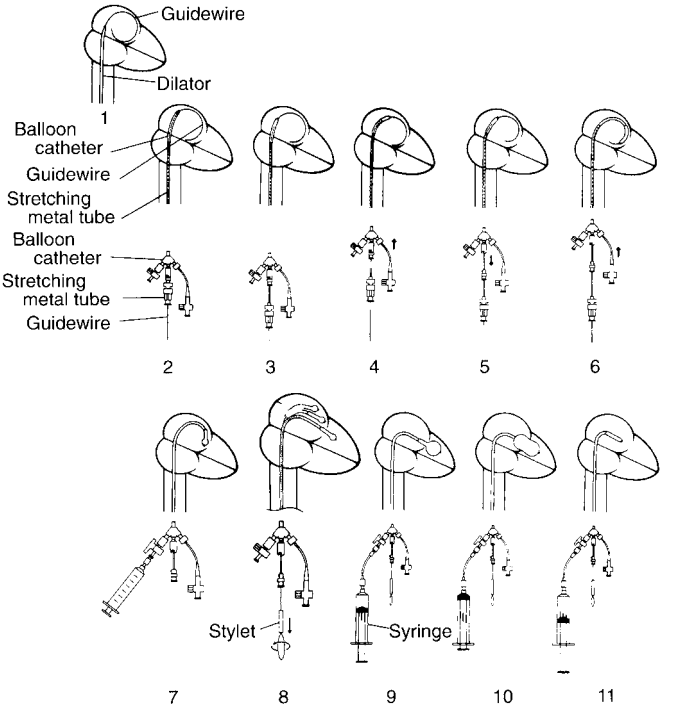

Fig. 12-5 Schematic illustration of the Inoue balloon mitral valvotomy procedure. (1) After a spring guidewire is introduced via a Mullins sheath into the left atrium, the interatrial septum is dilated using a rigid 14F plastic dilator. (2) The elongated balloon catheter is advanced over the wire through the interatrial septum. (3) The stretching metal tube is partially withdrawn, allowing the balloon to shorten and curl within the left atrium. (4) The balloon is advanced through the interatrial septum. (5) The stretching metal tube and balloon straightening device are withdrawn further. (6) The balloon is advanced beyond the mitral orifice. (7) The distal portion of the balloon is partially inflated with a contrast-saline mixture. (8) With counterclockwise rotation of the stylet, slight advancement of the catheter shaft, and withdrawal of the stylet, the balloon is directed through the mitral orifice and left ventricle. (9) The partially inflated balloon is withdrawn against the mitral orifice. (10) The balloon is fully and rapidly inflated and allowed to deflate. (11) After deflation, in most instances, the balloon passively returns to the left atrium from the left ventricle. (Courtesy of Toray, Inc., Tokyo, Japan.)

reaches the inferior portion of the left atrium so it overlies the mitral orifice before removing the wire.

The balloon catheter can be flushed and connected to a pressure transducer. The transmitral pressure gradient can be remeasured through the balloon catheter to verify that the pressure wave form is similar to that obtained through the Mullins sheath. The wave form appears slightly damped through the lumen of the Inoue balloon.

Before crossing the mitral valve with the balloon, it is useful to change the x-ray projection from straight anteroposterior to shallow right anterior oblique.

The distal half of the balloon is partially inflated and, once positioned in the left ventricle, the balloon is gently withdrawn until the mitral valve is engaged. The proximal half of the balloon is inflated. When the position of the balloon appears correct, full inflation is achieved. The balloon is allowed to deflate passively. This ensures a constant amount of dead space fluid for subsequent balloon inflations. The entire cycle of inflation and deflation takes 5 sec or less and it is unusual for patients to sense the inflation, as frequent ventricular ectopy does not usually occur and hypotension persists for no more than a few cardiac cycles.

To cross the mitral valve, the tip of the balloon is inflated and the steering stylet is passed into the catheter for its full length. It is important to completely advance the steering stylet within the shaft of the catheter. The stylet is then rotated in a counterclockwise direction as the balloon catheter is advanced and withdrawn over a 2–5 cm range, allowing the tip of the balloon to find its way across the mitral valve in a manner similar to that in which a pulmonary artery flotation catheter crosses from the right atrium through the tricuspid orifice into the right ventricle. As the balloon passes across the mitral orifice, the stylet is withdrawn about 5–10 cm. The balloon must be advanced gently and moved forwards and backwards to make sure it is free of entanglements in the subvalvular apparatus. The stylet may be bent to accentuate its curve to facilitate passage of the balloon across the mitral valve if initially crossing the valve is very difficult (Fig. 12-6). A useful observation during balloon inflation is the "popping sign" denoting splitting of one or both commissures. During the final portion of the inflation, one observes the inferior or superior margin of the mid section of the balloon suddenly

popping outward. This is a welcome sign that shows a clear decrease in gradient due to the commissurotomy.

After dilatations, as the balloon deflates, it usually falls back into the left atrium with no specific manipulation. If it does not, a gentle clockwise rotation of the balloon catheter will move the balloon back into the left atrium. The stylet is withdrawn and the balloon shaft is connected to a pressure transducer for reassessment of the transmitral pressure gradient.

A Doppler and echocardiographic examination can be performed to evaluate changes in mitral regurgitation and assess whether either fused commissure has been opened, as is best seen in a short-axis view.

Stepwise Balloon Inflation. If a transmitral gradient persists and no significant increase in mitral regurgitation has occurred, another balloon inflation is performed at an inflated diameter 1 mm greater than the preceding inflation, as shown in Figure 12-7. This sequence is repeated until either an increase in mitral regurgitation or a sufficient decrease in the transmitral gradient occurs. Balloons can be over-inflated by 1–2 mm diameter by using an additional 1–2 ml inflation volume above the maximal calibrated balloon size. If sufficient reduction in gradient is not achieved after maximal or supermaximal inflation, a larger balloon size can be used.

It is very useful to monitor the effect of each balloon inflation in the mitral valve by echocardiography in the catheterization laboratory. Of course, the procedure may be performed without this adjunct but monitoring of the results is facilitated by echo examination. An in-lab Doppler examination will demonstrate if mitral regurgitation is increased. More importantly, the short-axis two-dimensional examination will show the degree of commissural separation (Fig. 12-8). Note the degree of commissural fusion in the short-axis examination before the balloon dilatation. Separation of one commissure while the other remains fused will facilitate the decision to proceed with further balloon inflations. If mitral regurgitation is not worsened, attempts to complete commissurotomy may be pursued. Conversely, if one commissure is opened completely and a significant amount of mitral regurgitation has developed, this will signify at least an adequate result.

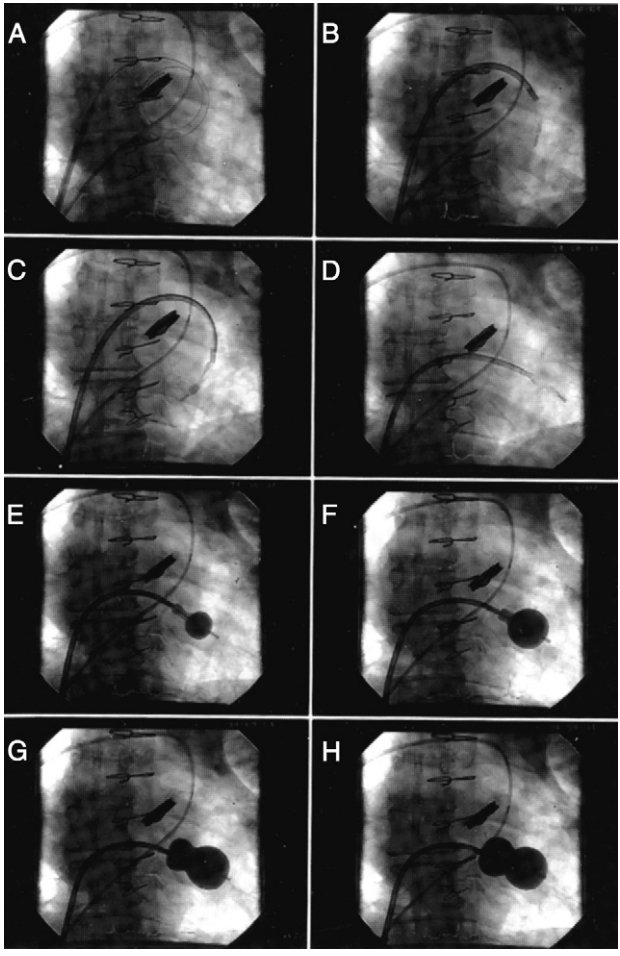

Fig. 12-6 The major steps in percutaneous transvenous mitral commissurotomy. **A,** A 14F dilator is advanced over a spring-coiled guidewire. The guidewire has been introduced into the left atrium via a transseptal puncture. The 14F dilator dilates both the subcutaneous tissue at the groin catheter insertion site and the left atrial puncture. A prosthetic aortic valve marks the location of the aortic root. A pulmonary artery catheter traverses the right atrium, right ventricular outflow, and pulmonary artery. **B,** The uninflated balloon catheter has been introduced over the course of the spring wire. The wire has been removed. The tip of the catheter overlays the mitral orifice. **C,** The tip of the balloon catheter is partially inflated so that it may be manipulated across the mitral valve using a steering stylet. This is analogous to crossing the tricuspid valve from balloon tip catheter. **D,** The uninflated

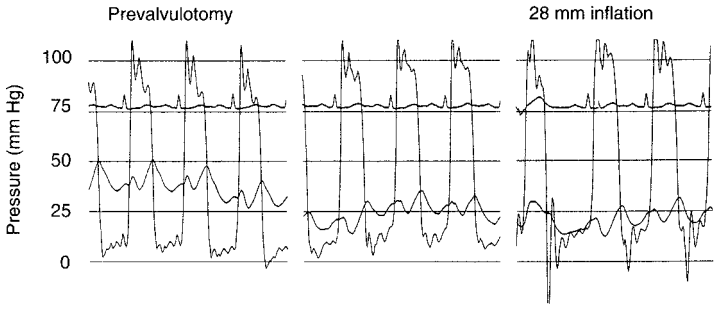

Fig. 12-7 Stepwise dilatations result in a progressive decline in left atrial pressure and transmitral pressure gradient. In the prevalvulotomy tracing, the mean left atrial pressure is well over 25 mm Hg and the gradient is extreme. After a 27 mm diameter balloon inflation, the transmitral gradient and left atrial pressure have declined significantly. On the right, after only 1 mm increment in inflated balloon diameter, there is dramatic improvement in the transmitral gradient. (From Feldman T, Herrmann HC, Inoue K. Technique of percutaneous transvenous mitral commissurotomy using the Inoue balloon catheter, *Cathet Cardiovasc Diagn Suppl* 1994;2:26–34.)

The transmitral pressure gradient is the simplest parameter to monitor between balloon inflations. The absolute level of left atrial pressure is extremely important as well. In general, if the left atrial pressure remains constant or decreases after successive balloon inflations, mitral regurgitation has not yet become limiting. When the mean left atrial pressure rises following balloon inflation, even if a large V wave has not occurred, mitral regurgitation may have worsened significantly. If in-lab echocardiography is not available and left atrial pressure is increasing, a repeat left ventriculogram should be performed. The decision to proceed with further balloon inflations is among the most difficult to make. Use of all

balloon is now in the left ventricular apex. **E,** The front portion of the balloon has been inflated and pulled back until it engages the mitral valve orifice. **F,** The balloon is inflated further. **G,** Additional inflation of the balloon causes the proximal portion to inflate, leaving a waist in the middle. **H,** Full inflation of the balloon results in expansion of the center of the balloon, splitting the fused mitral commissures.

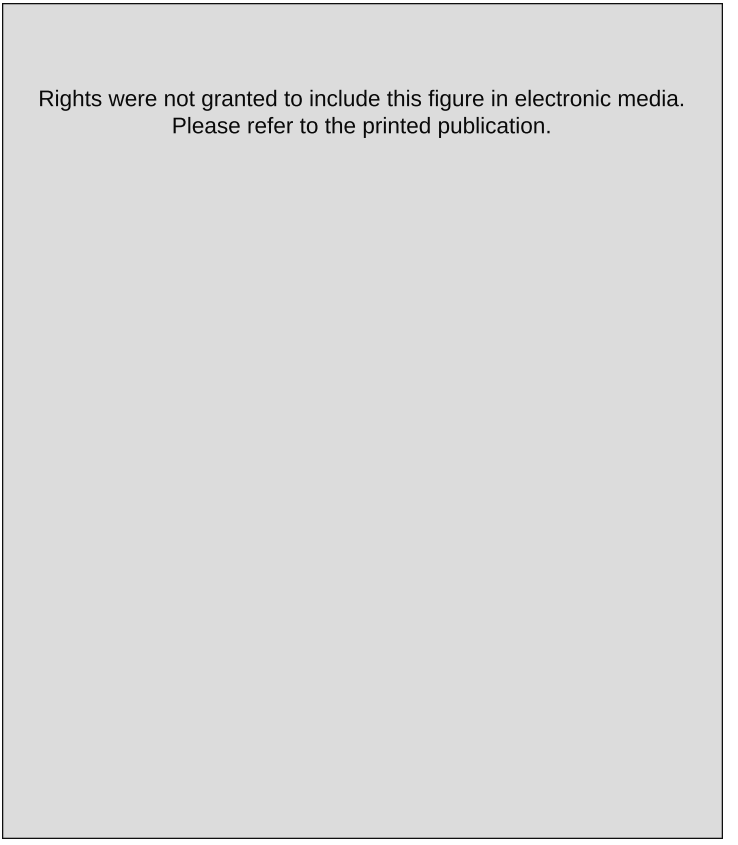

Fig. 12-8 Short-axis echocardiogram illustrating commissural splitting following balloon dilatation. **A,** Fishmouth orifice of the mitral valve. **B,** Bilateral commissural splitting, indicated by solid white arrows. (From Feldman T, Carroll JD: Cardiac catheterization, balloon angioplasty, and percutaneous valvuloplasty, in Hall JB, Schmidt GA, Wood LDH, eds: *Principles of critical care*, New York, 1992, McGraw-Hill, pp 343–360.)

available information, including two-dimensional and Doppler echocardiography, left atrial pressure and wave form (Fig. 12-8), auscultation, and ventriculography, is important. Hemodynamics before and after Inoue Balloon valvuloplasty are shown in Figure 12-9.

Technical Considerations. If the interatrial septum is crossed in a relatively superior or anterior location, passing the balloon

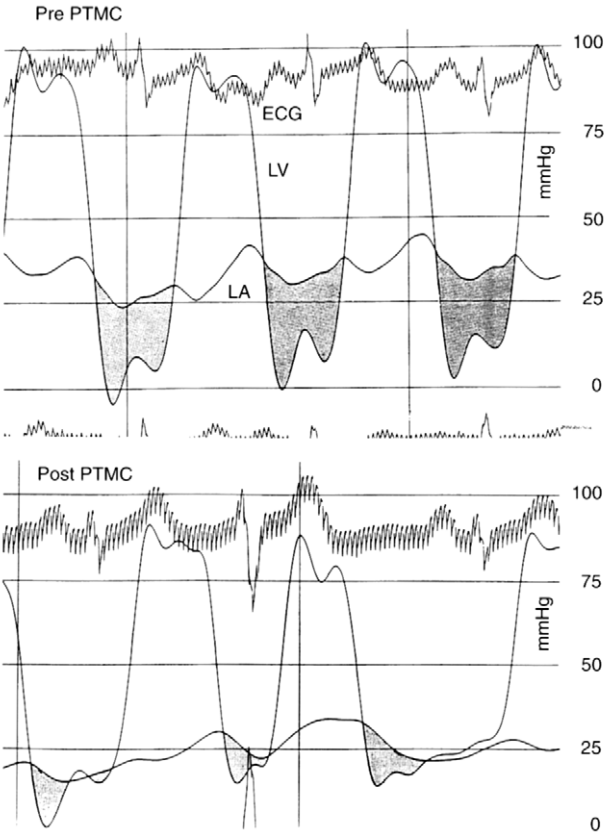

Fig. 12-9 Transmitral valve pressure gradients before and after percutaneous transvenous mitral commissurotomy (PTMC). In addition to evaluating the transmitral gradient, it is important to consider the magnitude of left atrial pressure and changes in left ventricular filling pressure. In this case, post-PTMC there is only a modest decline in left atrial pressure. Left ventricular filling pressure has risen significantly. In addition, the atrial fibrillation heart rate becomes irregular. These factors all can make evaluation of the success of a PTMC procedure difficult.

across the mitral valve may be difficult. In this circumstance, clockwise rather than counterclockwise rotation of the stylette will "bank" the balloon off the posterior atrial wall and allow it cross into the left ventricle after making a loop, as seen in Fig. 12-10. This alternative approach is sometimes limited by the short length of the balloon catheter shaft.

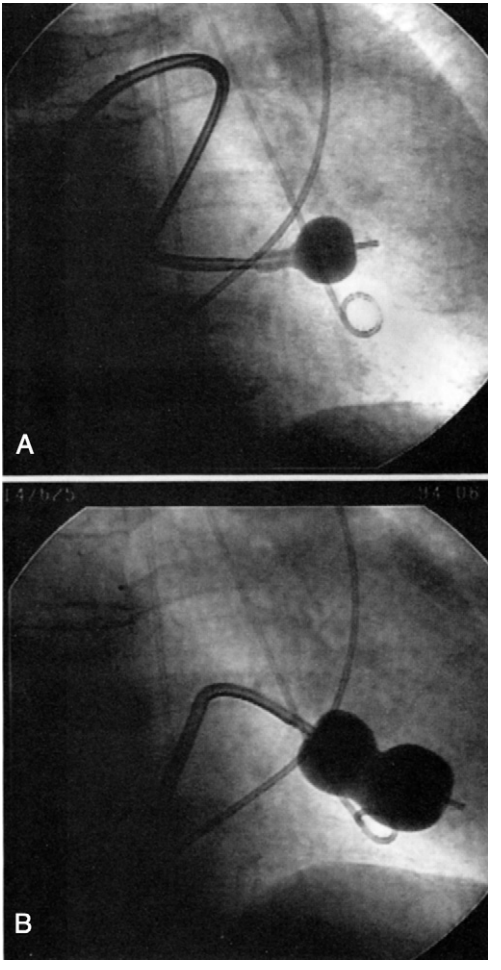

Fig. 12-10 When the balloon catheter will not cross the mitral valve using the conventional method, the catheter may be rotated clockwise and manipulated across the mitral valve using an alternative approach. The balloon is introduced into the left atrium in the usual manner and guided past the mitral orifice. With clockwise rotation of the stylet and catheter shaft, a loop is made directing the balloon off the posterior left atrial wall (**A**). Withdrawal of the stylet and advancement of the catheter shaft direct the balloon catheter across the mitral valve into the left ventricle (**B**).

In the event the balloon is withdrawn toward the septal puncture during manipulations across the mitral valve, it is sometimes necessary to reinsert the coiled-spring-tipped guidewire into the left atrium to be able to advance the balloon catheter toward the mitral valve. This is particularly true when the septum is markedly thickened and the septal dilatation does not result in free movement of the balloon catheter shaft through the atrial septum.

Occasionally the catheter shaft may be seen to be pinched or bound by the atrial septum. Advancing the shaft of the catheter may cause it to buckle in the right atrium without causing the balloon to move forward in the left atrium toward the mitral valve. Redilatation of the interatrial septum with a 14 French dilator or even a 6–8 mm diameter peripheral angioplasty balloon may sometimes be necessary.

Free movement of the balloon can be impaired by binding in the subcutaneous tissues at the groin puncture site. In very heavy patients the catheter may make a severe angle between the skin and the femoral vein. The use of a 14 French sheath in these situations will facilitate accomplishment of the procedure.

Postprocedure Evaluation and Balloon Withdrawal. Following dilatation a left ventriculogram is repeated to evaluate mitral regurgitation. Cardiac output measurement is repeated while the balloon catheter remains across the interatrial septum. It is important to leave the atrial septum occluded because withdrawal of the balloon might allow shunt flow across the atrial septal puncture site with a spurious increase in cardiac output. This has been demonstrated to yield valve area results that are falsely elevated.

The balloon catheter must then be withdrawn across the atrial septum. This is accomplished by reintroducing the balloon stretching tube, which has been preloaded with the 0.025 inch spring-tipped guidewire. The guidewire is advanced and curled in the left atrium. The balloon stretching tube is then locked to the gold metal hub of the balloon catheter. These two metal units are then advanced together into the plastic Luer lock to stretch the balloon. Special care must be taken not to stretch and stiffen the balloon through the roof of the left atrium. This is best accomplished by withdrawing the balloon backwards onto the stretching metal tube and then

withdrawing the plastic Luer lock onto the assembled metal hub apparatus. The balloon catheter is thus pulled back across the atrial septum as it is stretched and elongated rather than pushing the stretching metal tube forward through the septum. The balloon and wire can then be withdrawn from the left atrium. The wire is best removed while the stretched balloon is partly across the septum to avoid any "slicing" action of the wire on the septal puncture site, which can enlarge the septal defect.

The balloon can be left in the inferior vena cava until it is ready to be withdrawn for hemostasis. It may also be replaced with an 11 or 12 French sheath to facilitate patient movement and sheath removal. Finally, oximetry may be repeated to evaluate left-to-right shunting across the atrial septal puncture site.

Atrial Shunting. Following double balloon mitral valvotomy the occurrence of some left-to-right shunting results in a spurious increase in cardiac output and an artificially increased Gorlin mitral valve area calculation. Because the final cardiac output measurements after Inoue balloon PTMC are made while the shaft of the catheter remains across the atrial septal puncture, the magnitude of this spurious increase and calculated mitral valve area is insignificant. Use of a small balloon catheter to occlude the interatrial septum following balloon dilatation is not necessary for accurate post-PTMC valve area measurements with the Inoue catheter.

The best method for measurement of valve area following catheter commissurotomy is probably planimetric echo determination. This is ideally performed 24 hours post-procedure. Doppler estimates are highly variable because of the large swings in left atrial compliance and cardiac output associated with the acute changes of the valve dilatation procedure.

Recognition and Management of Complications

The two most serious complications of PTMC are cardiac tamponade and acute mitral regurgitation. Recognition of pericardial tamponade requires a high degree of suspicion on an ongoing basis. It is important to assess any chest, shoulder, or back pain of which the patient may complain during the procedure. Continuous attention to the pulsatile

fluoroscopic cardiac borders is important. Make use of right heart pressures as well. An intraprocedure echo is essential when the patient is hemodynamically unstable. It is not reasonable to perform PTMC without the ability to perform pericardiocentesis as well.

