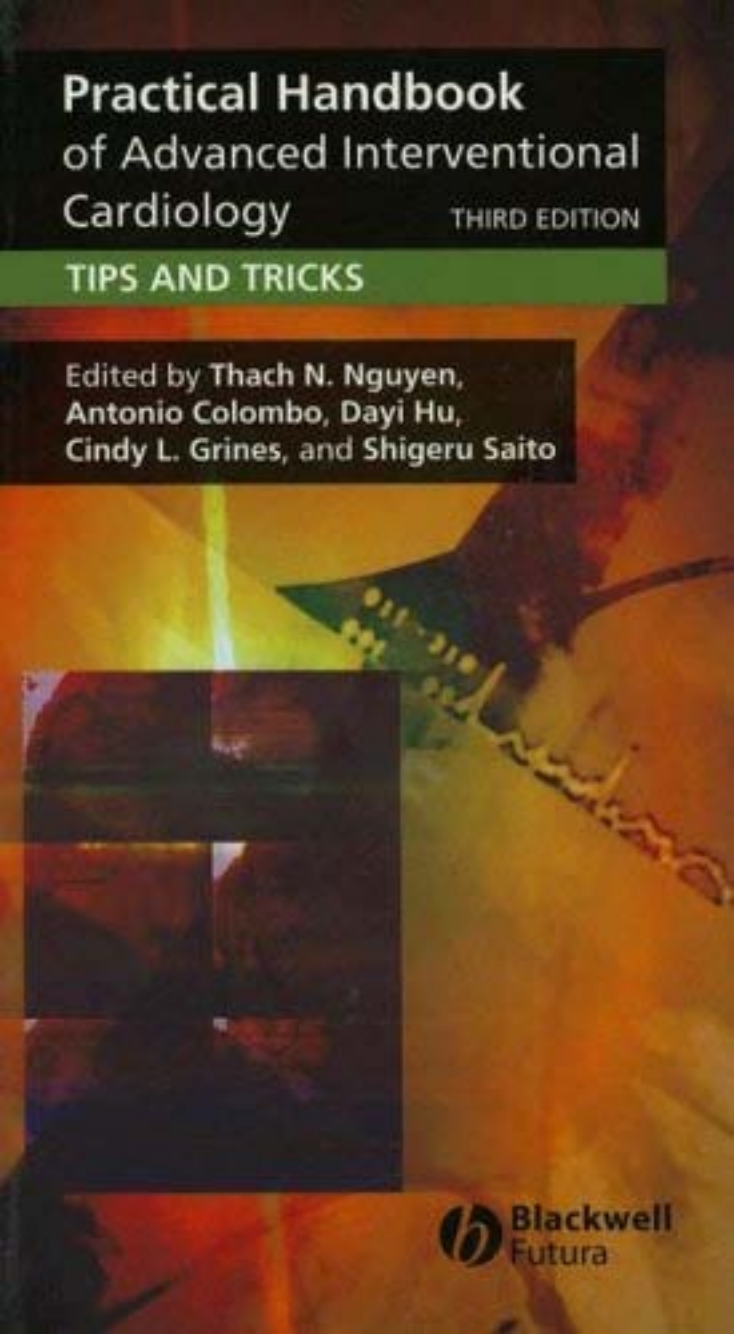
Practical Handbook of Advanced Interventional Cardiology

THIRD EDITION

TIPS AND TRICKS

Edited by Thach N. Nguyen, Antonio Colombo, Dayi Hu, Cindy L. Grines, and Shigeru Saito



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Practical Handbook of **Advanced Interventional Cardiology**

TIPS AND TRICKS

THIRD EDITION

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Foreword to the Second Edition

Interventional cardiovascular medicine has evolved from an extremely crude method of opening femoral arteries initiated by Dotter, to a field that has now been recognized as having a sufficient fund of knowledge to require boards sanctioned by the American Board of Internal Medicine. From Andreas Gruentzig's development of the noncompliant balloon method, we have seen an explosion of bioengineering technology. The discipline of interventional cardiovascular medicine has perhaps initiated more registries and clinical trials than any other discipline in medicine. Indeed, the whole emphasis on evidence-based medicine has evolved during the era of interventional cardiology. Many basic science breakthroughs have been stimulated by the advances produced in interventional cardiology, as well as the problems and complications created by the new technologies.

However, no matter how advanced the science becomes, the success of solving a patient's problem with interventional techniques usually depends on the operator's technical ability. This ability springs from the wealth of experience the operator has acquired to deal with routine situations as well as complex and almost unique problems that may present themselves. Because of the large number of interventional cardiologists and the rapidly expanding number of procedures that can be performed, it is difficult for many cardiologists to experience all of the situations that can be helpful in building this database.

Dr Thach N. Nguyen has prepared a remarkable book, rich with tips and tricks for performing interventional cardiovascular medicine procedures. He has enlisted numerous experts on various aspects of interventional cardiovascular medicine to describe their areas of expertise. Rather than let them recite the evidence from registries and trials that are available elsewhere, he forces the contributors to provide the practical tips that they have learned. It is almost as though Dr Nguyen is trying to simulate the type of scenarios that exist in the catheterization laboratories with new cardiology fellows or less experienced operators. It is the type of advice that he has often given to cardiologists in developing countries who are bringing interventional techniques to help cope with the rapidly expanding new threat in these countries, vascular disease. Since new techniques are constantly appearing, all operators, experienced or not, can benefit from these tips. Whereas every operator will not agree with every approach to a problem or a complication, it is always instructive to understand many potential approaches. In this regard, the book does a masterful job of collecting not only the authors' experiences, but those of many others collected from the published literature, from numerous postgraduate courses, and from one-on-one demonstrations throughout the world.

This book should be a valuable resource to trainees in formal programs that have now evolved in the United States and other countries, as well as the many preceptorships that are the major means of training in other countries. In addition, operators of all levels of experience will find many useful pearls of wisdom. Dr Nguyen and his colleagues are to be congratulated for compiling this most practical guide.

Spencer B. King III, MD
Atlanta, Georgia

Preface

ADVANCED INTERVENTIONAL CARDIOLOGY ART AND SCIENCE

In 2007, more than 25 years after its humble beginning, interventional cardiology is a mature and major player in the management of complex cardiovascular problems. Thanks to modern micro-technology and nano-engineering, with miniaturized equipment, the techniques of interventional cardiology have also made giant leaps forward to become ever-more effective and user-friendly. These techniques can be formulated as a sequence of rigorously controlled maneuvers which can be taught to fellows or staff, or programmed into robots. To understand and explain the physical, chemical, biologic, and engineering mechanism of any of these techniques or maneuvers is a science. To perform a procedure cost- and time-effectively in a humane manner is an art. In any interventional laboratory, a lesion could be dilated with one guide, wire, balloon or stent by a senior operator or by x (> 1) numbers of devices by a beginner. This is the result of mixing science and art.

Which is the Best Option to Apply to this Real-Life Situation? During a procedure, each operator (*or any commander on the battlefield*) has the luxury (*and the responsibility*) to select, to change, delete or modify direction, position of a device (*a tank*), a drug (*a battalion*), a strategy (*conventional, guerilla or urban house-to-house combat*); or to be forced to use one when the others are not available. These options are listed plentifully and discussed ad nauseam everywhere in the print or electronic media. However, the main question always remains: which one is the best option to apply to this real-world situation (*jungle or urban*), with the equipment (*arms and troop*) available in this particular cardiac interventional laboratory (*or in Iraq*)? In this third edition of this handbook, the authors try to answer this question and give practical suggestions derived from their own daily labor in the cardiac interventional laboratories.

In the "Strategic Mapping" boxes, the operator (*as commander*) draws in his or her mind a global schema of procedural sequences he or she would execute in order to achieve success (*victory*). This strategic map also includes preventive or corrective measures in case of crisis situations such as complications or suboptimal performance of any tactics or strategy.

In the "Tactical Move" boxes, the authors break up the whole strategy into detailed procedural sequences with limited local goals (i.e., tactics). At the beginning, it is how to select an appropriate device, e.g. guides (best choice among many), so it can achieve its goal at a

first attempt. Then, if a device does not function as expected, there are many simple maneuvers (best maneuvers chosen from many) to correct or reverse the situation. In any case, the operators try to exhaust the full potential of any device first without prematurely and wastefully discarding it. However, at the same time, while there are many parallel competing tactics or strategies, which one is objectively best in which situation? This is the role of critical thinking, because a subjective change of tactics could save the whole procedure and lead either to success or to our worst nightmare. In this section, each maneuver is graded according to time spent, cost of extra equipment and the level of risk of complication. For every extra 10 minutes, it is one more hour on the clock. One dollar sign means that $100.00 US extra are spent. One drop of blood is the symbol for moderate complication; for two drops, there is high risk.

In the "Caveat" boxes, we warn the readers of any treacherous images, deceiving roadside sign posts or wrong moves that harbinger impending disaster. This information combines past personal failures, near death experience of the patient (*or of the operators*) and successful (*almost miraculous*) resolution of the critical events. Altogether they constitute a collective memory of how to avoid failure and how to achieve success: this is called experience. If the hard earned lessons of that collective memory are applied in real life, the rate of procedural success would be higher and the incidence of complications would be lower. The rate of complications depends on the operator's skill, the technology available, and patient selection. Rigorous preventive measures learned from that collective memory (i.e., experience) preempt the appearance of complications (*one of the best ways to avoid EVERY complication is to perform NO procedures*). With the use of current low-profile balloons and high torqueable wires, most patients with "simple" stenoses will have good results, even in the hands of relatively inexperienced operators. However, in patients with complex anatomy or when simple cases become complicated, experienced operators are likely to have superior outcomes. This is what experience is for [1].

To the readers who are all friends and colleagues: The authors and editors, who are all your friends and colleagues, labor every day in the cardiac interventional laboratories, like yourself. We write from our limited subjective experiences and our hearts, during many sleepless nights. This handbook contains practical advice aimed at you the readers, and at us, the authors ourselves. We practice what we preach. They are not from an ivory tower: they are practiced by those with experience and beginners, by the young and old, by men and women, by serious operators and by the urban week-end warriors, so there are no questions of class, age, sex, or race division here. In this book, we try to highlight these practical suggestions with all of the dramatic colorings or ups and downs – reminiscent of an Italian opera – which happen daily in every interventional laboratory

across the globe. However, we hope the outcome of these procedures is always happy and beautiful as the end of any Chinese martial arts movie.

The bottom line is that we practice interventional cardiology in a responsible manner: cost- and time-effective without causing more harm (*prima non nocere*). All of us are equal in this quest of striving for the best procedural and clinical success. This is the only goal of this handbook.

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Above all, we are indebted to our patients – the purpose of our care, the source of our quests, the inspiration of our daily work. To them we give our heartfelt thanks.

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Chapter 1
Vascular Access

Thach N. Nguyen, Hoang Pham, Ta Tien Phuoc

Femoral Approach
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- **Technical tips**
- *Directing the needle
- *If the wire cannot be inserted
- *Sequential order for arterial and venous puncture
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Closure Devices
- Preclosure of large arterial access
- Preclosure of large venous access
- **Technical tips**
- ***Preclosure of the venous access with the perclose
- **Caveat:** Suspecting intra-arterial deployment of collagen plug

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ < 100.00 $US extra; **$$** > 100.00 $US extra
⌛ < 10 minutes extra; ⌛⌛ > 10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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FEMORAL APPROACH

Locate the femoral artery and inguinal ligament that runs from the anterior superior iliac spine to the pubic tubercle. The true position of the inguinal ligament is 1–2 cm below that line [1]. Ninety-seven percent of patients have the femoral artery lying on the medial third of the femoral head. Only 3% have the artery totally medial to the femoral head [2].

Preparations in Obese Patients: The femoral pulse at the inguinal crease is not a reliable landmark for the common femoral artery (CFA), particularly in the obese or elderly patients whose crease tends to be much lower than the inguinal ligament. The protruding abdomen and panniculus should be retracted, and taped to the chest with 3- to 4-inch tapes that are in turn secured to the sides of the catheterization table. Keep the tissue layer above the artery as thin and taut as possible, so the needle will not be deflected from the projected angle and selected pathway.

TECHNICAL TIPS

***Directing the Needle:** Once the needle tip is near the artery, it tends to pulsate except in those patients with severe local scarring (following many prior remote femoral artery cannulations, total hip replacement, or in severely calcified arteries etc.). If the hub inclines to the right, the needle should be withdrawn by 1 or 2 cm and the tip redirected to the right before advancing forward. If the hub inclines to the

left side, the tip is redirected to the left before pushing in. If the needle pulsates on the vertical axis, it just needs to be inserted deeper [2].

***If the Wire Cannot be Inserted:** Most often, this is because the needle hit the contralateral wall. Just move the needle by a slight pull or rotate it a little, the wire may be able to be inserted. If there is a problem, it is better to withdraw the needle and re-puncture the artery rather than dissect the artery with a slippery wire [3]. After the sheath has been inserted where there is strong arterial back flow and the wire is not able to negotiate the tortuous iliac artery, then pull the sheath a little (to disengage it from under a plaque if that is what has happened) and a gentle injection of contrast may help to delineate the anatomy and determine the reason why the wire could not be advanced. If there is no strong back flow, then the sheath is not in the arterial lumen. In a very tortuous iliac artery, a diagnostic Judkins Right (JR) catheter can be inserted with caution and advanced in order to help steer the wire tip. Injection through the JR would also help to find out why there is a problem advancing the wire.

***Sequential Order for Arterial and Venous Puncture:** The order of arterial and venous access is often a matter of personal preference. We prefer to puncture the vein first and insert a wire inside the vein to secure the access. Then, less than a few seconds later, after puncturing the artery, we would insert the sheath into the artery and the vein. Because there is only a wire in the vein, there is minimal distortion of the arterial puncture site, which may be caused by the placement of the venous sheath. Less than 1 minute without a sheath will not produce a hematoma at the venous site. If inadvertently the artery is punctured first, we would cannulate the artery, then puncture the vein under fluoroscopy, with the needle medial and parallel to the arterial sheath.

****Kinked Wire:** It is not unusual that the wire will pass into the lumen easily but attempts to advance any dilator over the wire will result in kinking of the wire at the point of entry. Instead of exchanging the wire, if the wire is not too crooked, the first best maneuver is to advance the wire further, so the dilator can be advanced to dilate the entry site on a straight and stiff segment of the wire. If the wire is too soft, then the second best maneuver is to exchange the softer wire with a stiffer wire over the needle at the straight portion or over a smallest size 4F dilator [4].

****Puncture of Pulseless Femoral Artery:** As usual, the artery should be punctured over the middle of the medial third of the femoral head. Localize the skin puncture site by fluoroscopy just below the inferior border of the femoral head in order to prevent high punctures (above the inferior epigastric artery) that may lead to uncontrollable bleeding. However, these proportions are valid only in the anterior-posterior (AP), neutral position. Internal or external rotation of the femur can considerably change the relationship of the femoral artery

to the femoral head [5]. Another way to puncture the femoral artery is to use Doppler guidance with the SmartNeedle (Escalon Medical, New Berlin, WI), which is an arteriotomy needle that incorporates a continuous Doppler probe, and enable the identification of arterial or venous vessels by means of continuous auditory feedback. This technique is very helpful in puncturing an artery with very weak pulse or a pulseless artery, especially when the standard anatomy is disturbed by a large hematoma, or thick scar after surgery for artificial femoral head replacement [6].

*****Puncture in Cyanotic Patients:** In children with cyanotic heart disease, especially those weighing less than 15 kg and with severe polycythemia, the blood flow from a femoral puncture can resemble a venous sample: a gentle flow of dark blood. This color is due to arterial desaturation and hyperviscosity secondary to polycythemia. If there is doubt, confirm the arterial puncture by attaching the needle to a pressure transducer or by making a small contrast injection into the arterial lumen [7].

TROUBLE-SHOOTING TRICKS

*****Insertion of Intra-Aortic Balloon Pump (IABP) Through Diseased Iliac Artery:** When an IABP needs to be inserted and an iliac lesion is found, the lesion should be dilated first. Insert the balloon pump, then perform stenting of the lesion later after the IABP is removed. When a balloon pump is to be inserted through a previously stented iliac artery, do it under fluoroscopy to be sure the balloon does not get stuck on the stent struts. To remove the IABP deflated balloon, insert a large femoral sheath and withdraw the winged balloon into the sheath so the folds of the winged balloon are not caught by struts at the stent edges. Chronic endothelialization of the stent struts should diminish this problem.

*****Two Catheters Inserted With One Puncture Technique:** Used in situations such as angioplasty for chronic total occlusion (CTO) when there is a need for contralateral injection. Another puncture higher or lower than the puncture site of the first site of vascular access, or in the contralateral artery, is suggested. However, if there is no need for another puncture, then change the sheath to an 8F introducer. There two 4-Fr diagnostic catheters can be inserted and attached to separate injection manifolds for diagnostic purposes [8].