Acute mitral regurgitation is easily recognized by the left atrial pressure magnitude and wave form. It occurs in some cases even with a single balloon inflation or sometimes a 0.5 mm diameter increment in balloon inflation size. Confirmation of the diagnosis with ventriculography is important. Nitroprusside therapy or intravenous nitroglycerin are the mainstays of immediate management for this hemodynamic complication. Between 1% and 2% of patients undergoing PTMC will develop severe acute mitral regurgitation requiring valve replacement as a result of the procedure.

Post-Procedure Care and Sheath Removal

The left femoral 5 French arterial sheath and the right femoral large venous sheath do not require any special management. Sheaths may be removed when the activated clotting time falls below 180 sec. Younger patients with excellent hemodynamic results can be sent home on an outpatient basis at the end of the day of the procedure. For patients who require warfarin therapy, it is our usual practice to reinstitute warfarin therapy 1 or 2 days post-PTMC without a loading dose. In patients who are at special risk for thrombosis or a history of prior thrombotic episodes, heparin therapy or outpatient subcutaneous low-molecular-weight heparin therapy following sheath removal and a transition to warfarin therapy may be necessary.

The availability of femoral closure devices has facilitated the rapid discharge of patients who are candidates for this procedure on an outpatient basis. In some cases we have used percutaneous suture closure for the arterial puncture and both venous punctures in these patients.

AORTIC VALVULOPLASTY

Balloon aortic valvuloplasty showed great promise as an alternative to surgical aortic valve replacement when the procedure was initially described in the early 1980s. Balloon dilatation of the aortic valve results in an immediate increase in aortic valve area with the expected fall in the transvalvular

pressure gradient and a rise in cardiac output. Most patients have immediate clinical improvement and this is accomplished with a percutaneous procedure resulting in substantially less morbidity than valve replacement surgery. Unfortunately it was quickly discovered that the durability of these results is short-lived. Disappointment with the clinical results of this procedure over a 1–2-year followup resulted in a pendulum-like movement away from the performance of balloon aortic valvuloplasty. However, a number of important clinical indications still exist for this procedure.

Indications

There are currently five clinical situations in which balloon aortic valvuloplasty is useful.

- Patients who present with aortic stenosis and cardiogenic shock may be stabilized for the short term. Balloon dilatation can be accomplished at the same session as diagnostic catheterization and further decisions regarding therapy can be made after the patient has stabilized.
- Among patients with severe left ventricular dysfunction or shock in whom aortic valve replacement is planned, balloon dilatation may be performed to allow improvement in left ventricular performance before surgery. This can reduce a major comorbid factor for valve replacement surgery because left ventricular function is directly associated with surgical mortality. In addition, these patients frequently have prerenal azotemia associated with their medical therapy and balloon dilatation allows this to improve.
- Patients found to have aortic stenosis during the evaluation for major noncardiac surgery may undergo valvuloplasty. This is especially useful for patients with malignancies.
- Hospital-bound patients with severe aortic stenosis who are not candidates for valve replacement surgery may undergo balloon dilatation with successful short-term improvement. This is useful for patients who are dependent on intravenous pressors and in an intensive care unit. Although valvuloplasty does not improve their long-term prognosis, it may allow them to be transferred to a regular floor or discharged from the hospital so that they may have a better quality of life, at least in the short term.

- There is a group of patients in whom balloon valvuloplasty may be performed as a diagnostic test. This is useful when the valve area is between 0.8 and 1.0 cm^2 with low cardiac output and a low transvalvular pressure gradient. In this group of patients the severity of valvular stenosis is especially difficult to ascertain. Poor ventricular function has made therapy in this group difficult. In the past, valve replacement could be performed and if the patient had improvement in left ventricular function then survival was good. Unfortunately, for those patients who did not show improvement in left ventricular performance, perioperative mortality was very high. Balloon dilatation may be performed and serial echocardiography used to monitor changes in left ventricular function. If symptoms and left ventricular performance improve with opening of the aortic valve using valvuloplasty, later valve replacement surgery can be undertaken with a high expectation of long-term success.

Bioprosthetic Aortic Valves. One special group is patients with bioprosthetic stenosis. *In vitro* evaluation of balloon dilatation for stenotic bioprosthetic valves has been disappointing. Prosthetic tissue is often friable and is frequently not severely calcified but the potential for leaflet perforation or evulsion is significant in this group of patients. This balloon dilation of bioprosthetic aortic valves is infrequently performed.

Results of Balloon Dilatation for Aortic Stenosis

The aortic valve area usually increases between 80% and 100% after valvuloplasty. The transvalvular pressure gradient declines by more than 50%. Postdilatation valve area ranges between 0.7 and 1.1 cm^2. An increase in valve area of 0.5–0.7 cm^2 or more will be associated with dramatic clinical improvement in most patients. Predilatation valve areas greater than 0.5 cm^2 may ultimately yield postdilatation valve areas of 1 cm^2 or more. It is notable that prosthetic aortic valves have an area between 0.9 and 1.2 cm^2, especially in small women with small aortic annuli.

The greatest limitation of balloon aortic valvuloplasty is the almost inevitable occurrence of restenosis following dilatation. The majority of patients have anatomic and symptomatic restenosis between 6 and 18 months after the procedure. Survival is not clearly improved with aortic valvuloplasty. The

mechanism of restenosis may be related to the mechanism of relief of aortic stenosis. The majority of these elderly patients have calcific tri-leaflet aortic stenosis with calcification and thickening of the valve cusps and no commissural fusion. The calcium deposits are acellular and nodular. Histologically the nodules are encased densely in fibrous tissue. This explains the striking lack of embolization during this procedure. After balloon dilatation small fractures or cracks may be seen in the calcified nodules. This allows increased leaflet mobility due to the presence of many "hinge points" or fissures. The restenosis process probably involves regrowth of granulation tissue, fibrosis, and possibly true ossification of these fissures. This active process of restenosis follows a time course that is consistent with new scar formation.

Technique of Aortic Valvuloplasty

In most cases diagnostic coronary arteriography and ventriculography are performed immediately before balloon dilatation. It is unusual to encounter a new patient with aortic stenosis who has not had adequate echocardiographic evaluation before catheterization. An assessment of aortic insufficiency is usually accomplished echocardiographically prior to cardiac catheterization, obviating the need for routine aortography. Single-session diagnostic and therapeutic catheterization procedures may decrease morbidity in this very elderly and ill population.

Arterial Access. It is important to place the femoral puncture comparatively high (cranial) so that the large sheath necessary for valvuloplasty will not be inserted into a branch vessel. Laying a hemostat or thin-walled needle on the femoral crease and using fluoroscopy to locate the mid femoral head prior to puncture helps with accurate puncture placement. The puncture should be somewhere in the middle third of the femoral head to have the greatest chance of a common femoral artery puncture. After the puncture is accomplished and before placing a sheath it is our practice to examine the course of the wire fluoroscopically. If the ileofemoral system is extremely tortuous, we may pass a wire on the contralateral side and choose the straighter course for sheath placement and eventual passage of the balloon. Lower abdominal angiography may be

helpful. Sheath angiography is always performed prior to insertion of the large sheath. A 6 or 8 French sheath can be inserted in the arterial system after the initial puncture and sheath angiography will verify that it is in the common femoral artery rather than a branch. Placing a 12 or 14 French sheath in the superficial or profunda femoris can cause complications.

Heparin. Heparinization is recommended at a dose of 5000 units for the average-sized patient. Supplemental doses will only be necessary for prolonged procedures. An activated clotting time between 220 and 275 sec is usually our goal. Early sheath removal is important in this elderly population, so excessive anticoagulation is to be avoided.

Retrograde Technique. After the transvalvular gradient and cardiac output determinations confirm the presence of severe aortic stenosis, the arterial sheath is exchanged for a 12.5 French sheath. This exchange is performed over an extra stiff 0.038 inch guidewire to minimize the chance of the large arterial dilator perforating the iliac vessels. The sheath is exchanged over a 360 cm extra stiff wire that is left curled in the left ventricular apex. To maximize the safety of the tip of this wire in the left ventricle, a "ram's horn" curve is put on the end of the guidewire (Fig. 12-11). This is done by grasping the wire between the thumb and the edge of a curved hemostat and pulling along the wire rapidly in the same manner one uses to put a curl on gift wrapping ribbon. After the sheath has been exchanged and flushed, it is connected to arterial pressure, and a 20 mm diameter × 5.5 cm long valvuloplasty balloon catheter (Mansfield, Inc., Watertown, MA; Boston Scientific) is passed over the wire and across the aortic valve. When the aortic annulus diameter on echo is less than 20 mm a smaller balloon is used. Occasionally a 15 or 18 mm diameter balloon may be used initially to facilitate crossing an exceptionally narrowed valve outface.

Balloon Inflation. The technique of balloon inflation in the aortic valve is especially important. The balloon is positioned midway across the valve. When ventricular function is poor, maintaining valve position may be very simple. A dynamic or vigorous left ventricle will typically eject the balloon during

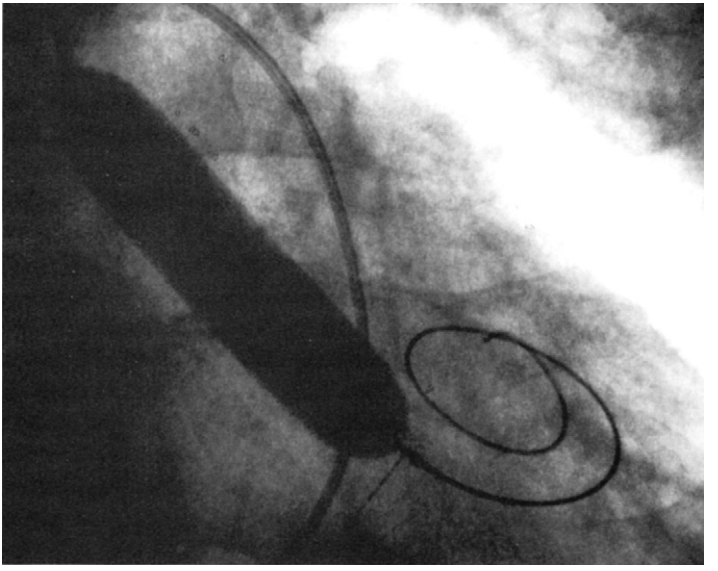

Fig. 12-11 A 5.5 cm long, 20 mm diameter Mansfield aortic valvuloplasty catheter. The extra-support exchange guidewire has been looped into a ram's horn configuration by pulling it over the end of a hemostat as one would curl ribbon when wrapping a package.

attempts to inflate it. Substantial forward pressure may be necessary to maintain the position of the balloon in the valve in that case.

Initially the balloon is inflated via a high pressure stopcock using a 60 ml syringe partially filled with dilute saline and contrast mixture. The dilute mix (seven parts of saline to one part of contrast) minimizes the viscosity of the solution while at the same time maintaining fluoroscopic visibility. In addition, high-osmolarity conventional contrast is less viscous than low osmolarity. A 10 ml syringe filled with contrast mixture is placed on the sidearm of the high pressure stopcock used for inflating the balloon. After the 60 ml syringe has been used to inflate the balloon as much as possible, the operator flips the stopcock so that the smaller syringe can be used to inject additional saline/contrast mixture under very high pressure. This "boost" in inflation is very important to achieve maximal balloon expansion. The balloon can be appreciated

to completely expand or to "plump" out along its sides when this is done. Adequate valve dilatation is usually not achieved unless this can be accomplished. Hypotension and ventricular tachycardia are typical during balloon inflations (Fig. 12-12).

As soon as balloon deflation commences, the balloon can be pulled back in the aortic root while maintaining the guidewire in the left ventricle. This allows pressure to recover as rapidly as possible. Once the balloon inflation is performed without boosting to determine how well the patient will tolerate the inflations, a second inflation is performed to maximal balloon inflation (Fig. 12-13). If the balloon has not ruptured, a third inflation is performed with the intent of rupturing the balloon. It is thus important to prepare the balloon very carefully to be sure that no small air bubbles remain during test inflations outside the body. After maximal inflation and balloon rupture, the balloon is withdrawn over the guidewire and the balloon and sheath are removed together as a unit. The ruptured balloon material is often hard to get all the way back into the sheath. Pulling too forcefully will tear off the end of the balloon shaft. Frequently as the balloon is pulled into the sheath it will cause the sheath to concertina; thus firm pressure to withdraw the balloon part way into the sheath is important and then the combined catheter and sheath are removed as a unit. A new sheath can be introduced over the wire and a diagnostic pigtail catheter inserted in the left ventricle to evaluate the final valvuloplasty result.

Procedural End-Point. If the transvalvular gradient has fallen by more than 50% and if cardiac output is at least unchanged or has risen, successful valve dilation has been accomplished (Fig. 12-14). In some cases evaluation of valve resistance is helpful (Fig. 12-15). Resistance over 250 dynes/sec/cm^{-5} is consistent with persistent stenosis while values below 200 dynes/sec/cm^{-5} signify relief of obstruction. It is necessary to size up to a 14 French arterial sheath so that a 23 mm diameter balloon can be used in 10–20% of cases. A 23 mm balloon is commercially available but it is only 3 cm in length and must be specially ordered at a 4 cm length in this diameter to be realistically useable for aortic valvuloplasty.

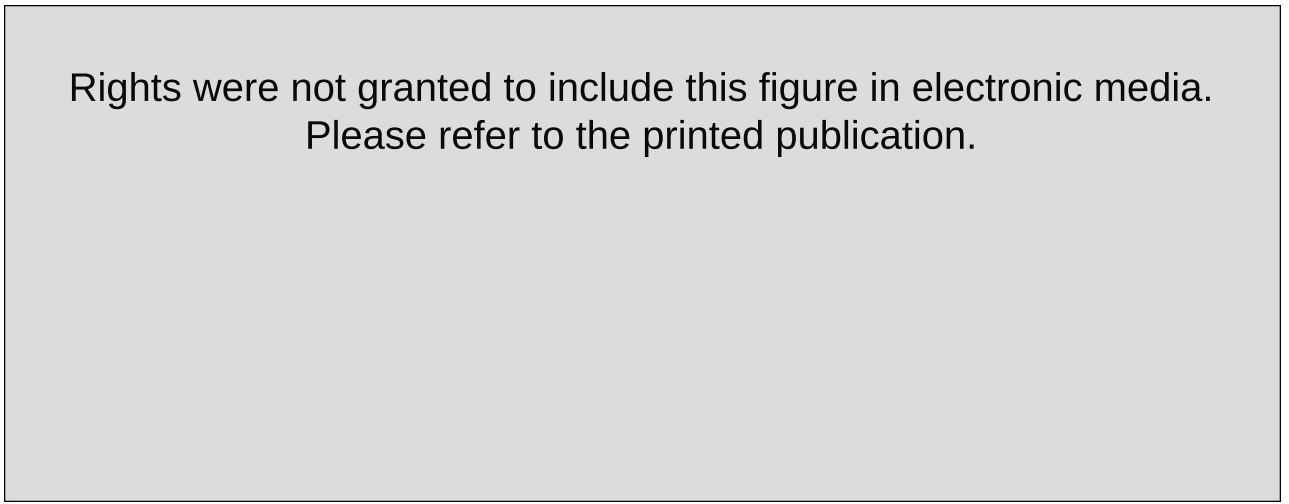

Fig. 12-12 Hemodynamic tracing during balloon inflation in a patient undergoing aortic valvuloplasty. Ventricular tachycardia and hypotension are usual during balloon inflations. (From Feldman T, Carroll JD. Cardiac catheterization, balloon angioplasty, and percutaneous valvuloplasty. In: Hall JB, Schmidt GA, Wood LDH, eds. *Principles of critical care*. New York: McGraw-Hill, 1992:343–360.)

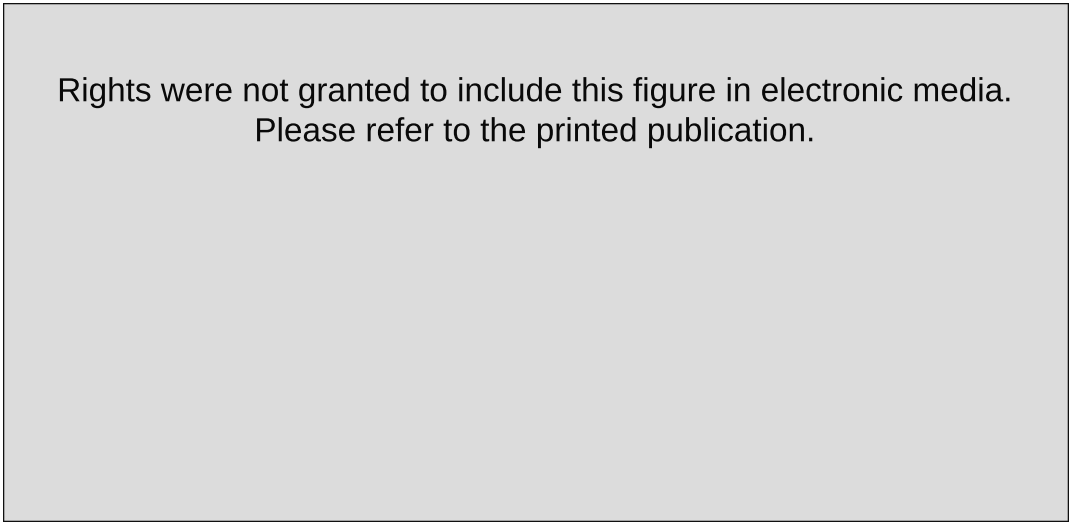

Fig. 12-13 Aortic valvuloplasty. **A,** The valvuloplasty balloon can be seen to be indented by the calcified aortic valve leaflets. **B,** The indentation has expanded as the calcific nodules in the rigid valve leaflets have been fractured. (From Feldman T, Carroll JD. Cardiac catheterization, balloon angioplasty, and percutaneous valvuloplasty. In: Hall JB, Schmidt GA, Wood LDH, eds. *Principles of critical care*. New York: McGraw-Hill, 1992:343–360.)

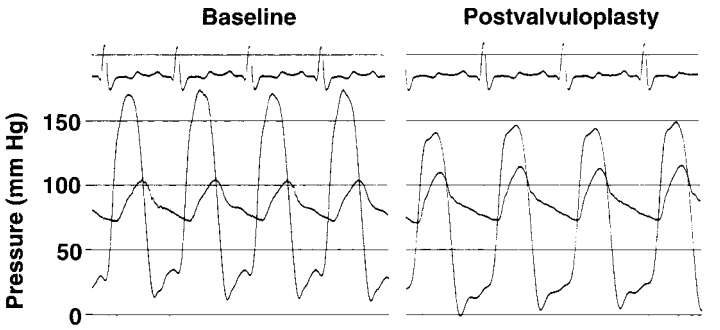

Fig. 12-14 Pre- and post-aortic-valvuloplasty pressure tracings. Not only has there been a marked decline in the transaortic pressure gradient but aortic pressure has risen and left ventricular systolic pressure has fallen. The upstroke of the aortic pressure wave has become deeper. The left ventricular end-diastolic pressure has fallen. This 64-year-old man had an increase in valve area from 0.5 cm^2 to 1.0 cm^2.

Sheath Removal and Post-Procedure Management. Heparin has been given during the procedure and in our usual practice none is given afterwards. Antibiotic prophylaxis is not used for these procedures. The sheaths are removed as soon as the activated clotting time falls below 180 sec if manual compression is to be used. It has become our practice in the last few years to "preclose" the puncture using percutaneous suture closure (Perclose™). For this technique a 6 or 8 French sheath is placed on the femoral artery and, if angiography demonstrates appropriate location in the common femoral artery, a wire is replaced in the sheath. A 10 French Perclose™ device is passed over the wire. The Perclose™ sutures are delivered into the puncture and the needle is pulled back through the skin.

$$\left[\frac{\text{Mean gradient}}{\left(\frac{\text{C.O.(l/min)}}{\text{SEP (sec/min)}}\right) \times 60}\right] \times 80$$

Fig. 12-15 Calculation of valve resistance (dynes/sec/cm^{-5}) may be very helpful in evaluating patients before and after aortic valvuloplasty. C.O., cardiac output; SEP, systolic ejection period. (From Kern MJ, Deligonul U, Donohue T, *et al.* Hemodynamic data. In: *The cardiac catheterization handbook*. St Louis, MO: Mosby, 1994:124.)

The needle is clipped off the suture and the four ends of the two sutures are left dangling outside the puncture. More recently the 6 French Closer™ device has been used, and in some instances, two 6 French devices are placed. The Perclose™ delivery system is partially withdrawn and a guidewire is reinserted through the device. This leads the wire through the purse string in the femoral artery. The Perclose™ delivery system is removed and disposed of and the 12.5 French sheath is passed over the wire. At this point the sheath is in between the sutures. At the conclusion of the procedure the sheath can be removed and the Perclose™ sutures tied in the usual fashion. This approach is successful in almost 90% of cases.

If manual compression is to be used it is important to use a FemoStop™ (RADI Medical, Uppsala, Sweden) for at least 30–60 min after manual compression has been completed since this large puncture has a strong tendency to rebleed. Clamp devices are harder to place and require careful monitoring, whereas the FemoStop™ can be adjusted more easily. Ambulation must be very gradual.

Patients who are not in critical condition before the procedure are able to leave the hospital on the morning following aortic valvuloplasty. It is important to obtain a post procedure echocardiogram prior to hospital discharge so that serial comparisons can be made.

Antegrade Technique for Aortic Valvuloplasty. It is possible to perform aortic valvuloplasty via a transseptal puncture with passage of the balloon through the left ventricle and antegrade across the aortic valve. Advantages of this approach include obviating the need for a large-caliber arterial sheath and the potential to use larger balloons that can be easily inserted on the arterial side. A 14 French sheath is placed in the right femoral vein. An image of antegrade aortic valvuloplasty is shown in Figure 12-16.