Puncture of Femoral Bypass Graft: The problems involving puncture of an old vascular graft in the femoral area include: uncontrollable bleeding and hematoma formation because of the nonvascular nature of the punctured graft; disruption of the anastomotic suture line with subsequent false aneurysm formation; infection of the graft site; and catheter damage, kinking, and separation due to scar tissue in the inguinal area and firmness of the healed graft material [4].

Inadvertent entry to the native arterial system may lead to the dead-end stump in the common femoral or iliac artery.

TECHNIQUE Bypass Graft Puncture: Because the exact location of the suture line is not known, to avoid puncture of the anastomotic site, it is best to puncture the proximal end of the inguinal incision site or as close to the inguinal ligament as possible. To avoid kinking of the catheter at the puncture site, it is better to introduce the needle at an angle of approximately 30–45° to the estimated long axis of the graft [4]. Sometimes, because of severe scarring, the entry site has to be prepped by sequential dilation with small to progressively larger dilators **up to 1Fr** size larger than the sheath selected for the procedure.

TROUBLE SHOOTING TRICKS

*****Parallel Technique:** If the native artery is punctured and the wire could not be advanced because the artery ends up with a dead-end pouch, then leave the small 4F sheath inside as a landmark. Palpate again the femoral artery and try to feel the two pulsations there: the first one is the native artery with the sheath and the second stronger and harder one is the bypass graft. Then puncture the second pulsatile artery while avoiding the one with the sheath in it. This can be done under fluoroscope guidance to avoid any area near the first sheath.

ANTEGRADE PUNCTURE

The antegrade femoral puncture can be greatly simplified and is more successful if the tissue thickness between the skin surface and the artery is as thin as possible. This may be made possible by placing a pillow under the buttocks. The hyperextension of the hip joint by this maneuver stretches the skin taut over the puncture site and decreases tremendously the tissue thickness. In obese patients, fatty panniculus may have to be retracted away from the puncture site manually and taped in position before the puncture is attempted [5].

TECHNIQUE Antegrade Puncture: The first step is to localize the CFA and its bifurcation under fluoroscopy. The CFA usually overlies the medial third of the femoral head and the bifurcation occurs below the lower border of the femoral head [3]. Once the landmark is located, to make the puncture, the needle may be directed toward the superior aspect of the femoral head, under fluoroscopy. The purpose of this maneuver is to prevent the inadvertent puncture of either or both the superficial femoral or the profunda femoral arteries. It is important to puncture the femoral artery as high above the bifurcation as possible so that there will be enough space between the puncture site and the bifurcation for catheter exchanges and manipulation of catheters into the SFA.

Using fluoroscopy, the site of the intended arterial puncture is identified (upper or middle third of the femoral head). The femoral pulse

is palpated against the femoral head. Local anesthetic is infiltrated 2–3 cm cranial to the intended site of puncture. A 18 gauge needle is advanced at 45–60° directed caudally, aiming at the intended site of arterial puncture. Once pulsatile flow is obtained, a soft tip wire is inserted toward the SFA. The wire should follow a straight caudal course in to the SFA. Lateral deviation indicated entry into the profunda femoral artery (PFA). The wire can be withdrawn and the needle tip deflected laterally to redirect the wire into the SFA.

TECHNICAL TIPS

****Manipulation of Wire:** If the wire was inserted into the PFA, it can be withdrawn and redirected by angling the tip of the needle medially toward the SFA. The other option is to have a wire with a curved tip and manipulate it so the tip points toward the SFA. The needle may be exchanged for a short dilator with a gently curved tip, which can be directed toward the SFA. This dilator can be withdrawn slowly from the PFA while injecting the contrast agent. Once the orifice of the SFA is seen under fluoroscopy, it can be selectively catheterized or it can be used to direct a wire into the SFA [5].

****Puncture of CFA with High Bifurcation:** In patients with high bifurcation, one single puncture can result in entries of both the SFA and PFA. When this occurs, the first spurt of blood may indicate that the PFA is punctured. Do not remove the needle completely. Instead, withdraw it slowly and watch for a second spurt of blood. At this point, the contrast injection may show that the needle is in the SFA. In the rare cases of high bifurcation, it may not be possible to puncture the CFA that is excessively high in the pelvic area [5]. When the bifurcation is located more proximally, puncture of the CFA is more challenging, especially in the obese. In these cases, it may be acceptable to selectively puncture and cannulate the SFA, if this appears without significant atherosclerotic disease and of adequate size [9].

****Puncture with Abduction and External Rotation of the Thigh:** Another option to cannulate the SFA is with the thigh in abduction and external rotation. The goal of this maneuver is to facilitate a more mediolateral puncture site in the CFA. In the usual antegrade puncture, the needle is seen to point more toward the PFA that is lateral to the SFA. In the abduction and external rotation position, the needle points more toward the SFA, and the PFA is seen medial to the SFA. This relationship is important when observing the course of the wire during its intended selective entry into the SFA. If the patient is punctured in this position, after the procedure, the local compression of the artery should be in the abduction and external rotation of the thigh because the puncture site is more mediolateral than usual [5].

BRACHIAL APPROACH

Even though the radial artery is the most common location used in the upper extremity, the brachial artery is still the access site of choice for procedures requiring a large sheath: subclavian artery stenting, renal stenting, or aortic aneurysm exclusion. The radial access is discussed in the following chapter.

TRANSLUMBAR APPROACH

In patients with total occlusion of arteries to lower and upper extremities, percutaneous coronary interventions (PCI) can still be performed through the translumbar approach [10]. This problem occurs rarely, only once in 6000–9000 cases. However, if the lumbar approach is the only access available in those rare circumstances in which conventional sites are not available, then it is worth offering the option to the patient [11].

TECHNIQUE Translumbar Puncture: The patient is placed in the prone position. Utilizing the left flank approach, an appropriate puncture site is selected, which is approximately four fingerbreadths lateral to the midline and two fingerbreadths below the left 12th rib margin. Verification that this position is below the posterior sulcus of the lung is made by fluoroscopy. After local anesthesia, a small skin incision is made with a blade and enlarged by the hemostat. The tip of the translumbar access needle (TLA) and the outer Teflon sheath (Cook, Bloomington, IN) are placed in the skin incision and directed toward the T12 vertebral body. Three successive attempts are made, with each increasing the vertical degree of the pass in order to "step off" the vertebral body. When the needle tip abuts the aorta, pulsation can be felt against the fingertip. The TLA needle is then given a short thrust until the initial resistance is not felt. The tip of needle is watched closely and should never cross the midline of the body. The inner stylet of the TLA needle is removed, and blood is seen at the hub. A floppy J wire is inserted and an introducer sheath is inserted in the usual fashion. Coronary angiogram and angioplasty are performed by the standard technique. After documentation of active clotting time (ACT) less than 150 sec, the sheath is removed without complication while the patient is in the prone position. Given the prone position of the patient, the fluoro scopic images appear in reverse, compared to standard images. This problem can be corrected by using the sweep reversal mode on the video monitor [10].

TRANSEPTAL APPROACH

Femoral and radial access is universally used for interventional procedures. However, in some patients with pulseless disease (Takayasu's arteritis), there are no arterial pulses in four extremities, then the PCI has to be done through the femoral vein approach. Tips and tricks for puncturing the septum are discussed and illustrated extensively in Chapter 24.

CAVEAT

Maintaining a Long Loop in the Ventricle: At all times, care must be taken to maintain a loop of catheter or wire in the left ventricular apex. Shortening of this loop to the straight path between the mitral and aortic valves could result in trauma to the anterior mitral leaflet causing acute mitral regurgitation [12]. There are few problems cannulating the left main (LM); however, it is difficult to cannulate the right coronary artery (RCA) because the catheters keep dropping into the ventricle when manipulated [13].

TRANSTHORACIC LEFT VENTRICULAR PUNCTURE

TECHNIQUE Transthoracic Apical Puncture: In order to perform left ventricular puncture, the left ventricular apex is identified by palpation or echocardiography. The skin and the intercostal space area below the apex is infiltrated with 1% xylocaine. Transthoracic puncture is performed, entering the chest wall in the intercostal space below the site of maximal impulse using a 5.5Fr × 20.5 cm One-Stem fluid drainage assembly (Electro-Catheter, Rahway, NJ). This assembly is composed of a trocar, a needle, and a pigtail catheter. The assembly is introduced percutaneously through the chest wall and directed posterior aiming to the right shoulder. Upon contact with the left ventricle, the assembly is advanced rapidly in a single motion aiming from the apex to the mitral plane. The needle and trocar are removed, leaving the pigtail catheter in the left ventricular cavity. The incidence of major complications of direct left ventricular puncture includes ventricular fibrillation (0.2%), tamponade (1.4%), pneumo- or hemothorax (2.7%), stroke or transient ischemic attack (0.3%), vasovagal reaction (1.0%), unsuccessful puncture (0.9%), and death (0.5%). In patients with multiple previous cardiac operations, this complication rate may be even lower as the pericardium becomes thickened and adhesive, making tamponade less likely to occur [14].

CLOSURE DEVICES

Closure device can be used after any procedure such PCI, valvuloplasty, IABP or due to inadvertent arterial puncture such as after cannulation of a subclavian artery instead of a jugular vein. The choice between collagen plugs and suture closure is largely a matter of personal preference and experience. The time needed to deploy the various devices is unique to each system. When physicians' time to utilize the device and staff time for adjunctive compression or puncture site management are considered together, sealing devices do not provide an advantage over manual pressure in decreasing complications [15]. Thorough training of operators in how to use any device is warranted to reduce vascular access complications. When deploying an AngioSeal device (St Jude

Medical Devices, Minneapolis MN), an iliac angiogram needs to show the artery diameter is at least 4 mm and there is no bifurcation within 2 cm of the arterial entry site.

Preclosure of Large Arterial Access: In cases where a large sized sheath is needed (e.g. for aortic valvuloplasty), preplacement of untied sutures using the Perclose percutaneous suture delivery system (Abbotts Vascular, Redwood City CA) prior to placement of a large intended sheath can be done. A 5Fr to 6Fr sheath may be used for arterial angiography to identify appropriate anatomy for suture delivery, and then a suture device is used to place untied sutures. At the end of the procedure, the existing "purse string" is then closed around the arteriotomy [16].

Preclosure of Large Venous Access: The technique of "preclosure" involves preloading a 6Fr Perclose suture closure device into the femoral vein after access with a 6Fr or 8Fr dilator, prior to insertion of a 14Fr venous introducer sheath used for antegrade aortic valvuloplasty. Intravenous placement of the Perclose device within the venous system is then verified by either back-bleeding from the marker port, or contrast injection through the marker port. Then the needles are pulled and the sutures clipped, and after the sutures are deployed, a wire is placed into the femoral vein through the Perclose device, and an exchange is made over the wire for a 14Fr sheath while the sutures are laid alongside of the puncture and covered with betadine-soaked gauze. Upon completion of the valvuloplasty procedure, a wire is passed through the 14Fr sheath to secure the vessel in case the suture closure fails. Heparin is not reversed. The sheath is then removed through the existing sutures, and the sutures are tied around the wire. If hemostasis is successfully achieved with the suture, the wire is gently removed, and the knot pushed further to complete the closure.

TECHNICAL TIPS

***Preclosure of the Venous Access with the Perclose:** Since veins are comparatively thin-walled, the amount of tension applied when pulling back the Perclose device is necessarily LESS than for arterial closure. It is possible to securely contact the vessel wall with the foot of the device while applying steady pressure, with LESS force than needed for arterial closure. Back-bleeding through the marker port occurs in the vast majority of cases. Due to the lower pressure in the venous system, this is of course less prominent than in arterial closure. Usually, a slow dribbling of blood from the marker port can be noted. There is a delay in the appearance of back-bleeding due also to the low venous pressure, and this may be accentuated by having the patient take in a deep breath or by employing the Valsalva maneuver [17].

CAVEAT

Suspecting Intra-Arterial Deployment of Collagen Plug: During deployment of an AngioSeal device, intra-arterial deployment of the collagen plug can be due to inadequate tension on the suture, vigorous tamping, too deep insertion of device into the artery then the anchor is caught in the posterior wall, etc. Suspicion of a problem is aroused when there is a long travel distance of the tamper tube or continued bleeding [18].

CASE REPORT Management of Intra-Arterial Deployment of Collagen Plug: In a case reported by Stein *et al.*, a possible intra-arterial deployment of the collagen plug was suspected. At that time, while inserting deeper the tamper tube, it was observed that it could be inserted much deeper than usual. The patient continued to bleed, so a tension spring was placed as usual. At that time, the author used a Hemostat to secure the end of the suture, and a FemoStop compression device (Femostop, Radi Medical Systems AB, Sweden) was applied above the AngioSeal to stop bleeding. Then the author waited for 4 hours, so the anchor that is composed of an absorbable polymer material, becomes softened and so pliable. A Hemostat was placed on the suture at the level of skin. If the suture were to break during traction, the hemostat would prevent the anchor and the collagen plus from embolizing. Then steady traction was applied to the suture, perpendicular to the femoral artery. The pressure should not be excessive. After 20 minutes, the plug was removed. The FemoStop was reapplied and hemostasis was achieved [18]. The management is summarized in Table 1-1.

Table 1-1 What To Do if Collagen is Inserted Intra-arterially [18]

1	Prevent the problem: always maintain tension on the suture and avoid tamping with excessive force
2	Recognize the problem: absence of resistance during tamping and inadequate hemostasis are clues
3	Duplex ultrasound can document intra-arterial collagen
4	Apply tension string in the usual fashion; secure suture with hemostat at the skin level to add security
5	**Do not cut suture:** embolization of the anchor and plug may occur
6	If there are signs of embolism and thrombosis, obtain vascular surgery consultation
7	Wait at least 4 hours to allow softening of the anchor
8	Steady vertical traction on suture with approximately 10 lbs of force
9	If removal of the device is achieved, maintain manual compression to achieve hemostasis
10	Femo-Stop device should be ready for rapid deployment after device is removed
11	Remove the collagen plug by atherectomy device (not needed)

Removal of Intra-arterial Collagen Plug by Atherectomy: One of the complications of using the AngioSeal device is the partial protrusion of the collagen sponge into the artery, likely due to insufficient tension on the suture while the collagen is deployed. It has been difficult to treat this problem with balloon angioplasty alone, because of the highly eccentric nature of the lesion in an otherwise relatively healthy CFA. Suboptimal results of balloon angioplasty in markedly eccentric lesions have been attributed to the compliance and elastic recoil of healthier portions of the arterial wall. Percutaneous approaches to AngioSeal-related complications include angioplasty, thrombectomy with the AngioJet system, extraction with a snare catheter or even with an endomyocardial biopsy device, and angioplasty followed by stenting. Stenting of the CFA should be avoided if at all possible, since it can result in deformation and strut fracture secondary to hip flexion and could compromise future arterial access and surgical repair. The unique design of the SilverHawk atherectomy catheter allows clear visualization of the cutter position during the procedure and enables preferential excision in the direction of the lesion, thus sparing the normal portions of the arterial wall. Although the initial technical success of this device in femoro-popliteal lesions is high, distal embolization may complicate the procedure, particularly in patients with poor distal runoff [17].

COMPLICATIONS

Hematoma: Their frequency is 1–3% and increases with the increasing size of the sheath, increasing level of anticoagulation, and the obesity of the patient [15]. Surgical evacuation is not required even for large hematomas, unless there is undue tension on adjacent structure or in the case of a truly huge hematoma. Surgical evacuation and arterial repair are required when the hematoma is pulsatile and expanding, an indication of communication between the hematoma and the femoral artery and the presence of a false aneurysm [19].

Arteriovenous Fstula (AVF): This happens rarely ($<0.4\%$) when the puncture is made where the artery overlies the vein [20]. Most small AVFs are asymptomatic and usually close spontaneously. A large AVF with symptoms of high output failure needs to be corrected surgically.

Acute Arterial Thrombosis: Occlusion of the femoral artery may occur due to thrombosis or local arterial injury. It happens in 0.1% of patients, mostly in women with small femoral arteries that are completely blocked by the catheter during the procedure and in patients whose superficial femoral artery (SFA) is catheterized rather than the CFA [20].

TECHNIQUE Mechanical Thrombectomy for Acute Thrombosis: If thrombosis of the femoral artery is suspected, access is obtained from the contralateral side and 5000 units of heparin are given. A 6F crossover sheath is placed in the external iliac artery over a 0.035" stiff Amplatz guide-wire. The occluded/thrombosed/embolized segment or the artery is crossed with a 0.014" or 0.018" wire. Any thrombectomy device is then introduced over the wire and tries to

remove any thrombi. If normal distal flow is established without any residual stenosis, the procedure is terminated. It there is still residual thrombus, then the segment is dilated with a peripheral balloon, and if the post-percutaneous transluminal angioplasty (PTA) result is not optimal, a self-expanding stent may be deployed [21].

If heavy thrombotic burden still persists after mechanical thrombectomy, then tPA 0.05 mg/kg can be given along with heparin through a multi-hole delivery catheter (e.g. 5Fr Mewissen of Boston Scientific, Boston MA) 4 hours later, an angiogram can be carried out to check the progress and if there is thrombus, the patient can undergo longer infusion (12–18 hours) [21].