After transseptal puncture the Mullins sheath is directed into the left ventricle with the use of a 7 French single-lumen balloon flotation catheter. This catheter can be looped in the left ventricle and floated across the aortic valve. It is our usual practice to advance a guidewire across the aortic valve via the balloon catheter positioned just below the valve and to deflate the balloon just prior to passing it through the aortic valve,

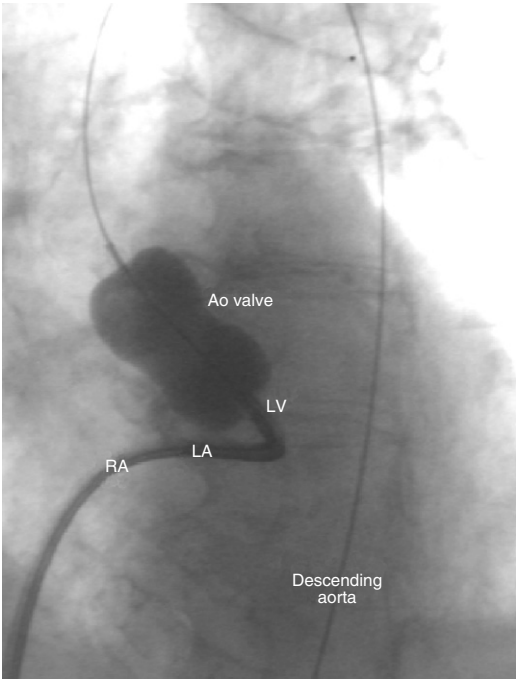

Fig. 12-16 Antegrade aortic valvuloplasty using the Inoue balloon. Beginning at the lower left corner of the figure a guidewire traverses the right atrium (RA) and then the left atrium (LA) via transseptal puncture and is then curled in the left ventricle (LV) and passed across the aortic (Ao) valve through the arch into the descending aorta. Passage of the wire requires a flexible, single-lumen balloon catheter to be floated through a transseptal sheath with a loop in the left ventricular apex. Then an extra stiff guidewire is passed through the balloon catheter and is snared in the descending aorta and exteriorized through a femoral arterial sheath. This rigid, stable wire rail is necessary to provide support for antegrade passage of the balloon catheter through the left ventricle and across the aortic valve. In this figure an Inoue balloon catheter is seen fully inflated in the aortic valve.

since the valve calcifications may cause balloon rupture. Once the balloon is in the aortic root, a 0.032 inch × 260 cm extra stiff guidewire can be passed through the balloon catheter into the descending aorta. Via a 6 French arterial sheath, using a 10 mm gooseneck snare, the wire is snared and the snare is either left in place in the aorta or pulled back so the wire can be

exteriorized through the arterial sheath. The wire thus enters the right femoral vein and passes into the right and then left atria, across the mitral valve, through the aortic valve into the aortic root, and ultimately out of the right femoral artery sheath. A clamp can be placed on a wire as it exits through the right femoral sheath.

It is important to maintain a loop of wire in the left ventricle throughout this procedure to keep from putting too much tension on the mitral valve and causing mitral regurgitation. At this point an extremely stable rail has been created throughout the circulation. It is possible to pass either a conventional balloon or an Inoue balloon antegrade across the septal puncture and into the aortic valve using this approach. This is useful when the aortic valve cannot be crossed retrograde as well. Some patients do not tolerate the wire, possibly because the mitral and/or aortic valves can be "propped" open.

If an Inoue balloon is to be used, the 26 mm maximum size balloon is passed into the left atrium using the same technique as in the mitral dilatation procedure. The wire is left in place throughout the procedure. The balloon is tracked into the aortic valve with the stretching metal tube withdrawn part way into the balloon shaft.

An advantage of the Inoue balloon catheter is that the inflate and deflate cycle is rapid, so that the hemodynamic tolerability of the procedure is enhanced. Conventional balloons can be passed antegrade without too much difficulty, but after they have become "winged" it may be difficult to withdraw them back across the atrial septal puncture. At the conclusion of the procedure a 5 or 6 French pigtail can be passed over the wire from the femoral vein and into the aorta to provide a sleeve for reduced friction when removing the wire.

There is some theoretical benefit to the Inoue balloon in that the waist of the Inoue balloon may fit in the aortic valve annulus while the larger distal bulbous portion may stretch the aortic leaflets more fully into the sinuses of Valsalva. This may result in larger valve areas after aortic valvuloplasty using the Inoue technique in this manner.

Complications

The major complications of aortic balloon valvuloplasty are ventricular perforation from the balloon or guidewires used in

the left ventricle, and femoral artery complications related to the large sheath size that is necessary for the retrograde technique.

Cardiac tamponade from catheter perforation has been reported in about 1% of cases. Vascular surgery for femoral arterial complications is required in as many as 5% of patients. This has been dramatically reduced in our recent experience using suture closure in association with retrograde aortic valvuloplasty or with the antegrade approach. We have also been able to "pre-close" 14 French venous punctures with suture closure with good success. Significant hematomas occur in up to 10% of the patients treated with manual compression, and transfusion rates in some series are as high as 20%. The need for transfusion has been completely eliminated in our practice using suture preclosure. Since the balloon catheter abrades the ventricular septum during balloon inflations, bundle branch block may occur and requires pacing in some cases. Rarely permanent pacemaker implantation is necessary. It is critical to place a temporary pacemaker prior to balloon dilatation in patients who have bundle branch block or high grades of heart block preprocedurally.

Severe aortic regurgitation is infrequent. Leaflet avulsion may occur, usually with oversized balloons. Aortic valvuloplasty in the setting of regurgitation as the predominant valve lesion will not result in clinical improvement for the patient.

Rarely, a progressive low-output state has been encountered after valvuloplasty, sometimes ending in death. Each balloon inflation causes a transient but substantial stress on the left ventricle. Outflow obstruction is acutely worsened and chamber dilatation occurs. Ventricular pressure generation decreases and coronary perfusion pressure drops. Several technical factors can cause this disastrous syndrome. First, inadequate valve dilatation results from an inability to position the balloon properly. Second, repeated inflations may be excessively prolonged. Third, ventricular tachycardia may contribute to left ventricular depression. Lastly, a "rest" between inflations of several minutes is often needed. During this rest period, one should observe a rebound in the aortic pressure, resolution of any ischemic electrocardiography changes, and resolution of any symptoms that have occurred during inflations.

In patients with a low initial cardiac output, less than 2.5 l/min, it is useful to initiate a dobutamine infusion prior to balloon dilatation. Some support for the blood pressure and cardiac output makes the procedure much more reasonable for both the patient and the operator to tolerate.

Bicuspid valves may be resistant to dilatation in adult patients more than degenerated trileaflet valves.

SUMMARY

Mitral stenosis can be treated successfully with catheter commissurotomy with good long-term results and an acceptably low complication rate.

Aortic balloon valvuloplasty is reserved for selected patients at high risk of death from aortic valve surgery or those that require a bridge to surgery, or temporary relief of aortic stenosis to facilitate other medical treatment or noncardiac surgery.

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13

PERICARDIOCENTESIS, BALLOON PERICARDIOTOMY, AND SPECIAL TECHNIQUES

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PERICARDIOCENTESIS

Pericardiocentesis is required for management of acute pericardial effusions and cardiac tamponade complicating a variety of interventional procedures. This is a life-saving technique. Unexplained hypotension during interventional procedures may be caused by right ventricular perforation during pacing catheter placement or coronary perforation during angioplasty. Pericardial tamponade also complicates about 1% of transseptal puncture procedures. A sufficient degree of operator skill must be employed to prevent further damage to the heart, coronary and skeletal arteries, and pericardium. Pericardiocentesis is an essential skill that should be acquired during the diagnostic cardiac catheterization training experience. A complete description of the technique is available in *The Cardiac Catheterization Handbook*. The technique is reviewed here in the context of coronary, valvular, and pericardial interventions.

Procedure

Pericardiocentesis is often preceded by echocardiographic confirmation of the pericardial fluid; however, in the interventional suite tamponade is often acute. Echocardiography is

not required and may be detrimental by delaying needed intervention. Although monitoring of pericardial pressure is not essential, evidence of cardiac tamponade and resolution of pericardial pressure is helpful. A standard 3.5 inch long 18 gauge thin-walled needle can be connected to a pressure transducer for pericardiocentesis. Longer needles are usually not necessary.

Route to Pericardium. The preferred approach is the subxiphoid route but other sites are acceptable depending on the location and volume of the effusion. The advantage of the subxiphoid approach is the decreased likelihood of coronary and internal thoracic artery lacerations. In addition, the more lateral to the sternum the puncture is, the greater the risk of pneumothorax. In the acute setting the subxiphoid approach is most easily identified and is most familiar to most operators.

Setup and Positioning. The patient is raised to a 30–45° head-up angle using a pillow or wedge. This permits pooling of pericardial fluid on the inferior surface of the heart closest to the xiphoid access approach. Local anesthesia is instilled throughout the pericardial needle as it is advanced initially perpendicular to the skin and then at a sharp, low angle. The route of approach is analogous to subclavian puncture where it is important to "walk the needle" under the clavicle.

In the case of pericardiocentesis the needle must be angled to clear below the level of the bottom rib as it attaches to the inferior lateral surface above the xiphoid process. If the puncture is made too high up near the recess at the xiphoid angle, it is difficult to get under the rib. One fingerbreadth inferior and lateral to the edge of the xiphoid allows enough room for passage of the needle beneath the rib.

If right femoral venous access is available, a balloon-tipped catheter is placed in the pulmonary artery for assessment of equalization of the diastolic right-sided pressures. This is also useful to document changes during intervention. If a single-lumen catheter is used for right heart catheterization, the tip is withdrawn into the right atrium for pressure monitoring during pericardial puncture. It is useful to use a multilumen pulmonary artery catheter so that pressure from all the right-sided chambers can be easily monitored in sequence. It is also critical to closely monitor arterial pressure by invasive or non-invasive techniques throughout the procedure.

Puncturing the Pericardium. Aspiration of the needle during passage through the skin may block the needle with subcutaneous adipose tissue. Flush can be used liberally with either saline or lidocaine to clear any tissue that may have accumulated in the needle before passing through the pericardium. The pericardium is a rigid fibrous membrane; therefore pericardial puncture feels similar to a lumbar puncture. Acute pericardial effusions during interventions are bloody, do not generally clot in the syringe, and have a lower hematocrit than intravascular blood.

Immediate confirmation that the needle tip has entered the pericardial space can be obtained by observing the pressure wave form. Inadvertent right ventricular pressure can be recognized immediately. In case of tamponade, pericardial pressure will resemble right atrial pressure.

Hemodynamic monitoring is preferred over echocardiography- or electrocardiography (ECG)-guided pericardiocentesis for its ease of application in the catheterization laboratory. When echocardiographic guidance is used, the observance of echo contrast in the pericardial space is more useful than attempts to identify the tip of the needle. The plane of echo imaging will typically transect the body of the needle and give a false clue regarding the location of the tip of the needle. ECG guidance has also been used. An alligator clip is used to connect the needle to an ECG lead. If the needle tip contacts myocardium as it is being advanced, there is a current of injury, with acute ST segment elevation seen on the monitor.

Once the needle is in the pericardial space, as confirmed by fluid withdrawal, a soft guidewire is inserted under fluoroscopic guidance to the pericardial space. It is typical for this wire to loop in the pericardial space. Should the wire go far beyond the heart border, it is possible that the pleural space or pulmonary artery (via the right ventricle) has been entered. Injections of contrast through the needle may be necessary to identify that the needle is indeed in the pericardial space. Once the wire has been placed, a soft, multiple-side-hole, plastic catheter or sheath can be passed into the pericardial space. When a pericardial drain kit is not available, a pigtail catheter may be used. Pigtail catheters are typically difficult to keep patent and when used in an emergency setting can be

exchanged for a larger-bore pericardial drainage catheter when the acute situation has stabilized.

Pericardial and right atrial pressures are measured. If there is a question regarding the exact position of the needle or catheter, x-ray contrast or echo contrast may be used to verify catheter position. Contrast media pools in the dependent portion of the pericardial space but will wash out of the vascular space rapidly if a cardiac chamber has been inadvertently entered.

Pericardial drainage catheters can be sutured in place and the volume of output monitored. Considerations for conservative or surgical management are made after the drain is in place. Right ventricular and most coronary guidewire perforations can be managed conservatively but reversal of heparin may cause PCI vessel closure. Elective surgery or bypass may be a preferred option under these circumstances. Tamponade is particularly problematic to manage in patients who have received intravenous platelet inhibitors. The administration of platelets, fresh frozen plasma, and the time necessary for small-molecule agents to resolve is critical.

PERCUTANEOUS BALLOON PERICARDIOTOMY

Treatment options for patients with cardiac tamponade include percutaneous catheterization or surgical drainage of the pericardial effusion. Reaccumulation of fluid with recurrence of cardiac tamponade has been reported in between 15% and 50% of patients treated with simple catheter drainage. Creation of a "window" using operative techniques has been traditional therapy for this problem. Methods for creating such a window using balloon catheters have been described (Fig. 13-1). *In vitro* studies have demonstrated that balloon dilatation creates a 1.5–2 cm hole in the pericardium.

Malignancy is the most frequent cause of pericardial effusion and tamponade. Cancer patients have a very short life expectancy and are often debilitated, making them poor candidates for surgical therapy. In addition, surgery may impair what remaining active life they have; therefore the availability of a percutaneous method for pericardial drainage represents an important therapeutic option.

Patient Selection

Patients with large pericardial or recurrent effusions are felt to be at risk for tamponade and are appropriate candidates for a

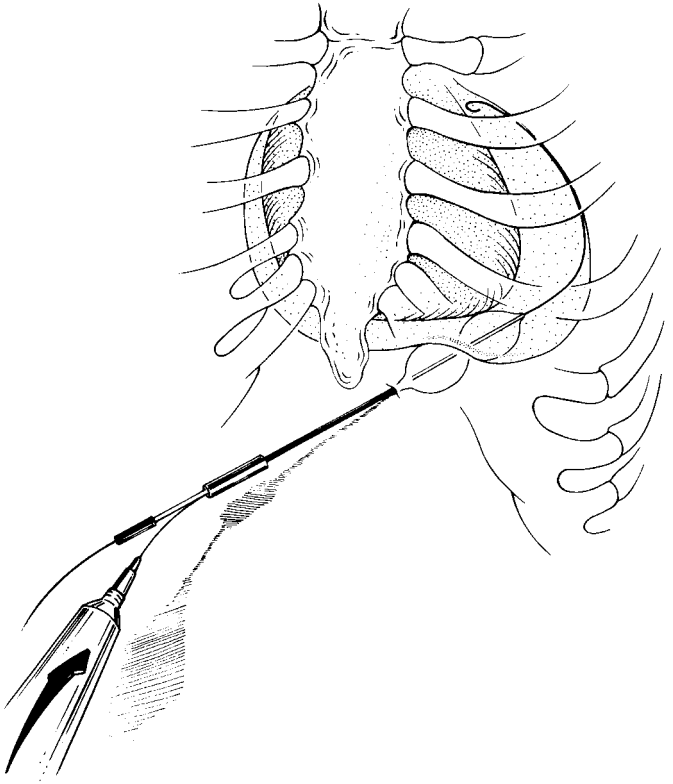

Fig. 13-1 Schematic representation of percutaneous balloon pericardiotomy technique. (From Ziskin AA, Pearce AC, Lemmon CC, *et al.* Percutaneous balloon pericardiotomy for the treatment of cardiac tamponade and large pericardial infusions: description of technique and report of the first 50 cases. *J Am Coll Cardiol* 1993;21:1–5.)

balloon window. Contraindications include refractory coagulopathy, platelet dysfunction, thrombocytopenia with abnormal bleeding, and a clinical history suggestive of a bacterial or fungal etiology for the effusion. Although malignancy is the major etiology in patients undergoing this procedure, some treated patients have had idiopathic effusions, human immunodeficiency virus (HIV)-related or uremic effusions, viral effusions, hypothyroidism, or posttraumatic effusions. Patients with malignancy may be candidates for percutaneous balloon pericardiotomy (PBP) if they have

undergone prior pericardiocentesis and have had either persistent drainage greater than 100 ml over 24 hours or recurrent pericardial effusion after prior catheter drainage. PBP may be offered as a primary treatment at the time of the initial pericardiocentesis when the diagnosis of malignancy is clear.

Bleeding complications can occur in patients with coagulopathies or platelet disorders. Echocardiography should be used to identify free pericardial fluid. Loculated effusions are best treated by surgery. Because left pleural effusions often develop after PBP, patients with marginal pulmonary function should be evaluated carefully before undergoing this procedure.

Results

Results of percutaneous balloon pericardiotomy in 123 patients were reported from a national registry. Of these, 85% had malignancy as the underlying condition. Previous pericardiocentesis had been performed in 58% of the patients. Pericardial tamponade was the presenting problem in 72% of the patients, whereas the remainder had large effusions. Balloon pericardiotomy was successful in 85%, while 4% were considered failures due to pericardial bleeding or persistent catheter drainage after the procedure. A recurrent pericardial effusion appeared in 10% of the patients after a mean time of about four months. Twelve of the 13 patients underwent a surgical pericardial window and six of these had recurrences.

Thoracentesis or chest tube placement was required in 17% of the patients with pre-existing pleural effusions and in 13% of those without pre-existing pleural effusions. Despite the short-term success of the balloon window procedure, the long-term survival of this group of patients was only 3 months.

Technique

Liberal sedation is given, although caution to avoid further hypotension or respiratory depression should be used in patients who are *in extremis* from severe tamponade. For balloon pericardiotomy prophylactic antibiotics are frequently administered.

Pericardial Puncture. Using a standard subxiphoid approach, special care must be taken not to make the needle insertion site

too close to the inferior margin of the ribs, as discussed previously. This will result in too sharp an angle for the wire and balloon catheter to turn from the skin into the pericardial space. The patients are placed on a 45° angle foam wedge. After local anesthesia to the skin, deeper anesthesia is given because the passage of the balloon catheter causes significant discomfort. Prophylactic atropine can be administered to minimize the risk of vagal reactions. The pericardium is entered with an 18 gauge thin-walled needle. A J tip guidewire is advanced in the pericardial space and the needle is removed. The entry channel is dilated using an 8 or 9 French rigid dilator. A straight pericardial drainage catheter with multiple side holes (Cook, Inc., Bloomington, IN) is placed in the pericardial space over the wire and the wire is removed.

Hemodynamic Measurements. Pericardial pressure is measured simultaneously with right atrial and right ventricular pressures (Fig. 13-2). Pericardial fluid is drawn for laboratory studies. Enough additional fluid to relieve hemodynamic evidence of pericardial tamponade is removed. It is important to leave at least 100–200 ml of fluid within the pericardium to provide a cushion for safety for balloon manipulation.

Balloon Catheter Insertion. If the patient has been elevated for the pericardial function, it is important to remove the pillow or wedge before performing balloon pericardiotomy. Table-mounted pressure transducers need to be lowered at this point. If the patient is left sitting partially upright and angulation is created between the lower margin of the ribs and the skin insertion for the balloon, this makes passage of the balloon underneath the ribs extremely difficult. If the patient is placed supine before the balloon catheter is inserted, there is a much straighter path for the wire and balloon to track over. A 0.038 inch extra stiff wire is advanced into the pericardial space and placed in a generous-sized loop (Fig. 13-3). The pericardial catheter is removed and predilatation is performed with a 10 French dilator followed by a 20 mm × 3 cm long Mansfield balloon catheter or a 26 mm Inoue balloon advanced over the guidewire to the pericardial membrane. Contrast can be injected in the pericardial space before passage of the balloon to make this position more identifiable.

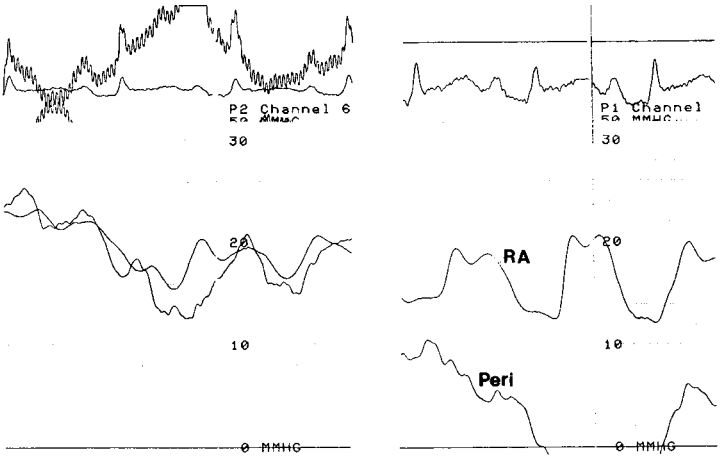

Fig. 13-2 On the left are simultaneous right atrial (RA) intrapericardial (Peri) pressures from a patient with pericardial tamponade. On the right, following pericardiocentesis, the right atrial pressure remains above the zero line, while the pericardial pressure now shows respiratory variation with its nadir below zero.

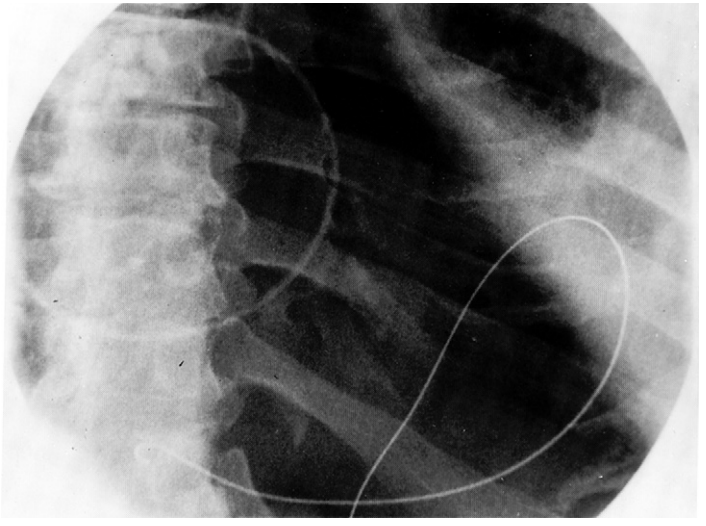

Fig. 13-3 Fluoroscopic image showing an extra-stiff guidewire looped in the pericardium, outlining the pericardial space. Note that the silhouette defined by the wire is substantially wider than the border defined by the cardiac chambers.

Balloon Inflation. Gentle inflation of the balloon may be helpful in locating the pericardial membrane (Figs 13-4, 13-5). When the location of the pericardium has been established by the appearance of a waist in the balloon, the balloon is inflated manually (Fig. 13-5). No more than two or three inflations are ordinarily required to be sure that a window has been created. Use of the Inoue balloon may facilitate positioning across the pericardium. If the pericardium is opposed to the chest wall, as indicated by failure of the proximal portion of the balloon to expand, a countertraction technique can be used in which the catheter is firmly advanced while the skin is pulled back in the opposite direction to help isolate the pericardium for dilatation (Fig. 13-6).