Limb Ischemia: Patients who develop acute limb ischemia following femoral artery catheterization must be carefully and immediately evaluated by duplex ultrasound. Angiography is mandatory and should not be delayed. The purpose of angiography is to identify the location (aorto-iliac inflow circulation, infra-inguinal outflow circulation, or runoff circulation), and cause (dissection, thrombosis, distal embolization, sheath/vessel mismatch) of ischemia, since these factors will help determine the treatment strategy (vascular surgery, percutaneous revascularization, thrombectomy, intra-arterial thrombolytic infusion). In most cases, digital subtraction angiography is best, since cineangiography may not permit adequate visualization of the runoff circulation [22].

TROUBLE-SHOOTING TRICKS

***Temporary Relief of Iatrogenic Ischemic Limb: Percutaneous Technique for In-Vivo Femoral Artery Bypass:** During PTA of high-risk patients, if the acute limb ischemia arises during femoral artery catheterization, the sheath in the right femoral artery and the sheath in the left common femoral artery can be connected using standard 12-inch pressure tubing and a male-to-male adapter. In general, this technique is considered a temporizing method to restore blood flow, minimize the metabolic consequences of acidosis and muscle necrosis, permit more definitive percutaneous or surgical revascularization as indicated, and to allow the use of devices for invasive hemodynamic support when such devices cause limb ischemia and there are no other therapeutic alternatives [22].

CAVEAT

Preventing Limb Ischemia: The steps to prevent limb ischemia include: (1) careful examination of femoral pulses and bruits before catheterization; (2) angiography prior to insertion of any hemodynamic support device; (3) angioplasty and stenting of suitable aorto-iliac stenoses before device insertion; and (4) use of a sheathless IABP might reduce the risk of ischemic complications in patients with diffuse aortoiliac disease or small vessels [22].

Retroperitoneal Hematoma: The clinical clues include hypotension without apparent reason, blood loss without possible source, suprainguinal tenderness and fullness, and flank discomfort. A small hematoma is not able to cause any hemodynamic disturbances or any increase of the retroperitoneal cavity pressure to cause neurological symptoms. Only a huge hematoma compressing the lumbar plexus can really produce numbness and weakness of the muscles below the knee. Usually, bleeding into the retroperitoneal site is self-limiting unless the patient is anticoagulated [19]. The management includes stopping heparin and reversing anticoagulation with protamine, then rapid fluid resuscitation to reverse hypovolemia. Transfusion may be needed. If the above treatment does not reverse the low blood pressure quickly, urgent iliac artery angiography is needed and surgical consultation should be called.

TECHNICAL TIPS

****How to Detect Retroperitoneal Hematoma in a 1 Second Maneuver?** Just an AP view of the pelvic area under fluoroscopy may give a clue to the problem. Usually, the bladder is seen round, filled with contrast. However, if the opacified bladder is seen displaced and its round shape is dented, retroperitoneal hematoma is strongly suspected (Figure 1-1). However, significant blood needs to be sequestered before unilateral external compression occurs [20].

TECHNIQUE How to Seal a Perforation: The initial angiogram revealesd laceration of the inferior epigastric artery arising at the origin of the right CFA. A 6Fr crossover sheath is positioned in the right external iliac artery, and a 6Fr right Judkins-4 guiding catheter is then advanced over the crossover sheath to engage the ostium of the lacerated inferior epigastric artery selectively. A 0.014" Balanced Middleweight guidewire (Guidant, Santa Clara, CA) is advanced into the inferior epigastric artery, and the tip positioned distal to the lacerated area. A 2 × 10 mm balloon catheter is then advanced and parked at the level of the laceration and inflated at 1 Atm on three sequential occasions for up to 20 min per inflation. Adequate balloon occlusion can be confirmed by injecting contrast through the guide. Further occlusion could seal the perforation. Nevertheless, if the angiogram reveals persistent and significant bleeding after each balloon deflation, attempts should be made to thrombose the lacerated vessel in order to stop the hemorrhage. Microcoils can be used for closure of the small artery. If there are no microcoils available, infusion of thrombin through the lumen of the inflated over-the-wire (OTW) balloon can be done. Careful positioning and sealing of the vessel are confirmed with injection of contrast from the guide and through the balloon lumen to ensure that there is no passage of contrast from the vessel lumen into the common femoral artery. Thrombin-JMI (Jones Pharma, St. Louis, MO) is to be diluted in normal saline at a concentration of 50 IU/cc. Subsequently, a total of three consecutive doses of 100 IU of thrombin can be administered through the balloon-catheter

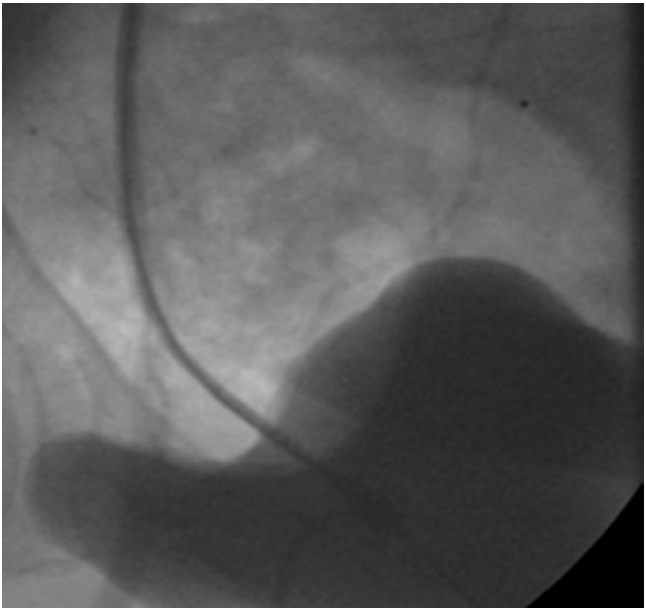

Figure 1-1 A dented bladder due to retroperitoneal hematoma. It looks different from the round shape of the bladder. (Courtesy of the Cardiac Catheterization Laboratories of Community Heathcare System, St Mary Medical Center, Hobart, IN. Reprinted with permission.)

lumen. Contrast can be injected through the balloon lumen after each dose of thrombin. When there is no further evidence of blood flow and no extravasation of contrast through the laceration, the balloon can be deflated. A selective injection with the guide in the common femoral artery is carried out to confirm the thrombotic occlusion of the lacerated inferior epigastric artery. An angiogram of the superficial femoral, popliteal, and infrapopliteal arteries consfirm the absence of thrombotic embolization and excellent flow in all these vessels [23].

Perforation: If a balloon bursts and perforates a peripheral artery below the inguinal ligament, the local bleeding can be controlled by direct pressure. In the case of higher perforation, a large peripheral balloon should be inflated above or at the rupture site to stop the bleeding and to seal the puncture site [24].

TECHNIQUE How to Seal a Perforation with a Covered Stent: Access is gained via the left femoral artery for retrograde approach to right iliofemoral angiography. A 6Fr internal mammary

catheter is inserted over a 0.035" glidewire, and this wire is used to cross into the right superficial femoral artery. This wire is exchanged for an 0.035" Amplatz super stiff wire, and an 8Fr × 65 cm long Superflex sheath advanced under fluoroscopy over the aortoiliac bifurcation to give good support in the right external iliac artery. Balloon tamponade of the perforation site is performed with a 5 minute inflation of a balloon at 2 Atm with persistent extravasation of contrast. An undersized self-expanding covered stent is then placed across the perforation site with a persistent leak. The stent graft can then be post-dilated with a balloon at 8 Atm with complete hemostasis and resolution of the free-flow contrast into the retro peritoneum [25].

Pseudoaneurysm: The incidence of pseudoaneurysm (PA) is 1–3% by clinical examination or 6% by ultrasound [26]. The main cause is inadvertent puncture of the SFA. Femoral PA forms when the puncture site does not close and there is continuous flow into a small perivascular space contained by the surrounding fibrous tissue and hematomas. It is suspected by the presence of a laterally pulsatile mass, an arterial bruit, and tenderness at the vascular access site. Confirmation is made by ultrasound, which shows a hypoechogenic cavity with flow through a neck directly visible by color Doppler, and pulsed Doppler evidence of to-and-fro flow between the cavity and the arterial lumen during systole and diastole [26]. Pseudoaneurysms are characterized by the presence of a to-and-fro blood flow across the PA neck during systole and diastole. Hematomas are seen as hypoechoic collections without any Doppler flow movement. Deep vein thrombosis (DVT) is characterized by a lack of venous compressibility, obstruction of venous return, and a hypoechoic or isoechoic signal [27].