Balloon inflations are generally painful for the patient. After pericardiocentesis but before balloon pericardiotomy, premedication with intravenous analgesics is useful. The balloon catheter is removed and the pericardial drainage catheter is replaced over the guidewire. X-ray contrast can be injected into the pericardial space to demonstrate the passage

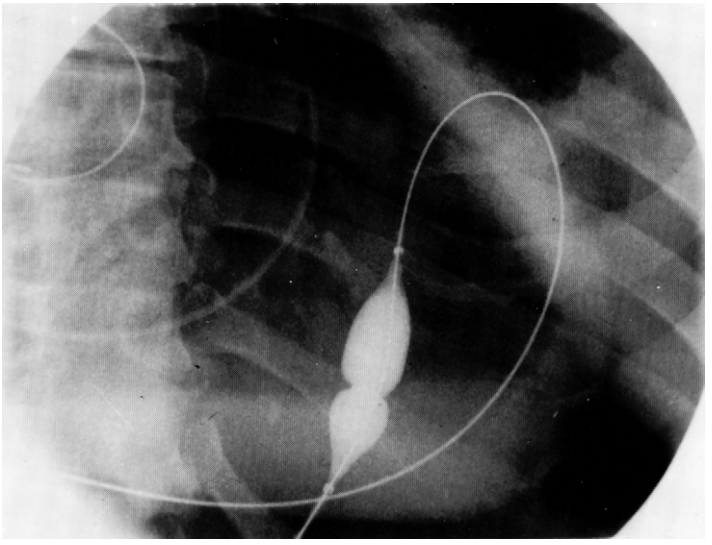

Fig. 13-4 Percutaneous balloon pericardiotomy: a 3 cm long balloon is straddling the pericardial membrane. The membrane has indented the balloon.

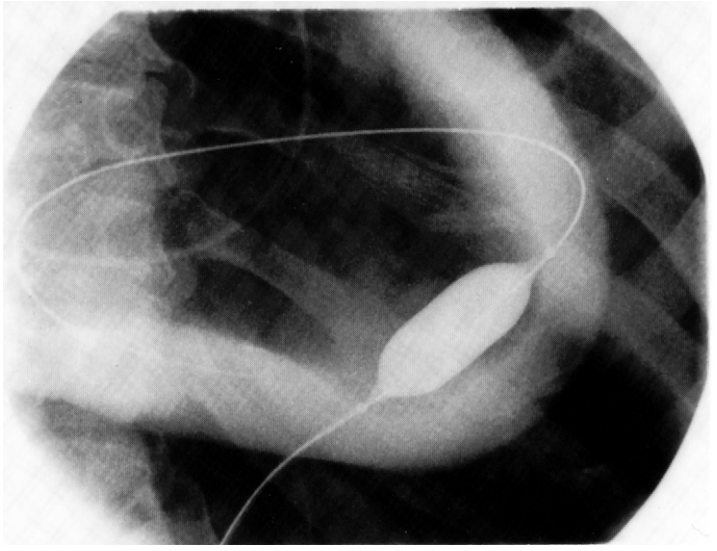

Fig. 13-5 Anteroposterior fluoroscopic images; 20 ml of radiographic contrast has been instilled into the pericardial space for illustration. A 0.038 inch guidewire has been advanced through the pigtail catheter and can be seen looping freely within the pericardial space. As the balloon is inflated manually, a waist is seen at the pericardial margin (Fig. 13-4). The waist disappears with full inflation of the balloon as the pericardial window is created.

of pericardial fluid out of the space into either the left chest or the abdomen. Any remaining fluid is drained from the pericardium. During the attempt at complete drainage, it is often common for the patient to complain of typical pericardial pain. This is a sign of diminished pericardial fluid, allowing the two pericardial surfaces to come in contact with one another. Forewarning the patient of this and giving adequate analgesics are important. The pericardial drain is connected to a collection bag.

The mechanism of PBP is tearing of the pericardium, leading to a communication of the pericardial and pleural or abdominal spaces. The use of a flexible fiberoptic pericardioscope introduced over a guidewire after PBP demonstrated that the pericardial window communicates freely with the left pleural space (Fig. 13-7).

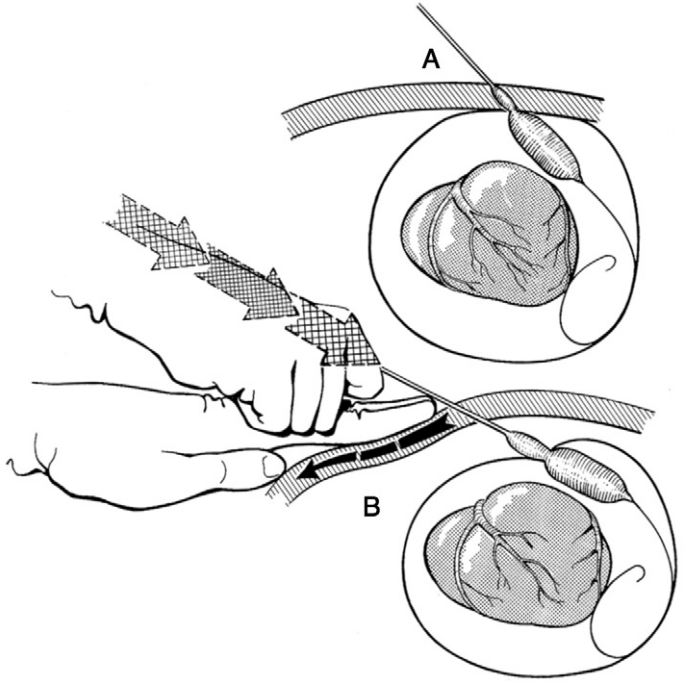

Fig. 13-6 Counter-traction technique to separate the epicardium from the adjacent chest wall (transverse view from below). **A,** Initial trial inflation of the balloon demonstrates trapping of the proximal portion of the balloon within the chest wall structures. **B,** Simultaneous traction on the skin and pushing of the balloon catheter results in displacement of the pericardium away from the chest wall, allowing proper inflation to occur. (From Ziskind AA, *et al.* Percutaneous balloon pericardiotomy for patients with pericardial effusion and tamponade. In: Topol EJ, ed. *Textbook of interventional cardiology*. Philadelphia, PA: WB Saunders, 1994:1315.)

Postprocedure Management

Before the patient leaves the catheterization laboratory a complete right heart catheterization is performed including cardiac output measurement and recording of the final intrapericardial pressure. This has three significant benefits:

- Post procedure management of intravascular volume is clarified
- The acute hemodynamic success of the procedure is proved
- Residual hemodynamic abnormalities are evaluated.

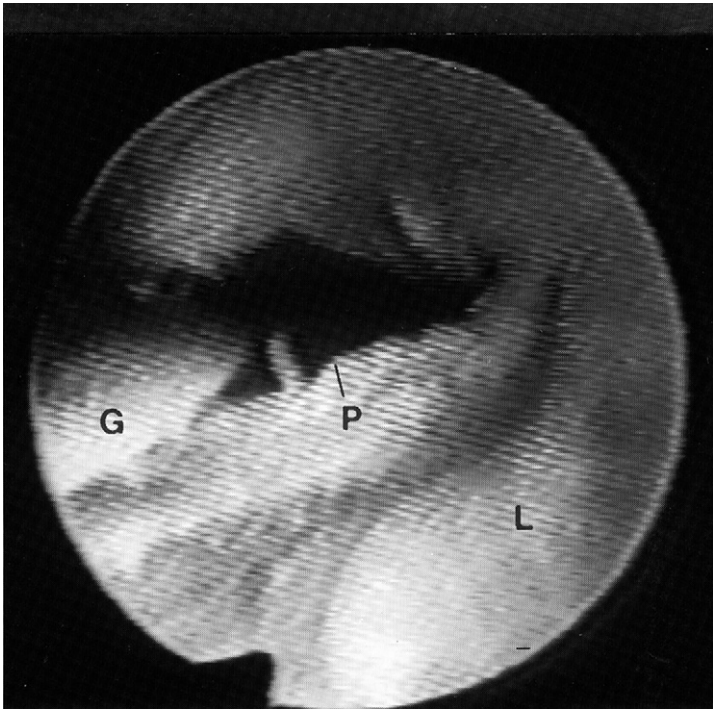

Fig. 13-7 Pericardioscopic view of the balloon pericardiotomy site. The fiberoptic scope has been withdrawn over a guidewire to visualize the external pericardial surface and demonstrate the free communication between pleural and pericardial spaces. G, Guidewire; P, pericardial window created by balloon dilation; L, lung in left pleural space immediately outside the pericardium. (From Ziskind AA, *et al.* Percutaneous balloon pericardiotomy for patients with pericardial effusion and tamponade. In: Topol EJ, ed. *Textbook of interventional cardiology*. Philadelphia, PA: WB Saunders, 1994:1317.)

The incidence of effusive-constrictive pericardial syndrome is high in this patient group. A persistently elevated mean right atrial pressure of greater than 10 mm Hg with an intrapericardial pressure of 0 mm Hg suggests this syndrome. The pericardial catheter may be aspirated and flushed with 5 ml of heparinized saline every 6–8 hours to help maintain pericardial catheter patency. Antibiotics can be administered while the drain is in place. When drainage decreases to less than

100 ml per day, the pericardial drain is removed. Usually this is within 24–48 hours but occasionally substantial drainage may persist for a week. Anti-inflammatory medicines may be useful.

Echocardiography and chest x-rays are performed after the procedure to verify the results. A left pleural effusion may result from successful pericardial drainage and require thoracentesis or chest tube placement in some cases. Fever may occur after the procedure and is often not attributable to any specific cause.

Clinical Results

Multicenter registry data on 130 patients (Table 13-1) from 16 centers demonstrated that no recurrence of pericardial effusion on echocardiographic follow-up was present in 84% of patients at 5 ± 6 months. Five patients were failures due to bleeding and required surgery. Fever, a minor complication, occurred in 13%. No patient had bacteremia or positive

Table 13-1

Clinical Characteristics of 130 Patients Undergoing Percutaneous Balloon Pericardiotomy

Age (years, mean ± SD)	58±13 (range 25–87)
Male/female	68/62
Tamponade present	90 (69%)
Prior pericardiocentesis	75 (58%)
Clinical history	
Known malignancy	110 (85%)
Lung	55
Breast	21
Other malignancies	34
Nonmalignant	20 (15%)
Idiopathic	5
HIV disease	4
Postoperative/trauma	4
Uremia	2
Renal transplant	1
Hypothyroidism	1
Congestive heart failure	1
Viral	1
Autoimmune	1

pericardial cultures. Pleural effusions that required thoracentesis or chest tubes occurred in 17 patients, 12 with preexisting pleural effusions. Of 110 patients with malignancy, 90 died, compared with 2 of 20 patients with nonmalignant pericardial effusions. Mean survival from malignant pleural effusion patients was 3.6 ± 3.8 months. No procedure-related variables were found to influence either survival or recurrence. There was no difference in recurrence rate when PBP was performed primarily or after failed prior pericardiocentesis.

SPECIAL TECHNIQUES

Retained PTCA Equipment Components

Rarely, fragments of interventional equipment may be broken and remain in a coronary artery. This may occur with guidewire tips from both fixed-wire and movable, over-the-wire balloon systems, or distal fragments of various other catheters. These retained intravascular fragments carry the risk of coronary artery occlusion, distal embolization of clot, vessel perforation, infection, and ischemic complications. Dislodgment of stents from the delivery balloons has also been a source of retained interventional equipment.

Removal of intravascular fragments and foreign bodies should be done immediately to avoid the complications mentioned above, as well as incorporation of this material after several days during which the objects become coated and interred within the vessel. There are several techniques for removal of retained intravascular foreign bodies. Baskets, forceps, and snares are available and are manufactured in sizes appropriate for placement within the coronary arteries. The Microvena gooseneck snare (Microvena, Minneapolis, MN) is the most commonly used device (Fig. 13-8). Retrieval of foreign bodies from within the heart has been described in detail in *The Cardiac Catheterization Handbook* and will not be repeated here.

Special Guidewire Retrieval Techniques

Multiple catheter systems have been designed to retrieve foreign bodies, which are usually fragments of prior catheters, guidewires, or stents. Most catheter fragments result from injudicious insertion or removal of catheters inserted from the

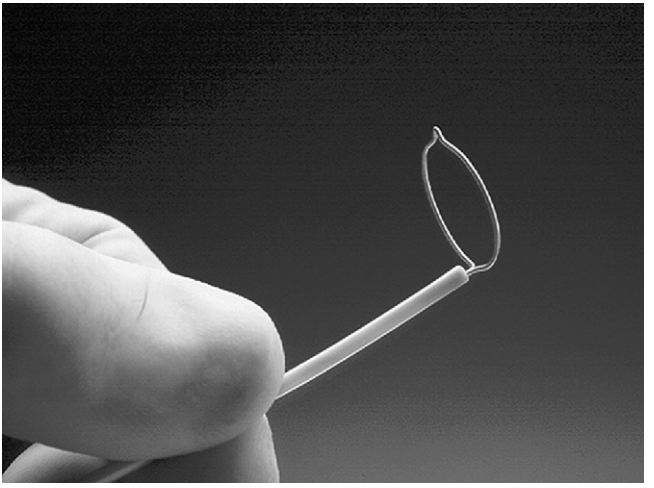

Fig. 13-8 The Microvena Amplatz gooseneck snare is shown in the figure. The nitinol loop of the snare exits the delivery sheath at a right angle making it possible to ensnare objects that would otherwise be coaxial to the long axis of the delivery system. (Courtesy of Microvena, Inc., Minneapolis, MN.)

subclavian, jugular, or rarely inferior vena caval approaches. Peripheral stents may also be dislodged within the arterial system. Most recently, angioplasty guidewire fracture has required refined removal techniques for coronary arteries. Gooseneck snares passing through an intracoronary guide catheter can be applied to retrieve intracoronary guidewire fragments from angioplasty systems. The snare and loop techniques have been used successfully in both venous and arterial applications. Importantly, the catheter fragment or guidewire material that is being retrieved may scratch or tear the cardiac chamber or vessel unless it is captured in such a manner that it can be withdrawn into a guide catheter with minimal buckling or collapsing (Fig. 13-9). In some cases it is possible to pass two intact coronary guidewires beyond a wire fragment and twist them together to ensnare and remove the fragment.

Another important approach to management of intravascular foreign bodies is using an additional stent to bury the fragment. Undeployed stents in the periphery may be pulled into the iliac arteries and then buried under another

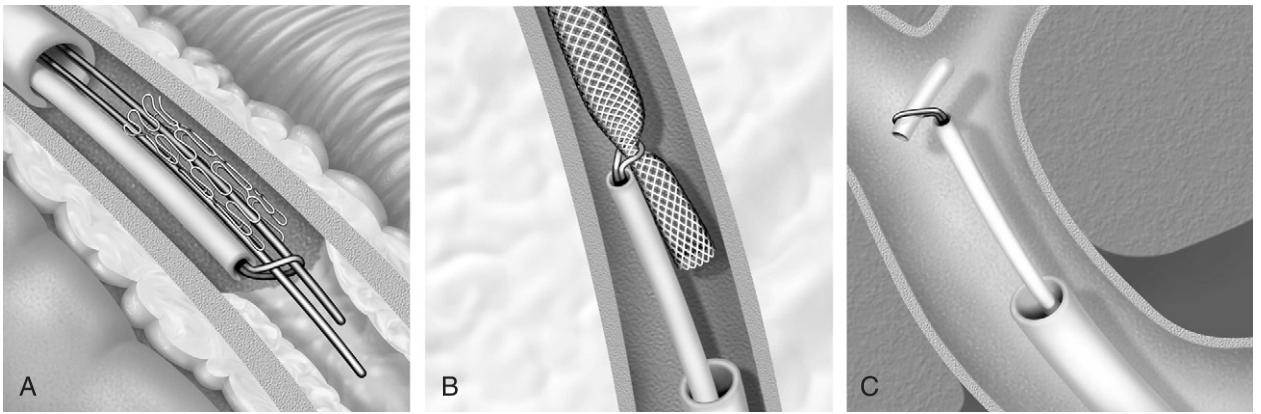

Fig. 13-9 Stent and foreign-body retrieval. **A,** Stent retrieval. Assuming that the original guidewire is still through the stent, a second guidewire may be passed alongside the existing wire to track distally past the stent. The microsnare may be used to grasp the second guidewire. Once the loop has been advanced past the stent, it can be opened and positioned beyond the original guidewire. The microsnare loop can be pulled over the original wire to grab the stent between the two guidewires. The microsnare and stent can then be removed through the guide catheter and sheath. **B,** Stent retrieval. Using a guidewire to reach a migrated or lost stent. The snare delivery catheter may be passed over the wire. The guidewire can be withdrawn and the snare loop passed through the delivery catheter to position the snare loop around the stent as close to the proximal edge as possible, so the stent will not buckle when it is withdrawn into the guide catheter. The snare can be tightened around the stent by advancing the snare catheter forward over the loop. The stent and snare may be manipulated into the femoral vessels and deployed, or removed as appropriate, depending on the operator's judgment. **C,** Foreign body fragment retrieval. A guide catheter is introduced proximal to the fragment. After the guidewire is in position, a wire can be passed distal to the fragment. The snare can be passed over the guidewire so that it tracks along the wire and advances until it reaches the catheter fragment. The wire is withdrawn and the snare loop is introduced into the snare catheter. The fragment is then grasped by the loop. After the loop is tightened, the snare and fragment can be withdrawn. (Courtesy of Microvena, Inc., Minneapolis, MN.)

balloon-expandable stent. This approach has also been used successfully in the coronary arteries for dislodged stents and guidewire tip fragments. An advantage of this approach is simplicity. There is minimal manipulation within the coronary, and the potential for dissection, thrombosis, or a protracted effort can be minimized.

Shortening Guiding Catheters

A technique has been described for shortening angioplasty guide catheter lengths when therapeutic catheters fail to reach a target lesion. Most standard guide catheters are 100 cm long and some shapes suitable for bypass grafts are available in 90 cm length. The Toughy connector adds another 6–10 cm in overall length. Because conventional angioplasty balloon catheter shafts are 135–145 cm the distal portion of the catheter extends 25–35 cm or less from the guide catheter tip. In some cases the limited length will prevent successful access to a distal lesion, particularly when approaching distal native lesions via surgical grafts. The technique for shortening the guide catheter *ex vivo* or *in vivo* while maintaining both guide catheter and guidewire position is described next.

To shorten an indwelling guide catheter a clamp is placed under the guide catheter proximal to the sheath hub by a distance equal to the desired length of catheter reduction. The clamp prevents unnecessary blood loss during the shortening procedure. The guide catheter is cut with a scalpel proximal to the clamp, taking care not to damage the indwelling coronary guidewire. A 0.035 inch or 0.038 inch buddy wire can be inserted into the guide to help support this procedure. The guide catheter hub and hemostatic valve are removed. Next, a standard femoral sheath 1 French size smaller than the guide catheter is cut to approximately 2 cm of the remaining sheath length from the hub. The newly shortened sheath is flared with a vessel dilator 1 French size larger than the nominal sheath size. This is accomplished by inserting the tapered end of the dilator retrograde into the sheath tip. The shortened and flared sheath is threaded over the indwelling guidewire, making sure that the stiff end of the guidewire does not perforate the diaphragm of the short sheath. A dilator or wire insertion tool placed through the sheath may facilitate passage of the guidewire while threading it onto the guide catheter. The flared

end of the sheath stub is advanced over the guidewire with firm friction and the hemostatic clamp is removed. The side port of the sheath is connected to the manifold. The newly assembled system is then aspirated and flushed. It is also possible to use a guide catheter of the same brand 1 French size larger than the indwelling guide catheter for the same purpose. The hub of the larger guide catheter is cut with scissors or scalpel, and then placed over the cut end of the indwelling smaller guide catheter. The Toughy is then reconnected and the procedure resumed. The balloon catheter can then be advanced over the guidewire through the diaphragm of the short sheath newly secured onto the in-dwelling guide catheter or through the newly attached additional guide catheter hub. The balloon catheter will then have additional length to reach the target lesion.

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14

SEPTAL ABLATION FOR HYPERTROPHIC OBSTRUCTIVE CARDIOMYOPATHY

Carey Kimmelstiel

Hypertrophic cardiomyopathy (HCM) is a relatively common genetic disorder that exhibits a wide variability in its clinical expression. Progression to New York Heart Association Functional Class (NYHAFC) III–IV symptoms with left ventricular outflow tract (LVOT) obstruction occurs in approximately 10% of HCM patients. The most common symptoms are exertional dyspnea, chest pain, and fatigue.

The initial therapy for improvement in quality of life in symptomatic HCM patients has traditionally focused on administration of negatively inotropic agents such as beta-adrenergic antagonists, verapamil, and disopyramide. In the minority of patients who develop progressive symptomatic disability despite drug treatment, mechanical approaches to eliminate left ventricular (LV) obstruction are employed. Until recently this approach involved surgical ventricular septal myectomy, a procedure with more than a 40-year proven track record.

The surgical treatment for obstructive HCM has been limited to a few medical centers. Patients with obstructive HCM may not have access to such a center and other patients may be suboptimal candidates for surgery. Consequently an alternative to a surgical approach for medically refractory HCM patients has been found in a percutaneous method, known by several names and acronyms: percutaneous transluminal septal myocardial ablation (PTSMA), transcoronary ablation of septal hypertrophy (TASH), nonsurgical septal reduction therapy

(NSRT), nonsurgical myocardial reduction (NSMR), and alcohol septal ablation (ASA). This procedure employs conventional interventional coronary techniques to cause alcohol-induced necrosis of the anterior basal interventricular septum, leading to reduced LV wall thickness and outflow tract obstruction and reducing mechanical resistance to LV ejection. The goal of PTSMA is to achieve the same morphologic and hemodynamic results as surgical myectomy.

CANDIDATES FOR SEPTAL ABLATION

As with any therapeutic intervention, appropriate patient selection is mandatory. This is especially true for PTSMA, a technique that is relatively new and has not been systematically compared to other treatment modalities.

Reasonable candidates for PTSMA include those with severe symptoms (i.e., NYHAFC III or IV) refractory to maximal drug therapy with an outflow gradient of more than 40–50 mm Hg at rest or more than 60 mm Hg following provocation with physiologic maneuvers such as Valsalva, exercise or following extrasystolic beats. Additional criteria for ethanol septal reduction are shown in Box 14-1. Other candidates for PTSMA include those of advanced age, with prominent surgical contraindications, or with insufficient motivation for surgery. It is important to appreciate that patients exhibiting LV outflow gradients solely in response to therapy with dobutamine should be viewed skeptically as candidates for PTSMA. Although it has at times been used to define patients in need

Box 14-1

Hypertrophic Obstructive Cardiomyopathy—Criteria for Ethanol Septal Reduction

- Severe symptoms refractory to medical treatment
  - —Congestive heart failure (NYHA class III or IV)
  - —Angina (CCS class III or IV)
  - —Syncope
- Septal-to-posterior-wall thickness ratio ≥1.3
- Septal wall thickness >1.8 cm
- Resting gradient ≥40 mm Hg
- Provoked gradient ≥60 mm Hg
- Moderate or less mitral regurgitation

of nonpharmacological intervention, dobutamine is an inotropic agent that is a powerful stimulant of subaortic gradients in normal hearts and in cardiac diseases other than HCM.

Patients with coexisting cardiac diseases that are best treated with surgery, e.g., multivessel coronary artery disease, mitral valve or papillary muscle abnormalities causing mitral regurgitation, should not undergo PTSMA, but should rather be referred for surgical myectomy at which time coexisting abnormalities could be addressed.

TECHNIQUE

Coronary Angiography

The presence or absence of significant epicardial coronary artery disease, particularly in the left anterior descending (LAD) coronary artery, is angiographically documented. Should single-vessel coronary disease in the distribution of the LAD be seen, angioplasty/stenting should be performed prior to PTSMA. Although combined procedures have been reported, currently it seems more prudent to assess the symptomatic results of coronary intervention prior to proceeding with PTSMA.

Temporary Pacing Wire Placement

Because of the possibility of third-degree heart block occurring as a consequence of intracoronary alcohol injection, a 5 French temporary pacemaker (balloon-tipped) is advanced to the right ventricular (RV) apex via the right internal jugular approach. This access will allow the temporary pacer to remain in place until a decision for a permanent pacer is made. The importance of positioning the pacing lead in the RV apex with documentation of an appropriate threshold cannot be overemphasized. It is crucial to avoid positioning the pacemaker in an area of myocardium that might be subject to necrosis following alcohol ablation. In such a scenario, pacemaker capture in the instance of complete heart block would not be possible.

Hemodynamic Monitoring

A pulmonary artery catheter is introduced via the left femoral vein. To monitor left ventricular pressure, an end hole or Halo

5 or 6 French pigtail catheter is positioned fluoroscopically from the left femoral artery. From the right femoral artery, an appropriately shaped 6 or 7 French guide catheter is advanced into the central aorta.

Baseline LV outflow gradient is continuously monitored for the duration of the procedure by observing LV (pigtail) and aortic (coronary guide catheter) pressures. Although usually obvious from preliminary noninvasive studies, initial hemodynamic assessment should localize the gradient to the proximal subvalvular (as opposed to the aortic valvular or mid to distal ventricular) level.

Pre-Medication

In preparation for chest discomfort associated with intracoronary alcohol injection, prophylactic analgesic medication (e.g., fentanyl 25 mg IV) is administered. Antithrombotic prophylaxis with intravenous, weight-adjusted heparin (e.g., 40 units/kg) is given.

Selecting the Septal Artery

The left coronary guide catheter engages the left main coronary artery and angiography is performed. The view that best visualizes the origin and course of the septal branches of the LAD is defined (usually a shallow right anterior oblique with cranial angulation). The septal artery, which supplies blood to the area of septal hypertrophy and obstruction, is identified. This vessel is then entered with an 0.014 inch coronary guidewire (a large double 45° bend is helpful). In smaller, tortuous vessels this may necessitate using wires with hydrophilic tips. A short (approximately 2 mm × 10 mm) over-the-wire balloon is passed over the wire into the septal artery. Short balloons allow balloon positioning entirely within the septal artery, thereby avoiding obstruction to blood flow, or dissection of the LAD. A slightly oversized balloon (2.0–2.5 mm diameter) compared with vessel caliber is used in an effort to avoid alcohol leakage down the LAD.

Confirm Septal Supply Before Alcohol Injection

Prior to ablation it is important to identify the most appropriate septal vessel in which to intervene. Occasionally this vessel takes its origin from an artery other than the LAD

(e.g., diagonal branch). The optimal method to accomplish this is unresolved, with some groups favoring a pressure- and fluoroscopy-guided technique and others utilizing a method employing myocardial contrast echocardiography.

In the pressure–fluoroscopic method, balloon occlusion of the septal artery is used to cause transient ischemia of the subtended septal tissue with consequent reduction of the outflow gradient. Subsequently, selective angiography is then utilized to target whether a branch or the entire septal artery will be ablated. Most practitioners of PTSMA identify the optimal septal artery to ablate by using myocardial contrast echocardiography (MCE). This technique involves two-dimensional echocardiographic and color Doppler monitoring during an injection of 1–2 ml of diluted echocardiographic contrast through the distal lumen of a balloon dilation catheter positioned in a proximal septal perforator. The "best septal" artery is characterized by echo contrast opacification of the area of the septum in closest proximity to the area of the gradient formation, or contact (or closest proximity) to the anterior mitral leaflet, as well as the area of Doppler defined maximal flow acceleration. The use of MCE reduces the number of vessels intervened on during PTSMA and the complications of the procedure. Identification of the correct vessel or branch to ablate using MCE is important in avoiding infarction of myocardium distant to the target area. Such areas include inferior LV and RV free walls and papillary muscles.

Alcohol Injection

Following identification of the optimal septal artery or branch, the angioplasty balloon is inflated in the target septal vessel. At this time it is mandatory to document that there is complete cessation of flow between the distal septal artery and its parent vessel (usually the LAD). Left coronary angiography is performed to insure that no contrast visualizes areas distal to the inflated balloon (Fig. 14-1). Sources of distal contrast filling of the septal region with an inflated balloon include balloon undersizing, underinflation, or poor positioning, especially too proximal, in the septal vessel.

After removing the coronary guidewire, angiographic contrast is injected through the distal balloon port to document no contrast refluxes into the LAD (Fig. 14-1).

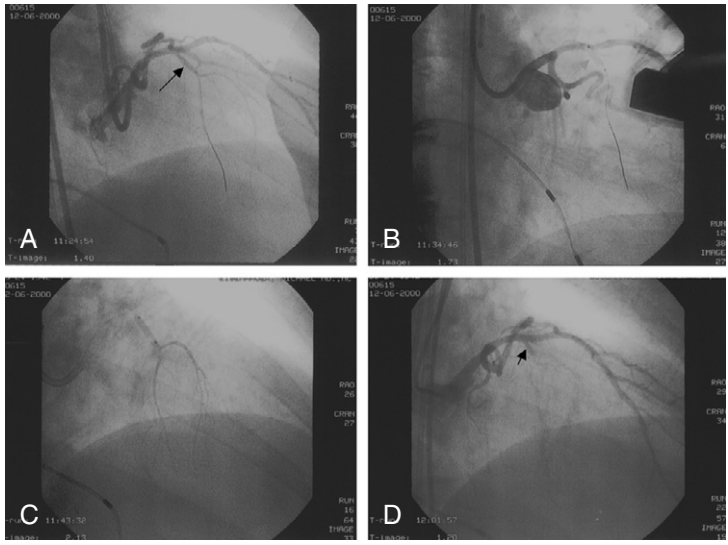

Fig. 14-1 **A,** A wire is positioned in the target septal artery (arrow). **B,** A balloon is inflated in the target septal artery. Contrast injection through the guide catheter documents complete balloon occlusion of the septal artery as no contrast is seen to flow beyond the inflated balloon. Normal contrast filling of the left anterior descending artery (LAD) is seen. **C,** Contrast injection through the distal port of the inflated balloon catheter documents solely anterograde septal filling without any contrast reflux into the LAD. **D,** Postprocedure angiography documents ablation of the target septal vessel.

Percutaneous transluminal septal myocardial ablation is then performed by the injection of 1–3 ml of 96% denatured ethanol into the target septal perforator in 0.5–1.0 ml aliquots at a rate of approximately 1 ml/min. The reported literature has documented that reduced amounts of ethanol and slower rates of infusion minimize complications, particularly high-grade atrioventricular block. Following the LVOT gradient response after the final administration of ethanol, the coronary balloon remains inflated for 5–10 min to insure that no alcohol refluxes into the LAD.

Success Criteria

The decision to terminate the PTSMA procedure is determined primarily by the acute hemodynamic result. The goal of

PTSMA is acute reduction in the resting (or provoked) gradient by 50% or to less than 20 mm Hg (Fig. 14-2). The immediate postablation gradient reduction is due to alcohol-mediated basal septal necrosis and stunning, a mechanism that is distinct from the septal thinning and ventricular remodeling associated with progressive gradient reduction noted on long-term post-PTSMA followup.

Postprocedure Care

Following PTSMA patients remain in a coronary care unit for approximately 24–48 hours. It is important to keep in mind that PTSMA induces a sizable infarction (Fig. 14-3) and that observation of the patient for arrhythmias, especially heart block, is important. In addition, analgesia for ongoing chest discomfort can be addressed at this time. Total hospitalization following PTSMA is usually 4–5 days to monitor for late heart block requiring a permanent pacemaker (Box 14-2).

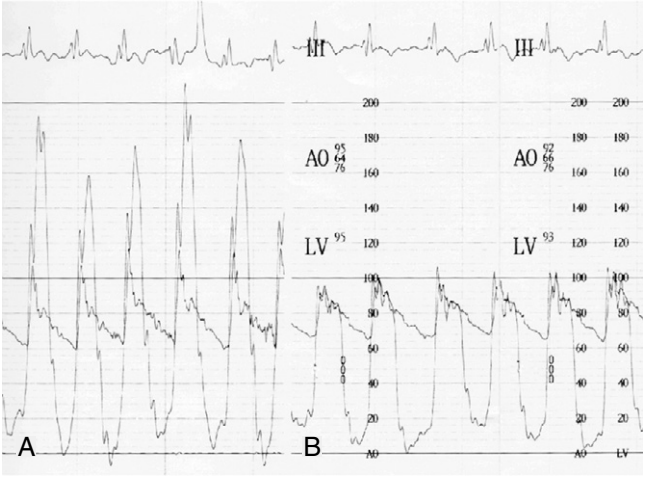

Fig. 14-2 **A,** Hemodynamics prior to septal ablation. There is a resting left ventricular (LV) outflow gradient of approximately 45 mm Hg. This patient exhibited a 120 mm Hg gradient following exercise (not shown). **B,** Hemodynamics immediately following septal ablation. The LV outflow obstruction has been eliminated.

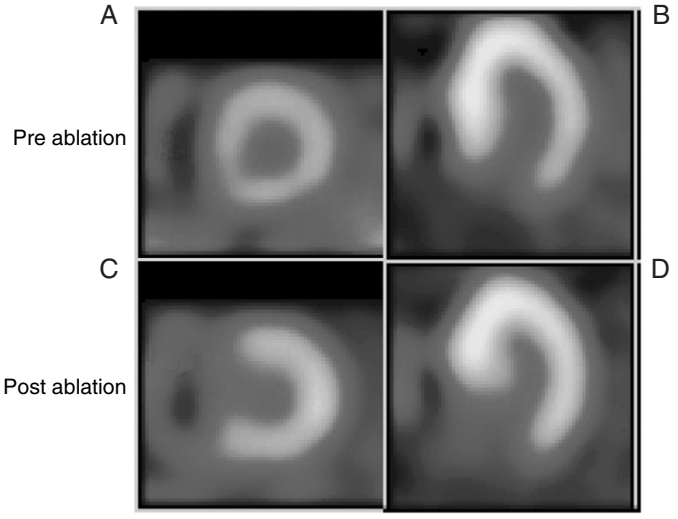

Fig. 14-3 Rest sestamibi imaging prior to and following septal ablation. **A** and **B** display preablation short axis and horizontal long axis views respectively. Postablation images document a septal (**C**) and basal septal infarction (**D**).

Box 14-2

Key Points for Percutaneous Transluminal Septal Myocardial Ablation

- Careful patient selection
  - — Symptoms refractory to maximal medical therapy
  - — Septal thickness ≥1.8 cm
  - — Left ventricular outflow tract (LVOT) gradient at rest ≥50 mm Hg or provokable gradient ≥60 mm Hg
- Identify and treat coronary artery disease before septal ablation
- Use septal balloon size to insure no left anterior descending artery alcohol reflux
- Use myocardial contrast echocardiography to check correct septal artery supplies obstruction and does not connect to remote left ventricular regions.
- Stop alcohol injection when hemodynamics have met success criteria (>50% decrease or <20 mm Hg LVOT gradient)
- Observe in hospital for 4–5 days because of potential for late heart block.

RESULTS

Percutaneous transluminal septal myocardial ablation has not been subjected to randomized clinical trials against the gold standard treatment of septal myectomy. However, the reported observational data from a number of centers in the USA and Europe over relatively short followup periods are reasonably consistent, attributing a number of favorable hemodynamic and clinical effects to PTSMA. Progressive reduction in outflow gradient and normalization of LV pressures, as well as alleviation of symptoms, are reported that are generally similar in magnitude to those resulting from surgery.

While many reports of symptomatic benefit following PTSMA have been based largely on retrospective and uncontrolled assessments, some data are now available describing clinical improvement following PTSMA by objective measures such as treadmill exercise time and peak oxygen consumption. Several groups have documented improvement in exercise capacity following successful PTSMA. Significant and progressive enhancement in exercise capacity, peak oxygen consumption and anaerobic threshold has been noted in followup ranging from 3 months to 2 years. One group has documented an improvement in maximum tolerated workload from 87.5 to 110.3 W 3 months following PTSMA. These improvements were maintained at 2-year followup. Patients with outflow obstruction solely on provocation, when compared to those with resting obstruction, have been reported to have a similar magnitude of benefit, averaging greater than 30% improvement in achieved workload 7 months following PTSMA.

Complications

Complication rates following PTSMA have declined since the initial reports, as a result of operator experience. Specifically, complications are reduced as a result of more precise identification of the most appropriate target septal perforator artery for intervention as well as the use of smaller amounts of alcohol introduced at slower rates limiting areas of myocardial necrosis and scarring. Experienced center PTSMA-related mortality is currently approximately 1–2%, similar to that of surgery. Conduction system abnormalities are relatively

common as complications of PTSMA, with permanent right bundle branch block occurring in about 50% of patients, transitory complete heart block in 60% and permanent pacemakers required for high-grade atrioventricular block in about 10–20%. There is particular concern regarding complete heart block because of its occasional unheralded late occurrence following PTSMA, which mandates in-patient monitoring for 4–5 days.

A profound potential complication of PTSMA is anterior myocardial infarction due to reflux of ethanol from the septal perforator branch into the LAD. This result can be avoided by scrupulous positioning of the balloon and angiographic monitoring. Other rare complications of PTSMA include coronary dissection, perforation, thrombosis and spasm.

LIMITATIONS AND CONCERNS

A concern regarding the widespread adoption of PTSMA is its long-term safety compared to surgical myectomy. The potential long-term consequences of the transmural intramyocardial septal scar produced by PTSMA but not by septal myectomy are not known.

Even before PTSMA, many patients with obstructive HCM harbor an electrically unstable myocardial substrate prone to re-entrant ventricular arrhythmias. A possibility that the resultant septal infarct could in fact enhance the likelihood of sudden death in some patients has been noted. The inherent difficulty in assessing this potential complication of PTSMA is due to the relatively short follow-up period (i.e., <5 years) currently available for the vast majority of PTSMA patients and the particularly long risk period implicit in young HCM patients. The widespread adoption of PTSMA in the absence of randomized trials must be carefully considered.

The reported literature suggests that the threshold for intervening in HCM patients is declining. This may be due, in part, to PTSMA being considered as a low-risk procedure by interventionalists whose inclination might be to treat a gradient rather than a patient, and who may not understand that the vast majority of HCM patients can be adequately treated with conventional medical therapy.

Percutaneous transluminal septal myocardial ablation is a relatively new therapy for obstructive HCM that uses conventional coronary interventional techniques. PTSMA is effective in improving symptomatic status, exercise capacity, and hemodynamic perturbations in HCM patients who are reported to be refractory to medical therapy. Intraprocedural assessment, usually using MCE, helps to insure procedural efficacy and reduce complications. The possibility of long-term complications related to PTSMA mandates continued systematic followup.

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15

TRANSCATHETER CLOSURE OF ATRIAL SEPTAL DEFECTS AND PATENT FORAMEN OVALE USING THE AMPLATZER® DEVICES

Hitendra T. Patel, Qi Ling Cao, and Ziyad M. Hijazi

ATRIAL SEPTAL DEFECTS

An atrial septal defect (ASD) is a communication between the right atrium and left atrium due to an abnormal septation. There are four types of ASD:

- Primum
- Secundum
- Sinus venosus (superior vena cava [SVC] type and inferior vena cava [IVC] type)
- Coronary sinus septal defect.

ASDs are among the most common congenital heart defects. Only the secundum type is amenable to transcatheter closure at present.

SECUNDUM ATRIAL SEPTAL DEFECT

This is a defect or deficiency in the septum primum in which the overlap with the septum secundum is incomplete, leading to the defect. The defect is therefore bordered by the limbus of the fossa ovalis or the C-shaped septum secundum. It comprises 7% of all congenital heart defects and is twice as common in females. Candidate genes for development of secundum ASDs have not been identified and most are sporadic. Holt–Oram syndrome, an autosomal dominant disorder characterized by upper limb skeletal abnormalities,

absent or hypoplastic radii, and conduction defects, is associated with secundum ASDs. Complete absence of the atrial septum is associated with Ellis–van Creveld syndrome. Associated anatomic lesions include mitral valve prolapse, partial anomalous pulmonary venous return, and complex congenital heart defects.

Pathophysiology: Atrial Level Left-to-Right Shunt

The flow across an ASD or other atrial level shunt occurs in diastole and its direction depends on the differences in the atrial pressures and compliance rather than on pulmonary vascular resistance (PVR) or systemic vascular resistance (SVR)—although the latter are indirect factors. The compliance of the atria is determined by their respective ventricular compliance. This is dependent on ventricular wall thickness, which is directly proportional to ventricular pressure and the PVR in the case of the right ventricle and SVR in the left ventricle. As right ventricular pressure and PVR increase, the wall thickness of the right ventricle increases, leading to a fall in right ventricular and right atrial compliance. Normally the mean left atrial pressure is 6–9 mm Hg and the mean right atrial pressure is 1–4 mm Hg. This favors a left-to-right shunt.

The pressure in the respective atria is dependent on the compliance of the respective ventricles. If the ventricles are poorly compliant or stiff, a higher atrial pressure is required for filling to occur. Unless the communicating defect is small, the amount of flow is dependent on the difference in compliance of the ventricles rather than the pressure, since a large defect will equalize the pressures in the atria. Normally, the right ventricular compliance is much higher than that in the left ventricle, resulting in a left-to-right shunt across the ASD, and its magnitude is dependent on the relative difference between the right ventricular and left ventricular compliance.

At birth the wall thicknesses of the right and left ventricles are similar since the PVR and SVR are similar. Hence, compliances of the ventricles are similar and the amount of flow across the ASD is minimal or can be bidirectional. As soon as lung inflation occurs and PVR starts to fall, the compliance of the right ventricle begins to increase relative to the left ventricle. This leads to left-to-right flow across the ASD, which

will continue to increase as the PVR continues to fall and the right ventricular compliance continues to increase.

Left ventricular compliance is relatively stable for the first 20–30 years of life. As aging occurs, the arteriolar elasticity decreases and SVR increases, leading to higher blood pressure. This leads to higher energy expenditure by the left ventricle to overcome increased afterload, and subsequent left ventricular hypertrophy. The compliance of the left ventricle decreases, with a subsequent elevation in left atrial pressure and increased left-to-right atrial level shunt. The shunt results in right-sided volume overload. Thus, the right atrium, right ventricle, pulmonary arteries, and pulmonary vascular bed are enlarged because of the increased volume of the shunt. There is increased flow across an otherwise normal tricuspid and pulmonary valve, leading to increased turbulence or a flow-related gradient across these valves.

Clinical Presentation

History. Small defects, usually less than 5 mm, without right-sided volume overload, have no effect on the natural history of the individual. Some of these defects may close spontaneously. Patients with moderate to large defects are usually asymptomatic until the second decade of life. The majority of patients are asymptomatic at the time of diagnosis. The physiologic impact of the left-to-right shunt increases with age. If the diagnosis is missed then, in the second decade of life, some of these patients may present with atrial arrhythmias, including atrial flutter, atrial fibrillation, and automatic atrial tachycardia. There is a progressive increase in symptoms in the second to third decade of life. There is progressive decrease in exercise tolerance and occasionally overt congestive heart failure may manifest. Unusual symptoms of orthodeoxia and platypnea can occur, especially in the fifth to sixth decade. Pulmonary vascular disease may develop in 5–10% of patients with significant defects, usually after the second decade and commonly after the fourth or fifth decade of life. Paradoxical cerebral or systemic emboli may occur. Infective endocarditis is very uncommon and prophylaxis for isolated secundum ASD is not recommended.

Physical Examination. The auscultatory findings reflect the physiology. An ASD results in an increased volume load on the

right atrium and right ventricle. This can produce a split S1 with a loud second component due to forceful and late closure of the tricuspid valve. The classical finding is a wide fixed splitting of S2. The wide split results from the late closure of the pulmonary valve. Late closure occurs because of the increased volume load ejected by the right ventricle. The late closure is accentuated by increased capacitance of the pulmonary bed. In patients with a dilated right ventricle the time required for depolarization is increased. This is manifest by the incomplete right bundle branch block observed on the surface electrocardiogram (ECG) in these patients. This further prolongs right ventricular ejection time, delaying pulmonary valve closure compared to the left ventricle.

Normal physiologic splitting of S2 results from respiratory variations in loading of the right and left side of the heart (i.e., increased venous return to the right ventricle with inspiration and increased venous return to the left ventricle with expiration). In the presence of an atrial septal communication these changes are transmitted to both sides and respiratory variation in splitting of S2 cannot occur. Ejection systolic murmur, usually grade 2–3/6, results from relative pulmonary valve stenosis produced by increased blood flow across it. Occasionally, middiastolic flow murmur can be heard secondary to increased flow across the tricuspid valve.

Laboratory Findings.

- **Chest radiography.** Mild to moderate cardiomegaly can be seen due to enlargement of the right atrium and right ventricle. There are increased pulmonary vascular markings in patients with large left-to-right shunts.
- **ECG.** Normal sinus rhythm with right axis deviation, intraventricular conduction delay with deep S wave in lead 1 and RSR′ in V1 and V3R. Occasionally a counterclockwise loop can be seen, with left axis deviation and Q-waves in leads 1 and AVL.
- **Echocardiography.** Transthoracic echocardiography (TTE) is helpful in delineating the left-to-right shunt. However, it is not adequate for precisely locating the location and surrounding rims of the ASD. A dilated right atrium and right ventricle, as well as paradoxical ventricular septal motion secondary to a volume-loaded right ventricle, are noted. Flow

acceleration across the tricuspid and pulmonary valves is seen because of the increased volume across these valves. It is important to ensure normal pulmonary venous return. Transesophageal echocardiography (TEE) is the imaging tool of choice to delineate the exact location and surrounding rims of the ASD. Furthermore, TEE can evaluate any associated cardiac anomalies and can determine suitability for catheter closure.