Indications for aggressive management include: large size of the PA, whether it has increased in size, and the need for continued anticoagulation. Usually the small PAs (<3 cm in diameter) will close spontaneously, presumably due to thrombosis. A follow-up ultrasound 1–2 weeks later often demonstrates spontaneous thrombosis and obviates need of surgical repair. The >3 cm diameter PAs are less likely to close spontaneously. When PAs persist beyond 2 weeks or expand, the risk of femoral artery rupture necessitates correction.

The simplest method of treatment is to use a mechanical compression device (Femostop, Radi Medical Systems AB, Sweden). The success rate is 74% with a mean compression of 33 min [26]. The failed patients underwent successful compression guided by ultrasound. Contraindications of mechanical compression are listed in Table 1-2. Ultrasound-guided compression is commonly used with success related to the anticoagulation status and a PA that can be readily visualized and compressed [26]. However, the newest modality of treatment is to inject thrombin into the PA. The technique is simple, quick, and painless [28]. Surgery is indicated rarely when the above-mentioned management fails.

Table 1-2 Contraindications of Mechanical Compression for Pseudoaneurysm

1	Sign of local infection
2	Critical limb ischemia
3	Large hematoma with overlying skin necrosis
4	Injuries above the inguinal ligament

TACTICAL MOVE

BEST Options for Exclusion of Pseudoaneurysm [28]

1 ⌛ **FIRST Best option:** Mechanical compression therapy if there is no thrombin available

2 **$**⌛ **SECOND Best option**: For patients who fail empiric compression: ultrasound-guided compression

3 **$** ⌛ ♦ For patients on anticoagulant or having contraindication to compression: percutaneous injection of thrombin

4 **$$** ⌛⌛ ♦♦ Investigational: coil embolization (disadvantage: coils may fall through the skin or irritate when moving the leg) and covered stent (disadvantage: 12% risk of stent occlusion).

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Chapter 2
Angiographic Views

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complication

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GENERAL OVERVIEW

The goal of coronary angiography is to delineate clearly a lesion in two orthogonal views, so its morphology can be assessed accurately, the subsequent results of interventions can be compared more objectively, and the changes due to complications can be detected early. Accurate views of the ostial segment of the artery involved, the direction, and course of the segments proximal to the target lesion, are also needed to plan the accurate and timely movement of interventional devices. In order to evaluate difficult eccentric lesions, multiple views may be needed at slightly different angles. However, when a vessel bends in more than one plane, no single angiographic view can overcome multiple foreshortenings. So one must individualize and select the degree of angulation that best visualizes the problem area [1]. In general, the art of angiography is to expose the most by showing the least foreshortened coronary artery segment at an angulation that causes the lowest radiation to the operators and by the least number of x-ray pictures needed.

Guidelines for Moving the Image Intensifier: There are a few rules that govern the visualization of the artery by moving the image intensifier (camera tube) above the patient. The first rule is that the left circumflex (LCX) goes with the image intensifier and the left anterior descending artery (LAD) goes in the opposite direction. In other words, moving the image intensifier leftward to the left anterior oblique (LAO) view will project the LCX to the left on the x-ray picture and the LAD to the right (rule #1). Cranial angulation will elevate the LCX up

Table 2-1 Movement of Vessels and Landmarks According to Direction of the Camera Tube

Same direction	Opposite direction
LCX	LAD
Spine	Diagonals
	Diaphragm

and pull the LAD down. It is the reverse with caudal angulation. The same rule is applied to the diaphragm and the spine (Table 2-1).

The second rule is that in order to straighten a very tortuous coronary segment, the image intensifier should be moved to an angle more-or-less 90º opposite to the present one, then the tortuous area will be seen as straightened (rule #2). The goal of these maneuvers is to view the arterial segment in its most direct (orthogonal) angle, with the least angled projection effect.

STRATEGIC MAPPING

How to Perform a Coronary Angiogram: There are many ways to start a coronary angiogram: The anterior-posterior (AP) or right anterior oblique (RAO) or LAO view. However, after the first basic view, the next picture should show either a lesion or prove the patency of a major branch. In the left coronary angiogram there are a few areas to be scrutinized: The left main (LM), proximal, mid and distal LAD, proximal, distal LCX, obtuse marginal (OM) and diagonals. The areas of interest in the right coronary angiogram are: the ostial, proximal, mid and distal RCA, with the right ventricular (RV), posterior descending artery (PDA), posterior lateral branch (PLB), and sinus node branches. Usually, the operator likes to visualize the area of interest with the culprit lesion quickly.

First Neutral AP View: We prefer the first view as the neutral AP view because in this view, we have a global assessment of the LM, the proximal segment of the LCX and the exact position of the LCX in comparison with the LAD. In this AP view, usually the distal LM, the proximal LAD and LCX would overlap each other so this view would dictate the next area to be elucidated and practically the next move of the image intensifier.

RAO and AP Caudal View: After a global assessment of the LM and to be sure there is no LM disease with the AP view, a severe LCX lesion can be ruled in or out very quickly by only one RAO caudal or AP caudal view. Most of the time, with only two views, we can rule out severe LM and LCX disease, then we can concentrate on scrutinizing the distal LM and the proximal

LAD. Usually, at this stage, the above views would have given enough information regarding the mid and distal segment of the LAD or LCX.

Two Views in a Case of LM: In case of left main disease, it is not safe to take too many pictures. The surgeon may need only the mid and distal LAD and the OM so they can insert the bypass grafts. So there are only two views which show all the left system (LM, LAD, LCX), needed for coronary artery bypass graft (CABG): the AP caudal and AP cranial views.

TECHNICAL TIPS

****How to Fully Expose the LCX (1):** After the first plain AP view, the goal of the next angulation is to see clearly the LM at the same time the LCX. If the LCX is below or at the same level with the LAD in the plain AP view, then the next view would be any caudal view or maneuver which pulls the LCX down further. Deep inspiration would elongate more the LCX, so there is no foreshortening in the proximal segment or no overlapping by the LAD (Figure 2-1). These are the two views: RAO caudal and AP caudal. If the proximal segment of the LCX is quite tortuous in the AP view then the RAO caudal view will elongate the LCX and straighten the proximal segment (rule #2). If in the AP view, the proximal segment of the LCX is just foreshortened without being too tortuous, then the next view should be the AP caudal view, with deep inspiration to elongate the heart, depress the diaphragm and so pull the LCX straight down further (rule #1). These two views (RAO caudal and AP caudal) will best expose the whole LCX. Most of the time, just after a second x-ray picture, almost all the segments of the LM and the LCX arteries are completely scrutinized.

****How to Fully Expose the LCX (2):** If the LCX is seen above the LAD on the plain AP view, then the best maneuver is still to try to elongate the LCX by pulling it down further in the RAO caudal or AP caudal view. The reason is that all cranial angulations will project and foreshorten the LCX further above the LAD. Then a LAO cranial or caudal (spider) view will be a good alternative view for the proximal LCX, however, it will seen over the hazy background of the spine. The LAO caudal view is also very helpful for wire entry into the LAD, LCX and of the ostium of the first OM (Table 2-2).

****How to Fully Expose the LAD by Moving Away the LCX:** If in the AP view, the LCX is clearly below the LAD, then the next view has to be any caudal view to pull the LCX down further in order to remove overlapping of the LCX over the proximal LAD (rule #1). This view could have been used to expose the LCX and the LAD at the same time. If the LCX is at the same level with the LAD, then the next angulation is still any caudal view with deep inspiration to pull the LCX down and to uncover the proximal LAD overlapped by the LCX.

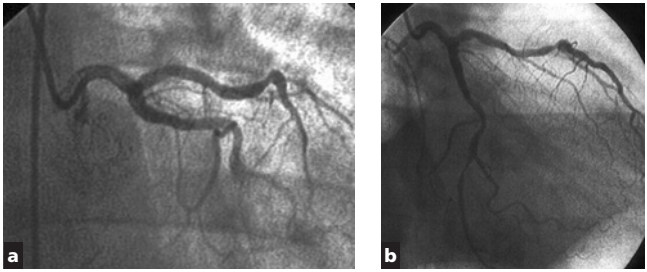

Figure 2-1 Angulation when the LCX is below the LAD. (a) The LCX is below the LAD in this plain AP view. It is also tortuous so the next view would be the RAO caudal view. (b) In this RAO caudal view, the LCX is well elongated and so fully exposed. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

Table 2-2 How to Expose the LCX

Location of LCX	Best next view
If the LCX is below or same level as the LAD in the AP view	RAO or AP caudal
If the LCX is above the LAD in the plain AP view	RAO or AP caudal LAO caudal (best alternative view) LAO cranial (best alternative view)

Usually the RAO caudal view would expose well the LCX and the LAD and has less interference by the diaphragm than the AP caudal view. However, the main effect of these two RAO and AP caudal views is to remove the LCX away from the LAD and does not purposely expose the LAD. The views that purposely expose the LAD are the RAO cranial and AP cranial views. If the LCX is above the LAD, then the only way to see the LAD is to move the LCX further above the LAD (rule #1) by using the AP cranial view, with cranial angulation as deep as possible. It may help if the pillow of the patient can be removed temporarily so the camera can be angled more in the cranial direction. An RAO cranial view is also good to lift the LCX up and expose totally the proximal and mid LAD. However, in these RAO or AP views with extreme cranial angulation, more OMs would overlap the mid and distal LAD, especially if the LCX is long and dominant (rule #1).