- **Cardiac catheterization** is not usually required for diagnosis. However, in older patients a diagnostic cardiac catheterization with full hemodynamic and coronary assessment may be justified.

Management: Transcatheter Device Closure of Secundum Atrial Septal Defect

A hemodynamically significant ASD requiring closure is said to exist if there is right atrial and ventricular volume overload. There is no real role for medical management of a hemodynamically significant ASD. In rare circumstances, a diuretic may be used to decrease pulmonary congestion, and afterload-reducing agents have a theoretical role in decreasing systemic relative to pulmonary vascular resistance (thereby decreasing the left-to-right shunt).

Currently in the USA the only FDA-approved device for catheter closure of secundum ASD is the Amplatzer® Septal Occluder device (AGA Medical Corporation, Golden Valley, MN). The Helex device (WL Gore Associates, Flagstaff, AZ) is still undergoing clinical trials.

In this chapter we will concentrate on the Amplatzer® Septal Occluder.

Indications. Secundum ASD with a significant left-to-right shunt as evidenced by right ventricular volume overload. Measurement of Qp:Qs is flawed, with a lot of errors. Therefore, we rely solely on TTE evidence of right-sided volume overload.

Contraindications.

- Patients with associated cardiac anomalies requiring cardiac surgery
- Patients with current systemic or local infection/sepsis within 1 month of device placement

- Patients with bleeding disorder, or with other contraindications to aspirin therapy, unless another antiplatelet drug can be administered for 6 months
- Presence of intracardiac thrombus
- Unsuitable defect anatomy including deficient rims superior/inferior or posterior. Deficient anterior rim is not a contraindication for the use of the Amplatzer®. We have closed defects with deficient either posterior or inferior rims as long as the other three rims are adequate
- Patients allergic to nickel may suffer an allergic reaction. This is a relative contraindication. Most nickel allergies are contact reactions. It is unclear if intracardiac devices will mount a similar reaction. A consultation with an allergist may be needed.

Device Description. The Amplatzer® Septal Occluder device is constructed from a 0.004–0.0075 inch Nitinol (55% nickel; 45% titanium) wire mesh tightly woven into two flat discs (Fig. 15-1). There is a 3–4 mm connecting waist between the two discs, corresponding to the thickness of the atrial septum. Nitinol has superelastic properties, with shape memory. This allows the device to be stretched into an almost linear configuration and placed inside a small sheath for delivery and then to return to its original configuration within the heart when not constrained by the sheath. The device size is determined by the diameter of its waist and is constructed in various sizes ranging from 4 to 40 mm (1 mm increments up to 20 mm; 2 mm increments up to the largest device currently available, 40 mm). The two flat discs extend radially beyond the central waist to provide secure anchorage.

Patients with secundum ASD usually have left-to-right shunt. Therefore, the left atrial (LA) disc is larger than the right atrial (RA) disc. For devices 4–10 mm in size, the LA disc is 12 mm and the RA disc is 8 mm larger than the waist. However, for devices larger than 11 mm and up to 34 mm in size, the LA disc is 14 mm and the RA disc is 10 mm larger than the connecting waist. For devices larger than 34 mm, the LA disc is 16 mm larger than the waist and the RA disc is 10 mm larger than the waist. Both discs are angled slightly towards each other to ensure firm contact of the discs to the atrial septum.

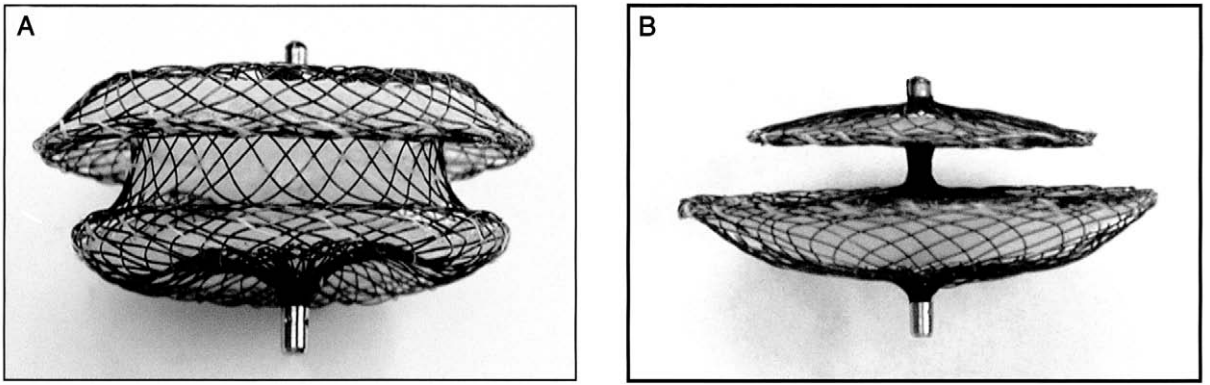

Fig.15-1 **A,** The Amplatzer® Septal Occluder, demonstrating the two discs (top left atrial, bottom right atrial disc) and the connecting waist. **B,** The Amplatzer® PFO Occluder, demonstrating the smaller left atrial disc on top and the larger right atrial disc on the bottom. Note the major difference in the size of the connecting waist between the two devices.

A total of three Dacron polyester patches are sewn securely with polyester thread into each disc and the connecting waist to increase the thrombogenicity of the device. A stainless steel sleeve with a female thread is laser-welded to the RA disc. This sleeve is used to screw the delivery cable to the device. For device deployment, we recommend using a 6 French delivery system for devices less than 10 mm in diameter, a 7 French delivery system for devices 10–15 mm; an 8 French sheath for devices 16–19 mm; a 9 French sheath for devices 20–26 mm; a 10 French sheath for devices 28–34 mm; a 12 French sheath for the 36 mm and 38 mm device and a 14 French sheath for the 40 mm device. Each device costs $3300.

Amplatzer® Delivery System. The delivery system is supplied sterilized and separate from the device. It contains all the equipment needed to facilitate device deployment. It consists of:

- Delivery sheath of specified French size and length, and appropriate dilator
- Loading device, used to collapse the device and introduce it into the delivery sheath
- Delivery cable (internal diameter [ID] 0.081 inch): the device is screwed onto its distal end and it allows for loading, placement and retrieval of the device
- Plastic Pin-vice: This facilitates unscrewing of the delivery cable from the device during device deployment
- Touhy-Borst Adapter with a side-arm for the sheath, to act as a one way stop-bleed valve.

All delivery sheaths have a 45° angled tip. The 6 French sheath has a length of 60 cm, the 7 French sheath is available in lengths of 60 and 80 cm and the 8, 9, 10 and 12 French sheaths are all 80 cm long. The Delivery System costs $350.

Amplatzer® Exchange (Rescue) System. This is made up of the same components as the Delivery System, the one exception being that the inner lumen and tip of the dilator can accommodate the delivery cable. It is available in two sizes: 9 French (dilator ID 0.087 inch) and 12 French (dilator ID 0.113 inch), with a 45° curve and 80 cm in length. The distal tip of the delivery cable can screw into the back of another delivery cable. This allows it to become an exchange length cable.

The damaged sheath then can be removed and the rescue sheath with its dilator can be inserted over the cable to recapture the device. The Exchange System costs $350.

Optional but Recommended Equipment

Amplatzer® Sizing Balloon. This is a double-lumen balloon catheter with a 7 French shaft size. The balloon is made from nylon and is very compliant, making it ideal for sizing secundum ASD by flow occlusion and preventing overstretching of the defect. The balloon catheter is angled at 45° and there are radio-opaque markers for calibration at 2, 5, and 10 mm. The balloon catheters are available in two sizes: 24 mm (maximum volume 30 ml and used to size defects up to 22 mm) and 34 mm (maximum volume 90 ml and used to size defects up to 40 mm). The cost is $195.

Amplatzer® Super Stiff Exchange Guidewire 0.035 inch is used to advance the delivery sheath and dilator into the left upper pulmonary vein. It costs $70.

Table 15-1 summarizes all the necessary materials for ASD closure.

Step by Step Technique: Transcatheter Device Closure of Secundum Atrial Septal Defect

Materials and Equipment

- Single- or bi-plane cardiac catheterization laboratory
- TEE or intracardiac echocardiography (ICE)

Table 15-1

Materials/Equipment Required for Transcatheter Procedures

Item	Size	Cost each ($)
Amplatzer® Septal Occluder	4–40 mm	3300
Amplatzer® PFO Occluder	18, 25, 35 mm	2800
Amplatzer® Delivery System	7–12 F	350
Amplatzer® Super Stiff exchange length guidewire	0.035 inch	70
Multipurpose catheter	6–7 F	10
Amplatzer® Sizing Balloon	24, 34 mm	195
Amplatzer® Rescue System	9, 12 F	350

- Full range of device sizes, delivery and exchange (rescue) systems
- Sizing balloon catheters
- A multipurpose catheter to engage the defect and the left upper pulmonary vein
- Super-stiff exchange length wire—we prefer the 0.035 inch Amplatzer® Super Stiff exchange length guidewire with a 1 cm floppy tip, but any extra-stiff J-tipped wire may be used.

Personnel

- Interventional cardiologist appropriately proctored to perform device closure
- Cardiologist—noninvasive to facilitate TEE or ICE
- Anesthesiologist, if procedure is performed under TEE guidance
- Nurse certified to administer unconscious sedation if performed under ICE guidance
- Catheterization laboratory technicians.

Method

Preprocedure. Review all pertinent data relating to the patient and to the defect to be closed and ensure that appropriate devices and delivery systems are available. The procedure and complications should be explained and opportunity given to ask questions. All preprocedure orders should be given to the patient. Aspirin 81–325 mg should be started 48 hours prior to the procedure. If allergic to aspirin, clopidogrel 75 mg should be used.

1. Access. The right femoral vein is accessed using a 7–8 French short sheath. An arterial monitoring line can be inserted in the right femoral artery, especially if the patient's condition is marginal or if the procedure is performed under TEE and general endotracheal anesthesia. If the femoral venous route is not available, we advocate the transhepatic approach. If a subclavian or internal jugular venous approach is used it is very difficult to maneuver the device deployment, especially with large defects.

We administer heparin to achieve an activated clotting time (ACT) of more than 200 sec at the time of device deployment. Antibiotic coverage for the procedure is recommended. We

usually use cefazolin 1 g intravenously, the first dose at the time of procedure and two subsequent doses 6–8 hours apart.

Routine right heart catheterization should be performed in all cases to ensure presence of normal pulmonary vascular resistance. The left-to-right shunt can also be calculated.

Echocardiographic assessment of the secundum ASD should be performed simultaneously using either TEE or ICE. A comprehensive study should be performed, looking at all aspects of the ASD anatomy (location, size, presence of additional defects, and adequacy of the various rims). Figure 15-2 demonstrates full assessment of the defect by ICE.

2. Rims. The important rims to look for are:

- Superior/SVC rim—best achieved using the bicaval view
- Superior posterior/right upper pulmonary vein rim
- Anterior superior/aortic rim—the least important rim; often, patients lack it
- Inferior/IVC and coronary sinus rim—an important rim to have
- Posterior rim—seen best in the short-axis view at the aortic valve level.

3. How to cross the atrial septal defect. Use a multipurpose catheter: the MP A2 catheter has the ideal angle. Place the catheter at the junction of the IVC and the right atrium. The IVC angle should guide the catheter to the ASD. Keep a clockwise torque on the catheter while advancing it towards the septum (posterior). If unsuccessful, place the catheter in the SVC and slowly pull it into the right atrium; keep a clockwise posterior torque to orient the catheter along the atrial septum until it crosses the defect. TEE/ICE can be very useful to guide the catheter across difficult defects.

4. Right upper pulmonary vein angiogram. It can be useful to perform an angiogram in the right upper pulmonary vein (Fig 15-3A) in the hepatoclavicular projection (35° left anterior oblique/35° cranial). This delineates the anatomy, shape, and length of the septum. This may come in handy when the device is deployed but not released—the operator can position the I/I in the same view of the angiogram and compare the position of the device with that obtained during the deployment (Fig. 15-3B, C).

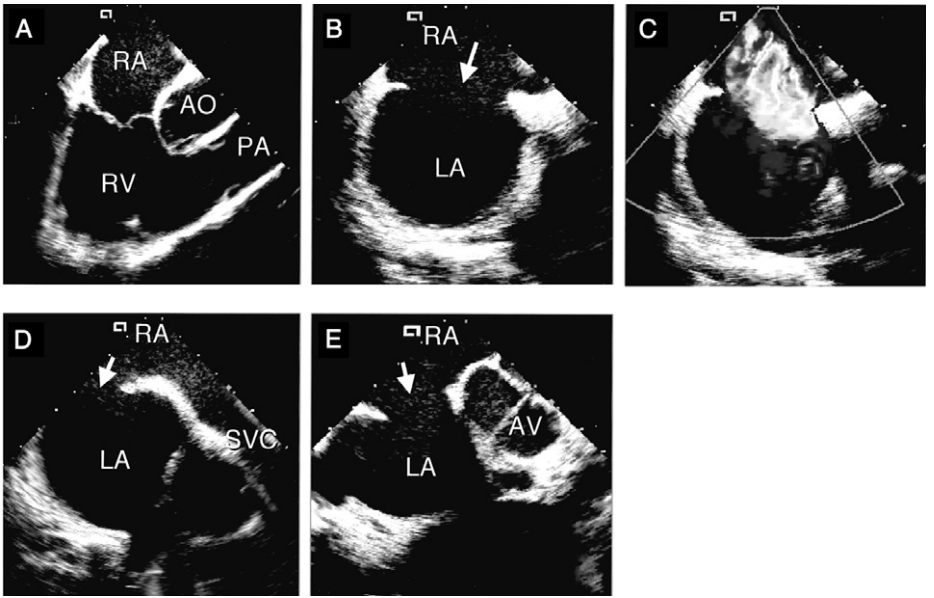

Fig.15-2 Intracardiac echocardiographic images in a patient with a large secundum atrial septal defect (ASD). **A,** Home view demonstrating the right atrium, tricuspid valve, right ventricle, aortic root, and pulmonary artery. **B,** Septal view, demonstrating the large ASD (arrow), the right and left atria, and the superior and inferior rims. **C,** Same view with color Doppler. **D,** Caval view demonstrating the entire superior rim and the defect (arrow). **E,** Short-axis view demonstrating the defect (arrow), the aortic root, the absent anterior rim and good posterior rim, and both atria.

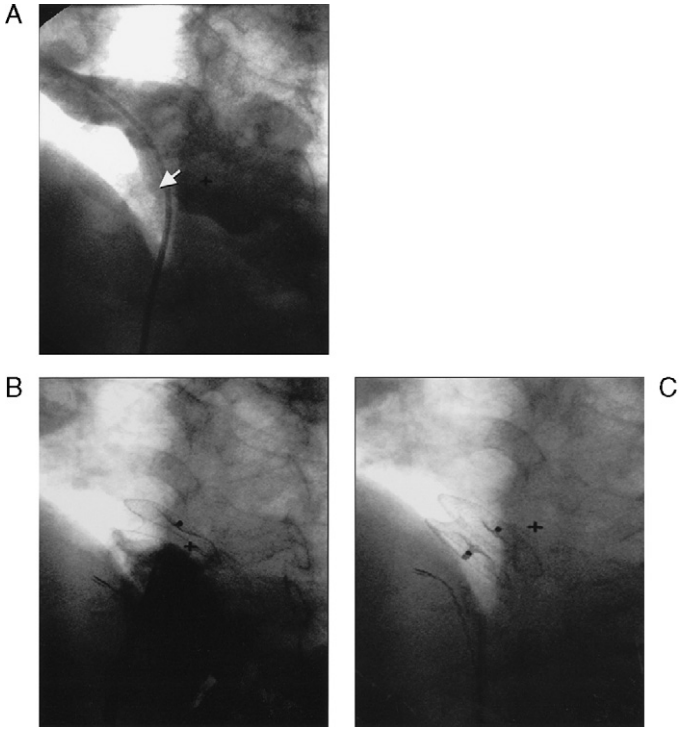

Fig.15-3 Cine angiographic images in a patient with secundum ASD. **A,** Angiogram in the right upper pulmonary vein in the hepatoclavicular projection (35° left anterior oblique/35° cranial) demonstrating left-to-right shunt. **B,** Angiogram in the right atrium in the hepatoclavicular projection prior to release of the device. Correct deployment manifest by opacification of the right atrial disc only and on levophase the left atrial disc only opacified. **C,** Cine image after the device has been released demonstrating good device alignment with the septum.

5. Defect Sizing. Position the multipurpose catheter in the left upper pulmonary vein. Prepare the appropriate size of balloon according to the manufacturer's guidelines. We prefer to use the 34 mm balloon since it is longer and during inflation it sits nicely over the defect. Pass an extra-stiff, floppy/J-tipped 0.035 inch exchange length guidewire (Fig. 15-4A). This gives the best support within the atrium for the balloon, especially in large defects. Remove the multipurpose catheter and the

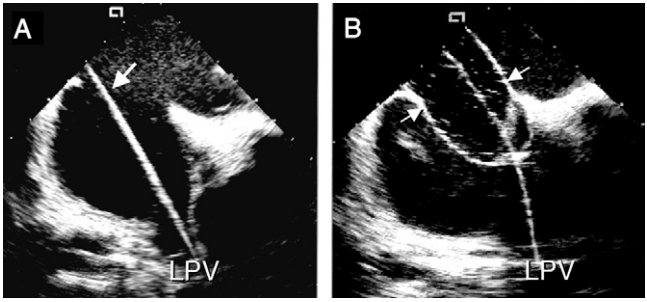

Fig.15-4 Intracardiac echocardiographic images in the patient in Figure 15-2, showing defect sizing. **A,** The exchange wire (arrow) across the defect into the left upper pulmonary vein. **B,** Sizing balloon occluding the defect. This is the stretched diameter (arrows) of the defect.

femoral sheath. We advance the sizing balloon catheter directly over the wire without a venous sheath. Most sizing balloons require an 8 or 9 French sheath. The balloon catheter is advanced over the wire and placed across the defect under both fluoroscopic and echocardiographic guidance. The balloon is then inflated with diluted contrast until the left-to-right shunt ceases, as observed by color flow Doppler TEE/ICE (flow occlusion). The best echo view for measurement is to observe the balloon in its long axis (Fig. 15-4B). In this view the indentation made by the margins of the ASD can be visualized and precise measurement made.

6. Fluoroscopic measurement. Angulate the x-ray tube so the beam is perpendicular to the balloon. This can be difficult but the various calibration markers can help. Ensure that the markers are separated and discrete. Measure the balloon diameter at the site of the indentation as per the diagnostic function of the laboratory (Fig. 15-5). If a discrepancy exists between the echocardiographic and the fluoroscopic measurements we have found that the echocardiographic measurement is usually more accurate.

Once the size is determined, deflate the balloon and pull it back into the junction of the right atrium and IVC, leaving the wire in the left upper pulmonary vein.

This is a good time to recheck the ACT and give the first dose of antibiotics.

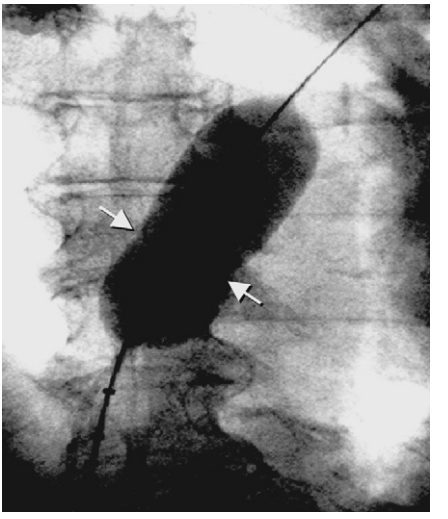

Fig.15-5 Cine angiographic image of the patient in Figure 15.3 during balloon sizing of the defect, demonstrating the stretched diameter (arrows) of the defect.

7. Device selection. If the defect has adequate rims (>5 mm), select a device 0–2 mm larger than the stretched diameter of the balloon. However, if the superior/anterior rim is deficient (5–7 mm), select a device 4 mm larger than the balloon stretched diameter.

8. Device Delivery. Once the device size is selected, open the appropriate-sized delivery system. Flush the sheath and dilator. The proper size of delivery sheath is advanced over the guidewire to the left upper pulmonary vein (Fig. 15-6A). Both dilator and wire are removed, keeping the tip of the sheath inside the left upper pulmonary vein. Extreme care must be exercised not to allow passage of air inside the delivery sheath. An alternative technique to minimize air embolism is passage of the sheath with the dilator over the wire until the IVC, at which point the dilator is removed and the sheath is advanced over the wire into the left atrium while continuously flushing the side arm of the sheath.

The device is then screwed to the tip of the delivery cable, immersed in normal saline and drawn into the underwater seal

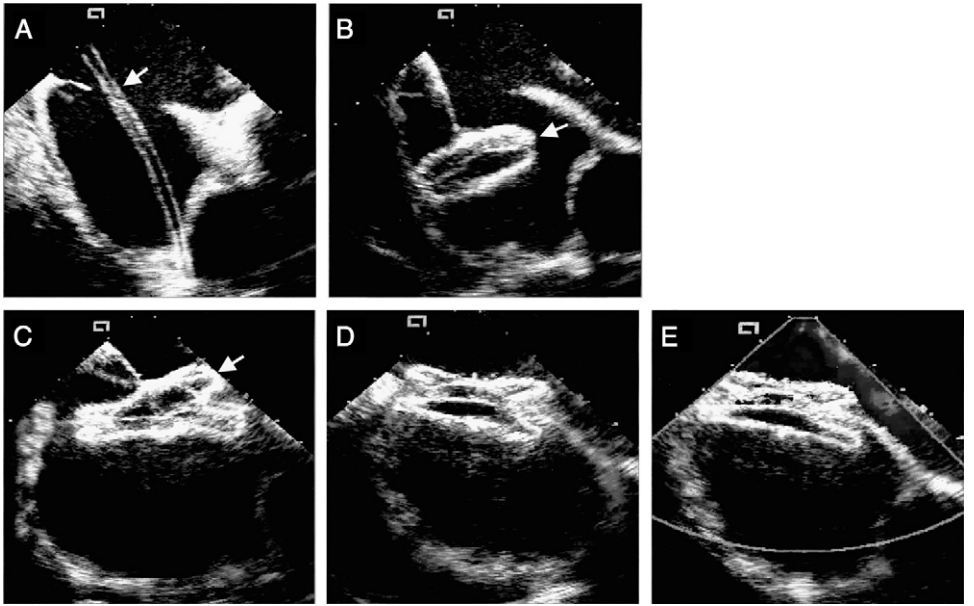

Fig.15-6 Intracardiac echocardiographic images in the patient in Figure 15-2, showing device delivery and deployment. **A,** Delivery sheath (arrow) across the defect into the left upper pulmonary vein. **B,** The left atrial disc (arrow) deployed in the left atrium. **C,** The right atrial disc (arrow) deployed in the right atrium. **D,** The device released, demonstrating good position **E,** Color Doppler demonstrating no residual shunt and patent superior vena cava.

of the loader to expel air bubbles out of the system. A Y connector is applied to the proximal end of the loader to allow flushing with saline. The loader containing the device is attached to the proximal hub of the delivery sheath. The cable with the Amplatzer® Septal Occluder device is advanced to the distal tip of the sheath, taking care not to rotate the cable while advancing it in the long sheath to prevent premature unscrewing of the device. Both cable and delivery sheath are pulled back as one unit to the middle of the left atrium. Position of the sheath can be verified using the cine fluoroscopy or TEE/ICE.

9. Device Deployment. The LA disc is deployed first under fluoroscopic and or echocardiographic guidance (Fig. 15-6B). Caution should be taken not to interfere with the LA appendage. Part of the connecting waist should be deployed in the left atrium, very close (a few millimeters) to the atrial septum (the mechanism of ASD closure using the Amplatzer® Septal Occluder is stenting of the defect). While applying constant pulling of the entire assembly and withdrawing the delivery sheath off the cable, the connecting waist and the RA disc are deployed in the ASD itself and in the right atrium respectively (Fig. 15-6C).

10. Device Positioning. Proper device position can be verified using different techniques:

- *Fluoroscopy in the same projection as that of the angiogram.* Good device position is evident by the presence of two discs that are parallel to each other and separated from each other by the atrial septum. In the same view the operator can perform the "Minnesota Wiggle" (the cable is pushed gently forward and pulled backward). Stable device position manifests by the lack of movement of the device in either direction.
- *TEE/ICE*. The echocardiographer should make sure that one disc is in each chamber. The long-axis view should be sufficient to evaluate the superior and inferior part of the septum and the short-axis view for the anterior and posterior part of the disc.
- *Angiography*. This is done with the camera in the same projection as for the first angiogram to profile the septum and device using either the side arm of the delivery sheath or

a separate angiographic catheter, inserted in the sheath used for ICE or via a separate puncture site. Good device position manifests by opacification of the RA disc alone when the contrast is in the right atrium and opacification of the LA disc alone on pulmonary levophase.

If the position of the device is not certain, or is questionable, after all these maneuvers, the device can be recaptured, entirely or partly, and repositioned following similar steps.

11. Device Release. Once the device position is verified, the device is released by counterclockwise rotation of the delivery cable using a pin vise. There is often a notable change in the angle of the device as it is released from the slight tension of the delivery cable and it self-centers within the ASD and aligns with the interatrial septum. To assess result of closure, repeat TEE/ICE, color Doppler and angiography in the four-chamber projection in the right atrium with pulmonary levophase are performed. Patients receive a dose of an appropriate antibiotic (commonly cephazolin 1 g) during the catheterization procedure and two further doses at 8-hour intervals. Patients are also asked to take endocarditis prophylaxis when necessary for 6 months after the procedure, as well as aspirin 81–325 mg orally once daily for 6 months. Full activity, including competitive sports, is usually allowed after 4 weeks of implantation. Magnetic resonance imaging (if required) can be performed any time after implantation.

12. Postprocedure. Once the procedure is complete, recheck the ACT and, if appropriate, remove the sheath and achieve hemostasis. If ACT is above 250 sec, reverse the effect of heparin by using protamine sulfate.

Procedure Monitoring. Patients recover overnight in a telemetry ward. Some patients may experience an increase in atrial ectopic beats. Rarely, some patients may have sustained atrial tachycardias. Resume aspirin therapy 81–325 mg per day after the procedure and continue it for 6 months.

The following day an ECG, a chest x-ray (posteroanterior and lateral) and a TTE with color Doppler should be performed to assess the position of the device and presence of residual shunt.

A repeat chest x-ray 1 week after the procedure to look for device position is recommended.

Recheck ECG, chest x-ray and TTE/TEE at 6 months post-procedure to assess everything. If device position is good, with no residual shunt, followup can be annual for the first 2 years, then every 3–5 years. Long-term followup of device performance should be assessed and any new information communicated to the patient.

The patient is asked not to engage in contact sports for 1 month after the procedure.

Procedure Medication. Aspirin is given as described above. Infective endocarditis prophylaxis should be given, when needed, for 6 months. After 6-months, if there is no residual shunt, prophylaxis and aspirin can be discontinued.

Troubleshooting

Air Embolism. Meticulous technique should be used to prevent air entry. The sheath should be positioned at the mouth of the left upper pulmonary vein. Doing so allows free flow of blood into the sheath. Forceful negative pressure should not be applied to aspirate the sheath. If a large amount of air is introduced on the left side, it will usually pool in the right coronary sinus and right coronary artery. This may manifest with bradycardia, asystole, or profound hypotension. If this occurs, immediately place a right coronary catheter in the right coronary sinus and forcefully inject saline or contrast to displace the air and hence reperfuse the right coronary system.

Cobra-Head Formation. This describes the situation when the left disc maintains a high profile when deployed, mimicking a cobra head. This can occur if the left disc is opened in the pulmonary vein or the left atrial appendage, or if the left atrium is too small to accommodate the device. It can also occur if the device is defective or has been loaded with unusual strain on it. If this occurs, check the site of deployment; if appropriate, recapture the device and remove and inspect it. If the "cobra head" forms outside the body, use a different device. If the disc forms normally, try deploying the device again. Do not release a device if the left disc has a "cobra-head" appearance.

Device Embolization. If a device embolizes, it has to be removed. This can be done surgically or by transcatheter snare and recapture into a long sheath. The transcatheter technique is difficult and should not be performed if the operator is inexperienced in snaring techniques. Furthermore, the catheter laboratory should be equipped with large Mullins-type sheaths (12–14F) and also should have various-sized snares. We use the goose-neck snare (Microvena Corporation) or the Ensnare (MD Technologies). The device should not be pulled across valves, since it may damage the chordae and leaflets. Always use a long sheath to pull the device outside the body. To snare a device, we usually use a sheath 2 French sizes larger than the sheath that was used to deliver the device. On rare occasions, if the LA disc cannot be collapsed inside the sheath, another snare is introduced from the right internal jugular vein to snare the stud of the microscrew of the LA disc and stretch it towards the internal jugular vein while the assistant pulls the device with snare towards the femoral vein. This allows the device to collapse further and come out of the sheath in the femoral vein.

Prolapse of the Left Disc Across the Defect During Deployment. On occasions, especially in patients with large defects with deficient anterior/superior rims, when the left disc is deployed it opens perpendicular to the plane of the atrial septum and prolapses through the anterior superior part of the defect. To overcome this problem, use a device in these cases that is at least 4 mm larger than the measured balloon diameter. If this is not possible, or it does not work, change the angle of the deployment by placing the sheath in the right upper pulmonary vein rather than the left. This may change the orientation of the disc. Another potential solution is to use a long sheath with a sharper curve that is stiffer (Mullins sheath) and rotate it posteriorly in the left atrium. Cook Inc. has developed a new sheath, the Hausdorf Sheath, which has a sine curve that can be quite useful in changing the deployment angle.

Recapture of the Device. To achieve the smallest sheath size for device delivery, sheath wall thickness is small, with a resultant decrease in strength. To recapture a device prior to its release,

the operator should hold the sheath at the groin with the left hand and with the right hand pull the delivery cable forcefully inside the sheath. If the sheath is damaged/kinked (accordion effect) use the exchange (rescue) system to change the damaged sheath. First, extend the length of the cable by screwing the tip of the rescue cable to the proximal end of the cable attached to the device. Then remove the sheath or, if the sheath is 9 or 12 French, introduce the dilator of the rescue system over the cable inside the sheath until it reaches few centimeters from the tip of the sheath. This dilator will significantly strengthen the sheath, allowing the operator to pull back the cable with the dilator as one unit inside it. Then the operator can decide what to do next (change the entire sheath system or the device).

Release of the Device with a Prominent Eustachian Valve. To avoid the possibility of cable entrapment during release, advance the sheath to the hub of the right disc. Then release the cable and immediately draw back inside the sheath before the position of the sheath is changed.

Closure of Multiple Secundum Atrial Septal Defect. If two defects are present and separated by more than 7 mm from each other, cross each defect separately. Size each one and then leave a delivery system in each defect. Initially deploy the smaller device, then the larger device, and release sequentially, starting with the smaller one.

If there are multiple fenestrations, use the Amplatzer® Multi-Fenestrated Septal Occluder—"Cribriform" (these devices are similar in design to the Amplatzer® PFO Occluder except that the two discs are equal in size). These devices are still investigational. The device should be deployed in the middle of the septum so that it can cover all fenestrations.

Complications

In the US phase II trial comparing device to open surgical closure, the incidence of complications was 7.2% for device closure, far less than what was encountered when using an open surgical technique (24%). Most complications were related to rhythm disturbance, with very few patients requiring long-term medical therapy.

- **Device embolization**, the majority of which were encountered during the early learning curve of the investigators
- **Heart block**: rarely reported (one patient)
- **Atrial arrhythmia**: significant increase in atrial arrhythmias following device placement, resolving by 6 months
- **Headaches**: reported in about 5% of patients following device placement, resolving within 6 months.

Results

Closure rates were similar to those achieved by open surgical results. However, patients who underwent device closure were somewhat older than those who underwent open surgical closure. Furthermore, the cost of device closure was much less than open surgical closure and the length of hospital stay was shorter (1 day) for the device group than the surgical group (3.4 days). In a study by Kim and Hijazi, the mean cost for transcatheter closure was $11,541 and for surgical closure was $21,780.

PATENT FORAMEN OVALE

A patent foramen ovale (PFO) is normal during fetal development. Following birth, with an increase in pulmonary blood flow and higher left atrial pressure than right atrial pressure, the foramen ovale is physiologically closed. The foramen is created by the overlap of the septum primum and septum secundum (Fig. 15-7). This behaves like a flap valve, which opens if the right atrial pressure exceeds the left atrial pressure and closes if the reverse is true. Pathologic studies have suggested that the foramen ovale may be probe-patent in 25% of the population.

There are three anatomic types:

- "Flap" type
- Tunnel type
- Aneurysmal septum primum with a PFO.

Clinical Significance

A PFO is a potential source for right-to-left intracardiac shunt and can result in paradoxical emboli. Presentation is usually in the third or fourth decade. Rarely it can present in adolescence.

Cerebrovascular Accident. Paradoxical arterial emboli leading to neurological deficit (or stroke), or other systemic emboli, is the

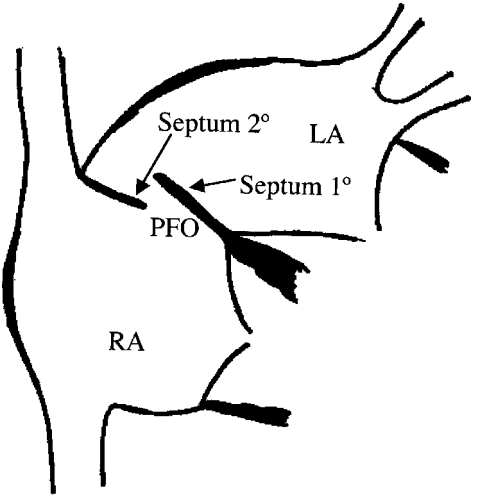

Fig.15-7 Schematic sketch demonstrating the patent foramen ovale as a flap-like structure created between septum primum (1°) and septum secundum (2°).

most easily recognized manifestation of PFO. Cryptogenic stroke (stroke with no identifiable cause) accounts for 40% of stroke in young adults. Contrast echocardiography has demonstrated a higher than normal prevalence of PFO in cryptogenic stroke patients less than 55 years old. This is not the case in patients older than 55 years. In addition, the presence of an aneurysmal septum primum with a PFO enhances this risk. The presence of a prominent eustachian valve of the IVC has also been postulated to enhance the risk for paradoxical emboli in the presence of a PFO. The eustachian valve is designed to direct the IVC flow toward the PFO, although no clear evidence exists for this hypothesis.

The recently reported PFO in Cryptogenic Stroke Study provides data from a large cohort of patients in a prospective manner. A PFO was present in 39% of the patients with cryptogenic stroke compared to 29% in patients with an identifiable cause for the stroke. Large PFOs (≥2 mm separation between septum primum and secundum or ≥10 microbubbles appearing in the left atrium with a contrast TEE) were present in 20% of the cryptogenic group versus 9.7% of the patients with an identifiable cause for their stroke.

Conditions Increasing Right-to-Left Shunt. Conditions associated with elevated right atrial pressure will enhance the potential for a right-to-left shunt. For example, chronic restrictive pulmonary or recurrent pulmonary embolus, hypercoagulable states, prothrombin gene G20210A, factor V Leiden mutations, anticardiolipin antibodies, protein S and C deficiencies, and thrombocytosis may promote venous thrombosis and increase the chance of a paradoxical embolus occurring.

Divers. An interesting group of patients are those who are deep-sea or scuba divers. This population may report decompression sickness with unusual symptoms despite following an appropriate and rigid protocol during a dive ascent. An incidence of neurologic symptoms as high as 61% has been reported. Decompression problems have also led to more brain defects in individuals with PFO than without. Migraine has a higher prevalence in patients with PFO—57% in one study. PFOs are a potential for postoperative complications in surgical procedures prone to venous fat or air embolism.

Risk of Recurrence After a Presumed Cryptogenic Transient Ischemic Attack or Stroke. A recurrence risk of 3–5% has been reported in most series with medical management of embolic stroke. However, the Warfarin–Aspirin Recurrent Stroke Study (WARSS) yielded a recurrent event or death over a 2-year period of 15% with warfarin and 17% with aspirin when the subset of patients with cryptogenic stroke only was analyzed. The Mayo Clinic reported a 4.1% recurrence rate of any neurologic event following surgical closure of PFO. It is likely that a percentage of these represent patients in whom the hypothesis that their problem resulted from a paradoxical embolus was incorrect.

Transcatheter Closure of Patent Foramen Ovale

Two devices are currently designed to specifically close PFO: the Amplatzer® PFO Occluder and the PFO Star devices. The CardioSEAL (NMT Medical) and the Helex (WL Gore Associates) were designed for ASD closure; however, they have also been used for PFO closure.

The Amplatzer® PFO Occluder is a self-expandable, double-disc device made from a Nitinol wire mesh (Fig. 15-1). The nitinol mesh wire is 0.005–0.006 inch in diameter.

The two discs are linked together by a connecting waist 2 mm in diameter and 4 mm in length. This thin waist allows free motion of each disc so that the device can conform to the PFO shape and position the two discs in the plane of the atrial septum. The discs are filled with a polyester fabric sewn securely to each disc by a polyester thread. The polyester increases the closing ability of the device by trapping blood forming the initial plug and then promoting "endothelialization" of the device.

The devices are available in three sizes, 18, 25 and 35 mm, corresponding to the diameter of the right disc. The diameter of the left disc is 18 mm for the 18 and 25 mm and 25 mm for the 35 mm device. The connecting waist is the same for both—2 mm in diameter and 4 mm in length.

The devices are packaged individually and supplied sterilized ready for use. The device costs $2800.

Amplatzer® Delivery System. This is the same as the secundum ASD delivery systems and the PFO occluder devices will deploy through 7–9 French sheath. The Delivery System costs $350. The device is available in the USA and is under two study protocols, which therefore determine the indications.

- **Humanitarian device exemption.** The Amplatzer® PFO Occluder is authorized by Federal law for the nonsurgical closure of a PFO in patients with recurrent cryptogenic stroke due to presumed paradoxical embolus through a PFO and who have failed conventional medical therapy. Appropriate IRB approval has to be obtained at each institution and the physician performing the procedure has to be trained in transcatheter closure techniques by proctors identified by AGA Medical Corporation.
- **Multicenter investigational device exemption, phase II study.** This is a randomized multicenter prospective study, with patients enrolled into two study arms: medical therapy versus transcatheter closure of the PFO. Patients can be enrolled following a cryptogenic stroke in the presence of a PFO. The trial's name is RESPECT (Randomized Evaluation of recurrent Stroke, TIA, or peripheral embolism comparing PFO closure to Established Current standard of care Treatment).

Contraindications. These are the same as for secundum ASD.

Step by Step Technique: Transcatheter Closure of Patent Foramen Ovale

Materials, Equipment, and Personnel. These are the same as for secundum ASD. The preprocedure evaluation is also the same.

Access. Place a 7 French sheath in the right femoral vein. An arterial monitoring line can be useful if procedure is performed under TEE with general anesthesia. Administer a full heparin dose and administer antibiotics as for secundum ASD. Perform a right heart hemodynamic study. Perform a TEE/ICE to assess the anatomy of the PFO and to perform a contrast bubble study with and without Valsalva maneuver.

The septal length and the distance of the surrounding structures, especially the free wall of the atrium, should be measured. The type of PFO—simple flap type, tunnel type or PFO with an aneurysmal septum primum—should be determined.

Suggested Measurements with TEE/ICE Images

- Total septal length and edge of the defect to the mitral valve in the four-chamber view
- SVC to the edge of the defect (long-axis TEE view/caval view by ICE)
- Edge of defect to the aorta in short-axis view.

Do *not* implant a device if the distance either from the defect to the SVC or from defect to the aortic root is *less than 9 mm.*

Device Selection. For defects without an aneurysmal septum primum use the 18 or 25 mm device, depending on the length of the septum. If there is a significant aneurysm or if the septum primum appears very thin and floppy, use the 35 mm device as long as both the distance from the SVC to the edge of the defect and from the edge of the defect to the aortic root is more than 17.5 mm. If there is aneurysm and distance from edge of defect to either SVC or aortic root is 12.5–17.5 mm, the 25 mm device should be used.

Procedure Steps. The procedure is identical to that described for secundum ASD except that balloon sizing is not performed. Prior to release, careful assessment should be made of the edge of the device along the free atrial wall by TEE/ICE. Do not

release the device if it does not conform to its original configuration or if it appears unstable. Recapture and redeploy. If the problem persists, recapture the device and use a new one. Figures 15-8 and 15-9 demonstrate the steps of PFO closure.

Postprocedure Followup. This is similar to secundum ASD except that most investigators maintain 81–325 mg aspirin per day for 6 months in combination with an antiplatelet agent, usually clopidogrel 75 mg/day, for 1–6 months. Followup echocardiogram at 3–6 months should include assessment for right-to-left atrial level shunt with a venous contrast injection, with Valsalva maneuver.

Complications

Right Atrial and Aortic Perforation. A total of two patients in the worldwide data (none in the USA experience) had this complication. There was one case report by Trepel *et al.* in a

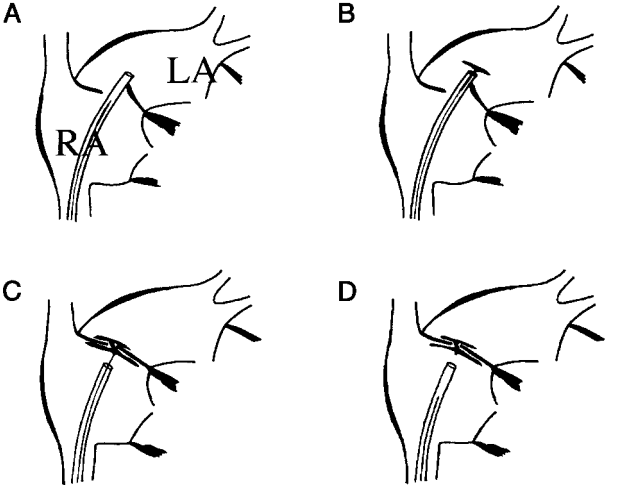

Fig.15-8 Schematic demonstration of patent foramen ovale closure using the Amplatzer® PFO Occluder. **A**, Sheath in mid left atrium (LA). **B**, Deployment of left atrial disc. **C**, Deployment of connecting waist and right atrial disc. **D**, Device released.

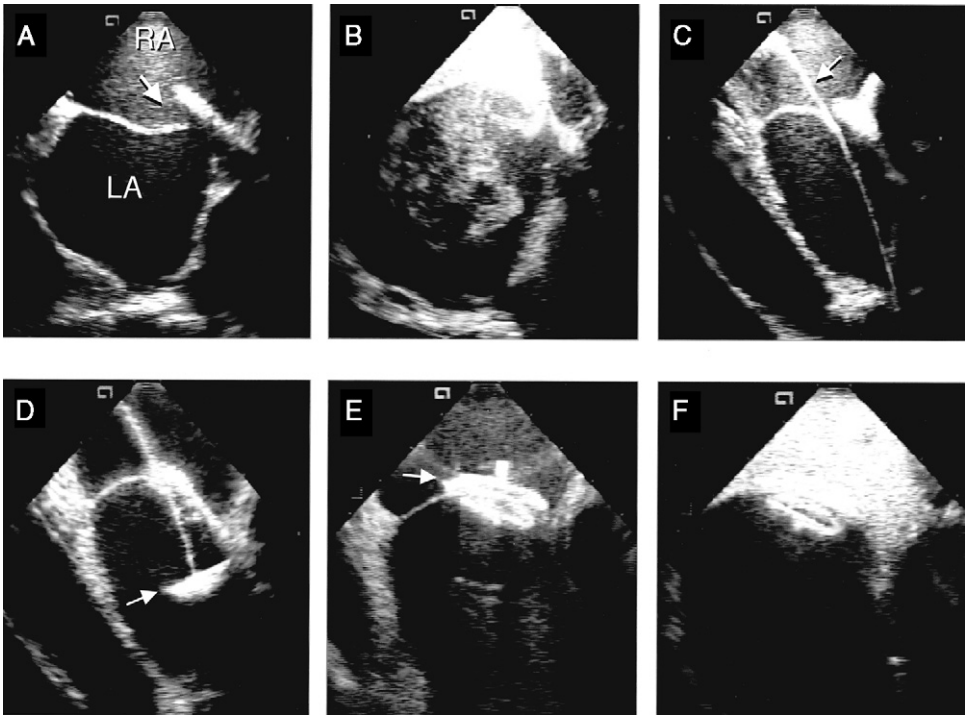

Fig.15-9 Intracardiac echocardiographic images in a young patient who has a patent foramen ovale (PFO), demonstrating the steps of closure. **A,** Septal view demonstrating the PFO (arrow) and the thin septum primum. **B,** Contrast bubble study demonstrating significant right-to-left shunt. **C,** Guidewire (arrow) positioned through the defect into the left pulmonary vein. **D,** Deployment of atrial the left disc (arrow). **E,** Deployment of the right atrial disc (arrow); release of the device. **F,** Contrast bubble study repeated after the device has been released demonstrating negative bubbles.

patient who presented with pericardial tamponade. At surgery, erosion of the right atrial roof and aortic root were noted. Following this report, the company introduced the septal measurements, emphasizing the distance of the free right atrial wall from the defect/device.