****How to Purposely Expose the LAD:** If the LAD is seen to be tortuous in the RAO caudal view (which is usually the second or third view of the angiographic sequence), then the next view to straighten the LAD is the RAO cranial view (rule #2). In order to remove the LCX overlapped on the LAD, if the LCX was seen above the LAD, then really deep inspiration will elevate the LCX further above the LAD. If the LCX was seen below the LAD in the AP view, then no deep inspiration

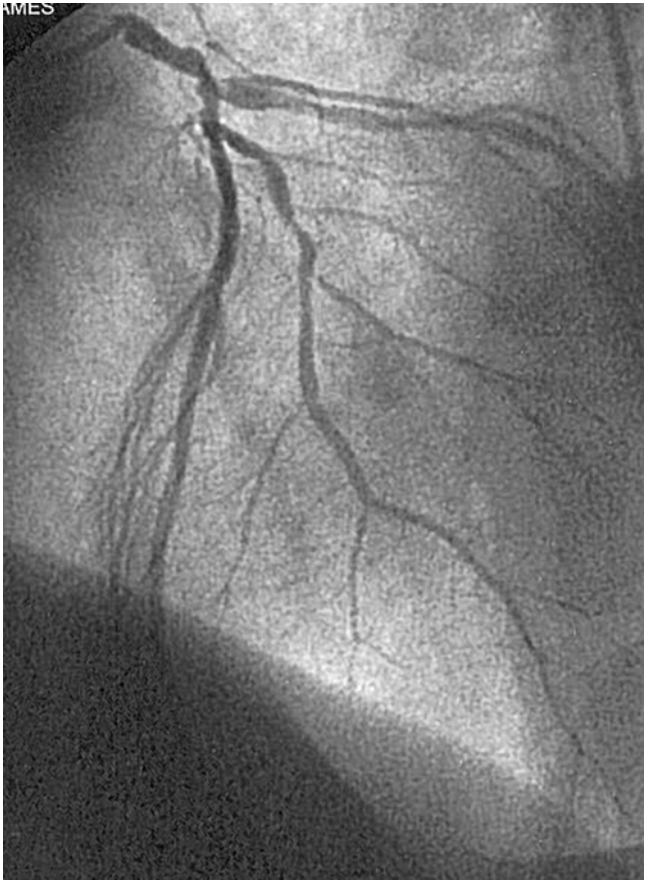

Figure 2-2 Boundaries of the LAO cranial view. A triangle created by the diaphragm, the spine, and the edge of the intensifier. The LAD should be seated at the top of this triangle for adequate visualization. The diagonals are on the right side and the septal arteries on the left side. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

is needed; if not, the LCX would be elevated and overlap the proximal LAD. These maneuvers can be repeated for the AP cranial view.

****In Order to Completely Scrutinize the LAD, do we Need a LAO Cranial View?** A full exposure of the LAD always requires the LAO cranial view to separate the LAD and the diagonals and expose the lesions at the ostium or bifurcations of the diagonals (Figure 2-2). In patients with prominent abdomen, the AP cranial view with really deep inspiration would move the LCX up high above the LAD (rule #1) and the diaphragm down in order to uncover the whole LAD.

Table 2-3 How to Expose the LAD and the Diagonals

Exposing the LAD	
First AP view	*Next view with deep inspiration*
If the LCX is below the LAD	RAO cranial or AP cranial
If the LCX is the same level	RAO cranial or AP cranial
If the LCX is above the LAD	RAO cranial or AP cranial with deep inspiration
Separating the LAD and the Diagonal	
First RAO view	Next view
If the first diagonal is above the LAD	RAO cranial view
If the first diagonal is below the LAD	RAO caudal view

****Angulations to Separate the LAD from Diagonals:** In the RAO view, if the first diagonal is above the LAD and overlaps the proximal LAD, a cranial angulation would separate well the LAD and its diagonals. However, the LCX would be moved up and overlap the proximal LAD. If the diagonals are seen below the LAD in the plain RAO view, then a caudal angulation would help to separate the LAD and its diagonals branches (Table 2-3) [2].

LEFT MAIN

The left main has variable length (1–10 mm), no side branches, and bifurcates into the LAD and LCX. Occasionally, there is no functional or very short LM and the LAD and LCX arise from separate ostia. The LM artery is examined through all the views that expose the LAD and LCX. It is an important part of the scrutinization of the coronary artery system, because a lesion on the LM is the main cause of mortality from even diagnostic coronary angiography. The first basic views of the LM are the AP view or the shallow RAO view. If these two views do not show clearly the LM, various views can be taken according to the vertical or horizontal positions of the heart and the length of the LM.

TECHNICAL TIPS

****How to Expose the LM, when the Heart is Horizontal:** The LM is short and the proximal LAD has a cephalad orientation; the LAO caudal view is better than the LAO cranial view [3]. The LAO caudal view would help to show the length of the LM, the direction of bifurcation of the LAD or LCX where the wire needs to be directed (Figure 2-3). To have the best LAO caudal (or spider) view, the tip of the guide should be positioned at the center of a full half-circle extending from 12 to 6 O'clock, formed by the shadow of the cardiac silhouette. However, angulation that is too steep would cause foreshortening of the LM and overlapping by the diaphragm and spine.

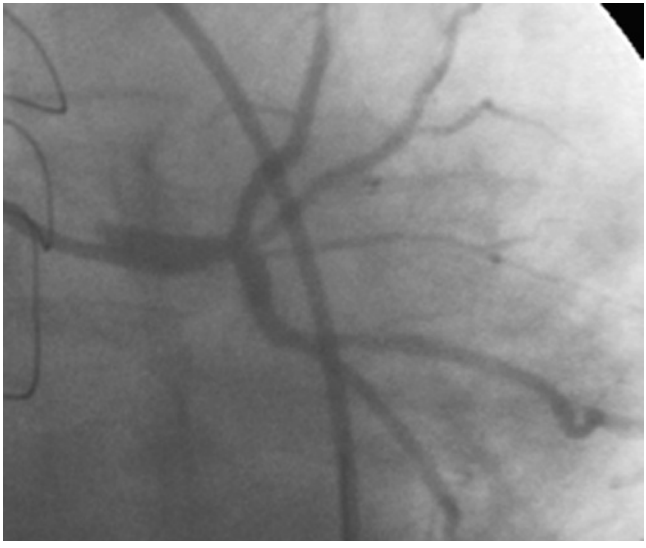

Figure 2-3 The LAO caudal view of the left main. In this (spider) view, the left main is seen very clearly at the bifurcation into the LAD and LCX [2]. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

Table 2-4 Other Possible Angles to Delineate the Left Main

Vessel segment	Routine views	Adjunctive views
Ostial	LAO caudal	AP caudal
Body	RAO caudal RAO cranial	AP caudal
Distal	LAO caudal RAO caudal	LAO cranial

****How to Expose the LM, when the LM is Long and has a Downward Direction:** As in vertical hearts, then the LAO cranial view will show best the LM and its bifurcation to the LAD and LCX [3]. An LAO/cranial angulation that is too steep would further foreshorten the LM and, in conjunction with poor inspiration, would produce a hazy background due to diaphragm overlapping.

If the above views could not clearly delineate the LM lesions, other possible views include all the possible combinations of angulation set by an image intensifier (except the AP cranial and lateral views) (Table 2-4) [4].

LEFT ANTERIOR DESCENDING ARTERY

The proximal LAD is defined as the segment from the ostium to the origin of the first septal. The distal end of the mid-segment is less rigorously defined and is typically the location where the LAD dips downward on a right anterior oblique view.

The AP Cranial View: This is one of the best first views that may delineate the ostial segment, then the mid- and distal segment of the LAD. In patients with long left main, during percutaneous coronary interventions (PCI) in the proximal or mid-LAD lesion, this view is very useful to show the course that the wire should enter the LAD and go forwards without interference from the septal and diagonal branches. The AP cranial view can be taken while the patient performs deep inspiration if there is intention to elevate the LCX above the proximal LAD. If the LCX is way below the LAD, then inspiration is not needed because it will elevate the LCX to overlap the LAD.