Entrapment of Prominent Eustachian Valve on the Delivery Cable. This caused no problems with device delivery and release but part of the Eustachian valve was avulsed. To avoid this, prior to release advance the sheath to the hub of the right side disc.

Results

During phase I of the US clinical trial, closure rates were in excess of 95% at 3–6 months followup. There were no complications related to the device. The length of hospital stay was about 1 day. No episodes of atrial arrhythmias have been reported.

Suggested Readings

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16

INTERVENTIONS FOR THE FAILING HEMODIALYSIS VASCULAR ACCESS

Steven J. Bander, Suresh Kumar Margassery, and Kevin J. Martin

INTRODUCTION

Percutaneous endovascular techniques are increasingly used as alternatives to surgical methods to maintain hemodialysis access. Numerous studies have found equivalent results between conventional surgical treatment and endovascular techniques. These two approaches are complementary and not mutually exclusive: the surgeon and interventionalist should work together as a team. The method chosen depends upon factors specific to the dialysis practice, such as caseload, availability, local expertise with the available treatment options, and patient preference.

Timing of Procedure

Endovascular procedures can usually (and should) be performed on the same day that the access problem is discovered, thus avoiding the need for a temporary hemodialysis catheter or a missed dialysis treatment. By using fistulography, the interventionalist can evaluate the graft, veins, and arterial inflow of the patient, revealing stenoses and suboptimal veins that may be not appreciated during a surgical procedure. Fistulography allows surgeons to perform more extensive and definitive repairs or revisions to the vascular access, thus providing treatments that are not possible using endovascular approaches.

Mechanisms of Graft Failure

Most hemodialysis access failures are due to the gradual development of two different types of lesion: intimal hyperplasia or intragraft stenosis. Intimal hyperplasia occurs 60% of the time at the venous anastomosis, although the lesions can be found anywhere from the arterial anastomosis to anywhere along the venous outflow. The characteristic lesion is a concentric, focal thickening of the vessel wall consisting of extracellular matrix and smooth muscle cells. When compared to atherosclerotic arterial lesions, intimal hyperplastic lesions are more difficult to dilate and tend to recur very quickly. Intragraft stenoses are distinct from venous anastomotic lesions. They are pseudointimal lesions composed of fibrin, plasma proteins, and red blood cells. Most intragraft stenoses occur at previous needle puncture sites and are very amenable to angioplasty.

Vascular Access Surveillance

Implementation of a vascular access surveillance program in the dialysis unit is crucial for maintaining and optimizing the performance and longevity of hemodialysis access. Intra-access blood flow monitoring is the most sensitive and specific method for the detection of access stenoses. Although there is substantial variability from patient to patient, a well functioning dialysis graft has a blood flow rate of 1000–1300 ml/min. The average blood flow in an arteriovenous fistula is 700–1000 ml/min.

A baseline measurement of blood flow should be obtained after the access is first placed. Sequential blood flow measurements should be done on a quarterly basis. The magnitude and rate of decrease in blood flow are predictive of impending thrombosis. If the access flow is less than 600 ml/min or if the access is less than 1000 ml/min and has decreased by more than 25% over a 3-month period, the patient should be referred for a fistulogram and possible endovascular intervention.

DIAGNOSTIC FISTULOGRAM

The diagnostic angiogram is performed to evaluate the internal structure of the hemodialysis graft as well as the entire vascular

access circuit, from the arterial anastomosis to the superior vena cava. A hemodynamically significant stenosis is defined as a 50% reduction of vessel diameter on two-dimensional imaging. This usually corresponds to a 75–80% decrease in cross-sectional area. Multiple angiographic images, obtained in different imaging planes, may be necessary to thoroughly evaluate the lesion before proceeding with any intervention. The angiogram may also show the presence of collateral channels surrounding the stenosis, a finding that strongly suggests that the lesion is hemodynamically significant. It is imperative to visualize the entire venous outflow, including the central venous system, to exclude the presence of occult occlusive disease. There is a significant incidence of central vein stenosis in patients who have previously undergone placement of subclavian or internal jugular vein placement of catheters. Inspection of the arterial inflow must also be performed because a recent study has shown that 28% of dysfunctional hemodialysis access has arterial problems.

Procedure

1. **Preparing the access site.** Establish the point where the access will be entered. It is usual to access the graft or fistula near the arterial anastomosis. To determine which end is the arterial anastomosis, occlude the blood flow with pressure in the middle of the graft or fistula. With the flow occluded palpate each anastomotic end to feel for an arterial pulse. Pulsation will indicate the arterial anastomosis and the one without flow the venous limb. Clean the skin surface with chlorhexidine or povidone-iodine and apply a sterile drape.
2. **Accessing the graft.** Use a syringe with a 25 gauge needle to raise a wheal with a small amount of 1% lidocaine at the proposed entry site. Use a small vessel access set—4 or 5 French (Microvascular Access Set, Cook, Inc., Bloomington, IN) that comes with all the components. Grasp the graft or fistula between the thumb and third finger of the hand holding the needle and advance the needle at a 45° angle until you feel a definite "pop" of the needle passing through the graft or fistula wall and see back flash of blood. You should then have easy passage for the 0.018 inch Nitinol guidewire. Place the 4 or 5 French catheter and inner stylet over the guidewire until it is well within the graft or fistula.

Remove the inner stylet and the guidewire. Attach intravenous extension tubing with an injection port or three-way stopcock.

3. Several small hand injections of contrast (5–10 ml), utilizing multiple projections, are usually required to visualize the graft, venous anastomosis, and complex collateral network of venous outflow channels up to and including the superior vena cava.
4. To assess the arterial inflow, direct pressure with a finger, a tourniquet, or blood pressure cuff inflated to a pressure exceeding systemic blood pressure will interrupt flow and allow contrast to reflux into the arterial limb of the graft or fistula, enabling visualization of the arterial anastomosis and arterial inflow.

BALLOON ANGIOPLASTY FOR MAINTENANCE OF ACCESS PATENCY

Percutaneous transluminal angioplasty is the most commonly performed endovascular procedure for the repair or maintenance of a failing or thrombosed vascular access. Stenosis involving the venous anastomosis or the venous outflow tract is the predominant reason for vascular access failure. The art of angioplasty is reflected in the varying opinions concerning the length of stenosis that should be dilated. Several reports have suggested that angioplasty of longer stenoses (>4 cm) was less successful and resulted in shorter long-term patency than shorter, focal lesions (<2 cm).

Procedural Recommendations

Balloon angioplasty is readily accomplished at the time of diagnostic angiography. An appropriately sized sheath (usually 7F) is advanced over a guidewire. Sheath size will be guided by the selection of balloon catheter. An appropriate selection of guidewires is necessary and should be available in a range of sizes (0.024–0.038 inch), varying lengths (140–270 cm), and varying qualities (hydrophilic, stiff, steerable). Angioplasty catheters of varying size (balloon diameter, catheter length, balloon length, shaft sizes, and burst pressures) should also be available.

Performance of balloon angioplasty is straightforward when certain principles are applied. One absolute is to maintain

guidewire access across the lesions and limit the advancement of the catheter to an over-the-wire technique. Balloon inflation is performed using a dilute (50/50 mix) solution of contrast and a syringe with a pressure monitoring capability. Each type of balloon has its own unique burst pressure, which should not be exceeded during inflation. Most lesions will dilate completely at 8–10 atm. However, a number of stenoses will require 17–20 atm to fully dilate the lesion; but it is sometimes necessary to exceed pressures of 25–30 atm to dilate resistant lesions. In addition cutting balloons are currently under investigation for peripheral lesions and at the venous anastomosis.

Selection of the appropriately sized balloon is important to successful angioplasty. This is dependent upon the knowledge surrounding the anatomical details of the graft or fistula and the size and location of the stenosis.

To adequately dilate a stenosis at the venous anastomosis when most grafts are constructed with 6 or 7 mm polytetrafluoroethylene material, a balloon diameter of at least 7 or 8 mm is required. Figure 16-1 illustrates a successful angioplasty at the venous anastomosis of an arteriovenous graft.

The most consistent success occurs when 7–8 mm balloons are used on peripheral lesions and 12 or 14 mm balloons for angioplasty of central vein lesions. For arterial lesions 4, 5, or 6 mm balloons are adequate to dilate arterial anastomotic lesions. Figure 16-2 illustrates angioplasty of a stenosis at the arterial anastomosis.

For intragraft lesions (Fig. 16-3), a 7 or 8 mm balloon is all that is necessary.

Procedure

1. First, an appropriately sized sheath should be placed. To continue from the diagnostic fistulogram, the 4 or 5 French cannula will accept a 0.035 inch guidewire (e.g., Benston). The wire is negotiated through the anticipated location of the venous anastomosis and, if possible, into the native vein.
2. Remove the previously inserted cannula and place a 6 or 7 French short sheath (<6 cm in length). A guide catheter such as a Kumpe or MPA is often required to advance the guidewire across the venous anastomosis.

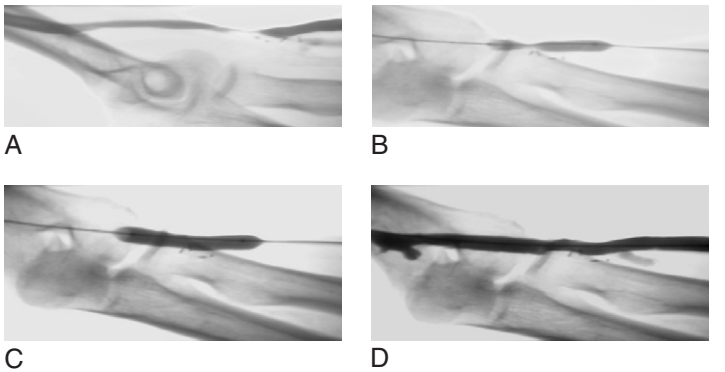


Fig. 16-1 Angioplasty of a stenosis at the venous anastomosis of an arteriovenous graft. **A,** The initial angiogram demonstrates a tight stenosis at the venous anastomosis. **B,** A 7 mm balloon in place, which successfully dilates the area, as shown in **C**. **D,** The final film demonstrating a satisfactory result of the angioplasty.

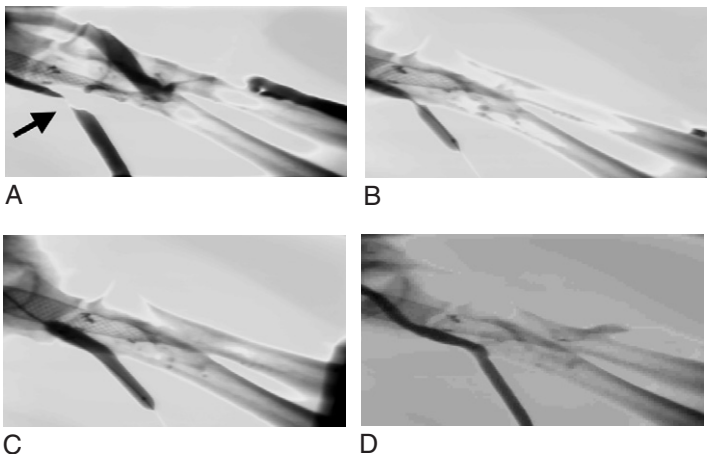
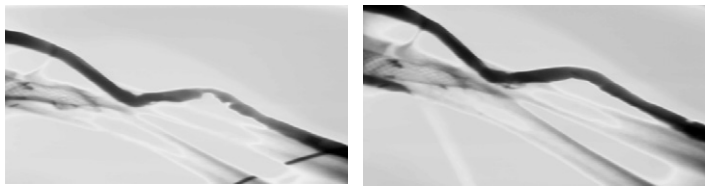


Fig. 16-2 An example of a stenosis at the arterial anastomosis. **A,** The arrow indicates an extreme narrowing at the arterial inlet into the arteriovenous graft. **B, C,** The angioplasty balloon in place. **D,** The final film showing resolution of the stenosis. This resulted in a marked improvement in access flow.



A B

Fig. 16-3 An example of (**A**) intragraft stenosis and (**B**) its satisfactory resolution by angioplasty.

3. Advance the balloon over a guidewire and direct it to the lesion to be dilated. Inflate the balloon to 8–10 atm or to the appropriate pressure to fully dilate the lesion.
4. Remove the balloon and leave the guidewire in place across the previously dilated lesion. Inject a small amount of contrast into the area, evaluate the flow with the graft or fistula after the angioplasty and verify that no complication of graft or vessel rupture has occurred, with extravasation of contrast. If no complication has occurred, remove the guidewire and perform a completion angiogram to confirm a widely patent access with rapid flow and an assessment of the residual stenosis remaining after the dilation.
5. Remove the sheath and achieve hemostasis either by pressure or after placing a purse string suture with 3.0 Ethilon.

ENDOVASCULAR STENTS IN DIALYSIS ACCESS SALVAGE

Vascular stents have been shown to be of value for treating stenoses in large or central veins such as the subclavian, brachiocephalic, and superior vena cava. The use of vascular stents in the peripheral veins remains controversial.

The introduction of indwelling catheters into central veins may lead to the development of stenosis. This response has been observed with pacemaker wires as well as with central venous catheters used for hemodialysis access. Problems caused by central vein stenosis are primarily related to the presence of ipsilateral dialysis access that drains into the affected central veins. The patient frequently develops an edematous access arm. The prevention of central venous

stenosis rests upon the avoidance of central vein catheters, especially subclavian dialysis catheters.

Incidence

The incidence of central vein stenosis varies with the location of the catheter placement in a central vein. With catheters placed in the subclavian vein the incidence is 50%; with catheters placed in the right internal jugular vein it is approximately 10%. Direct injury to the vessel wall, the curvature of the vessel, infection, and chronic inflammation are all thought to play a role in the development of the stenosis.

Treatment

Percutaneous intervention with angioplasty is the treatment of choice for central venous stenosis. However, the success rate and duration of patency with central lesions is suboptimal since many lesions are elastic in nature and the stenosis may recur within 3 months. Although there have been no prospective controlled studies, stenting of central lesions is a common procedure, especially for a lesion thought to be elastic or if the lesion recurs within 3 months. Figure 16-4 illustrates angioplasty and stenting of a severe stenosis of the brachiocephalic vein.

To date, studies have shown that stenting of stenoses located at the venous anastomosis or in native peripheral veins provides little, if any, additional benefit when compared to

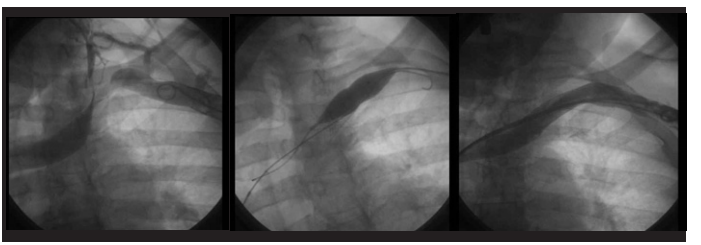


Fig. 16-4 A severe stenosis of the brachiocephalic vein resulting in severe arm swelling and inadequate dialysis. This lesion was treated by angioplasty but recoiled immediately and therefore, as shown in the middle panel, a 14 mm stent was deployed. Further angioplasty, resulted in satisfactory dilatation of the lesion. The arm swelling resolved and has not recurred after 6 months.

angioplasty alone. There are, however, ongoing studies currently re-evaluating this issue.

Stenting

There are several specific situations in which a self-expanding vascular stent may be of benefit for treating peripheral venous stenosis. If the angioplasty is successful and dilates the lesion but it has elastic recoil, causing it to restenose, then a self expanding vascular stent can be inserted to oppose the recoil. In addition, the use of a cutting balloon with its microtome blades to cut or score the lesion that recoils or to cut and then dilate the lesion that does not fully dilate with a standard balloon, followed by placement of a self-expanding endovascular stent, may be of value for resistant lesions and could delay the need for surgical revision of the access. One of the limiting factors here is the limited size of the currently available cutting balloon, which has a maximal diameter of 4 mm. Studies are currently under way evaluating the use of larger-diameter cutting balloons.

The use of the stent in these situations may improve the longevity of the access and does not preclude the possibility of a surgical revision in the future. Whether the use of drug-coated stents will be effective in the setting of hemodialysis vascular access is unknown.

TREATMENT OF THROMBOSED ACCESS SITES

Once a dialysis access site has failed and progressed to thrombosis, salvage with previously discussed percutaneous endovascular methods incorporating both angiography and balloon angioplasty is employed. In addition, thrombectomy of the graft or fistula can be achieved with a variety of mechanical or pharmaceutical techniques, or a combination of these modalities. Most interventionalists use mechanical thrombectomy techniques for clotted arteriovenous grafts because they allow for rapid resolution of occluded access without the expense of thrombolytic agents. Some interventionalist use the mechanical techniques described below but also inject 10 units of tissue plasminogen activator into the clot 30–60 min before attempting the thrombectomy to assist in lysing the clot and preventing embolization. Below we will describe the commonly used technique of using a crossed-catheter or sheath for clot maceration.

Procedure

1. **Accessing the graft.** Establish two points where you would enter the graft. This will depend upon its length and configuration. A loop graft can usually be accessed along both the arterial and venous limbs 5–10 cm from the respective anastomoses. A straight graft requires the insertion sites to have 5–10 cm of separation. Raise a wheal with a small amount of 1% lidocaine at the proposed entry site. (First, access the arterial limb of the graft with the needle pointing retrograde towards the venous anastomosis.) Grasp the graft along the arterial limb of the graft between the thumb and third finger. Advance an 18 gauge needle at 45° until you feel it "pop" through the graft wall. The patient should have little discomfort or pain. If the patient has a lot of pain the needle is outside the graft wall and should be withdrawn. Intraluminal position is confirmed by the easy passage of a guidewire. Advance the guidewire, under fluoroscopy, until it curves within the graft or crosses the venous anastomosis. Remove the needle and over the wire place a 7 French short sheath (a 7F sheath is used in order to accommodate the Microvena thrombectomy catheter whose use is described later, as well as to allow angioplasty).
2. With a 4 or 5 French guide catheter, a 0.035 or 0.038 inch guidewire is negotiated through the venous anastomosis into the native vein (often there is a tight stenosis at the venous anastomosis). Care must be taken to keep the course of the guidewire and catheter within the lumen of the vessel.
3. Perform a venogram of the central venous system to determine if there is a central vein stenosis. Perform contrast injection in the remainder of the native vein and at the venous anastomosis to look for the limits of the thrombus and for a mechanical stenosis. Extreme caution must be used here to avoid retrograde movement of thrombus into the arterial system.
4. If a mechanical stenosis is found at the venous anastomosis or along the venous outflow tract, use an angioplasty balloon as described previously to dilate the lesion. If we are unable to resolve the stenosis within the venous outflow, we abort the procedure, as re-established flow within the graft will not be maintained for an extended period of time.

5. If the stenosis is improved significantly we proceed with the mechanical thrombectomy. Through the previously placed sheath, our preferred method is to use a Microvena thrombectomy catheter (which creates a vortex of swirling saline to macerate the thrombus), which can then be aspirated through the sheath. A less expensive alternative procedure begins by placing a balloon catheter through the sheath (either the previously used angioplasty balloon or a 4 or 5 French Fogarty embolectomy catheter) into the arterial limb and out to the venous limb of the graft. The balloon is then inflated and thrombus can be drawn towards the sheath and aspirated. Thrombus can be macerated with the balloon into small fragments that do not obstruct flow.
6. Once the venous limb is cleared of thrombus a second 7 French sheath is placed into the venous limb, pointing toward the arterial anastomosis, in order to declot the arterial limb. The Microvena thrombectomy catheter is then passed to macerate the clot in the arterial limb of the graft. Then, advance a Fogarty embolectomy catheter into the venous limb retrogradely through the arterial anastomosis. Inflate the balloon and pull it back across the arterial anastomosis. This dislodges the fibrin plug, re-establishing flow in the graft. After final angiograms are taken, the sheaths are removed and hemostasis is achieved by placing a purse-string suture of 3.0 Ethilon.

COMPLICATIONS OF ENDOVASCULAR PROCEDURES

The most common complication of procedures performed to repair or maintain a failing hemodialysis access is bleeding and hematoma from the access site at the point of entry of the diagnostic and therapeutic sheaths. Manual pressure and a purse-string suture usually prevent expansion of the problem.

Rupture of the vein and or graft, with extravasation of blood, is the most common complication of angioplasty. A rupture usually occurs acutely and is recognized at the time of the angioplasty. If a rupture should occur the site should be manually compressed to achieve hemostasis, or inflate a blood pressure cuff above the arterial anastomosis. Also, vascular stents have been deployed to tamponade the rupture and maintain the vessel, or the angioplasty balloon may be

reinflated across the site to occlude the vessel and prevent further blood loss until a repair is made.

Distal embolization of thrombus toward the hand or toes from the arterial anastomosis following thrombectomy has been described. Salvage with an infusion of intra-arterial thrombolytics alone and in combination with vasodilators such as nitroglycerin have been successfully described.

Worrisome, but of unknown clinical significance, is the creation of small, multiple pulmonary emboli following thrombectomy of dialysis access. Pulmonary emboli have been documented following successful thrombectomy (in up to 59% of patients in one series when looked for following the procedure; however, the overwhelming majority of the patients did not have any clinical symptoms). The amount and size of the clot dislodged is thought to be very small and of no consequence.

Suggested Readings

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Page numbers in *italics* indicate the pages on which figures/tables/boxes appear. All entries relate to PCI unless otherwise indicated.

Abbreviations:

PCI, percutaneous coronary intervention
PTCA, percutaneous transluminal coronary angioplasty
PTMC, percutaneous mitral commissurotomy
PTSMA, percutaneous transluminal septal myocardial ablation

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