The LAO Cranial View: This view delineates clearly the course of the LAD, from its origin to the apex and the correlation with its septals and diagonals. In this view, the LAD is best seen if the tip of the guide is positioned in a triangle made with the spine, the diaphragm, and the edge of the intensifier, over a clear lung background (Figure 2-2). To move the spine away from the center, just move the tube to the left then the spine will be moved to the left (rule #1). If the result is suboptimal, better definition of the proximal LAD can be achieved by changing the steepness of the cranial angulation and by having the patient take a deep breath, to lower the diaphragm and thus making the heart more vertical.

This LAO cranial view would help to delineate any lesion on the LAD, especially at the bifurcations with the diagonals and septals. It helps to show the pathway of the wire. However, in this view, the proximal LAD is foreshortened so it does not give an accurate evaluation of the result of angioplasty or stenting in the very proximal LAD.

CRITICAL THINKING

How to Identify the LAD, Diagonals and Septals: The LAO cranial view is the best view to identify and confirm the identity of the LAD. The diagonals would be in the left and the septals would come out from the right of the screen. It is almost unthinkable if there are no septals. This view would confirm the identity of a compensatory enlarged diagonal because it has no septals (except in very rare cases) [3]. The diagonals would point more to the left side, however, a long LAD in a very dilated LV could have the apex moved towards the left of the screen too. Another way to differentiate the septals from the diagonals is that the diagonals move (buckle) during systole while the septals are straighter and move very little with ventricular contraction. The presence of the septals would confirm the identity of the artery that is the LAD.

The AP Cranial View: To see clearly the proximal and mid-segment of the LAD with its bifurcation, the AP cranial view could also show very clearly the ostial and the proximal segment of the LAD, if the LCX can be moved totally above the LAD. The mid-segment and distal segments of the LAD are well exposed in a single view on the screen so this view is a favorite view during percutaneous coronary intervention (PCI) where the operator can monitor the position of the tip of the guide, the movement of the devices in the proximal segment and of the tip of the wire at the distal segment.

The RAO Caudal View: It has much LAD and diagonal overlapping in the mid LAD. The proximal LAD also is foreshortened [5]. This view best demonstrates the lesions in the mid-LAD with relation to the septals.

The Lateral View: To highlight a lesion at the bifurcation of the LAD and the diagonals, the lateral view can help to pinpoint its location and assess its severity. It is more useful for a diagnostic injection rather than for an interventional procedure because a prolonged position of the arm above the head would make any patient tired and uncomfortable.

TECHNICAL TIPS

****Exposure of the High Diagonal:** Usually the ostium of a high diagonal is not well seen in the RAO cranial view because the area is overlapped by an elevated LCX, so the LAO with steep cranial view (LAO 10, cranial 40) can be tried. However, a good spider view with steep caudal angulation is most likely the best view to expose it (Figure 2-3).

****Best View for Ostial and Proximal LAD in Horizontal Heart or Short LM:** When the heart is horizontal, the LM is short and the proximal LAD has a cephalad orientation; the LAO caudal view is better than the LAO cranial view because of proximal circumflex overlapping [6]. Positioning the proximal end of a stent during PCI of the ostial LAD can be done best in the this caudal view. However, the proximal LAD segment is foreshortened so it is not best to check the complete deployment of a stent or the appearance of new dissection in this view.

****Best View for Ostial and Proximal LAD in Patients with Long LM:** If the LM is long, then the AP cranial view should be the first view, especially if the LCX can be moved above the LAD. If the LAD cannot be assessed in this view, the next best view is the RAO cranial which will best show the LM and its bifurcation to the LAD and LCX [3]. Thus the best views for distal LM or ostial LAD can be either with AP, RAO or LAO cranial angulation [4]. If not the next try would be the LAO caudal view. Appropriate views would

give the best delineation of the lesion if selected intelligently by the operator.

LEFT CIRCUMFLEX ARTERY

The proximal segment of the LCX begins from the ostium up to and including the origin of the first obtuse marginal (OM). The distal LCX is beyond this point. When looking at the LCX, a standard RAO caudal view may provide much needed information. However, a shallow angulation has two limitations: (1) it can foreshorten the proximal segments of the LCX, so the exact morphology of a lesion in that segment cannot be optimally assessed or the direction or its tortuosity is overlooked; (2) the ostial segment may be overlapped and is not seen clearly. The more caudal, the better the proximal part is seen. To complement the limitations of the RAO shallow caudal view, an AP caudal or an LAO caudal view can help to clarify the problem in that segment (Figure 2-3).

While taking this view, the patient is asked to take a deep breath, which moves the diaphragm downward and clears the field for optimal vessel opacification. In the case of mid- or distal LCX-OM intervention, an RAO caudal view can foreshorten the proximal LCX and could mask the severity of the takeoff angle with the LM, and the tortuosity and severity of any possible obstructive lesions at the proximal segment. On many occasions, only after unsuccessful advancement of balloons and stents across the proximal segment, is the severity of the lesions and tortuosity of the proximal segment of the LCX appreciated. If properly taken, the AP caudal or LAO caudal (spider) view would give a sharp delineation of the ostium and proximal segment of the LCX, so a wire can be shaped to enter the artery and the proximal segment without unexpected difficulty.

RIGHT CORONARY ARTERY

The proximal segment of the right coronary artery originates at the ostium and ends after the first curve. The mid-segment begins with the first curve and ends at the second curve, and is usually considered the straight segment in the RAO view. The distal segment is the remainder of the artery.

The origin of the RCA is extremely variable, from a straight perpendicular takeoff from the aorta, to a marked caudal direction, to a superior takeoff with the shepherd's crook configuration. A strong hand injection into the low right coronary cusp may help to show the origin of the RCA. If it is not seen, it may originate anteriorly, or from the left sinus of Valsava, or above the sinotubular ridge.

The LAO View: In the LAO projection, the artery appears like a letter C, while in the RAO position, it appears like a letter L. To check the correct alignment of the catheter with the RCA, the best view is the RAO-shallow cranial projection. In this location, the tip of the catheter is seen head-on as a circle (Figure 2-4a). If there is an angle formed by

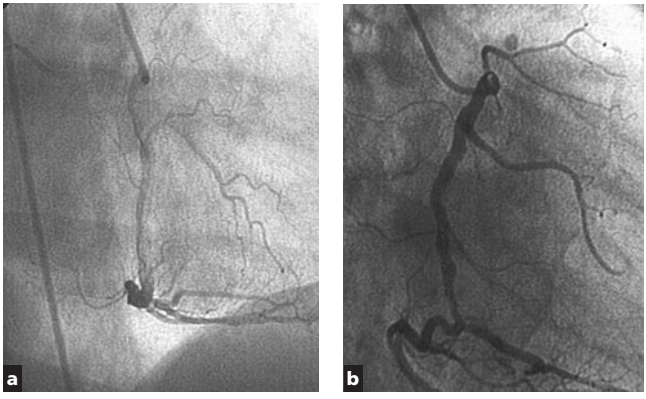

Figure 2-4 (a) The RAO view of RCA. In this view, the guide is truly coaxial, so the tip of guide will be seen head-on as a circle. (b) The RAO view of an RCA anomaly with the ostium in the anterior position. As the location of the ostium in this case is abnormal, so the tip of the guide points to the left. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

the tip of the catheter with the proximal RCA, then there is no coaxial alignment of the catheter (Figure 2-4b). There would be friction at the angle of transition, which may diminish the forwarding force, the torquing capacity, and obstruct the smooth advancement of interventional hardware across a tight lesion.

When imaging an ostial lesion, it is useful to place the catheter just under the coronary ostium and inject a large volume of contrast to define both the extent of the lesion and its precise position on the aortic wall. This is a particularly important view when contemplating deployment of a stent in the ostial RCA [7]. The exact location of the ostium can often be best seen in an extreme LAO position (more than 50°) or the LAO with caudal angulation.

In order to view the ostial segment of the RCA, the best view is the LAO caudal view that exposes well the ostial and proximal segment of the RCA. In order to view the distal PDA, then the LAO cranial view coupled with during deep inspiration, which moves the diaphragm outside the field, will expose the distal RCA and its bifurcation with the PDA. If there is a need for further exposure of the distal RCA, then an AP cranial view will complement fully the LAO caudal view.

The RAO View: In the LAO view, the mid-segment may not be visualized well because of overlap of the RV branch. The RAO view would separate the mid-segment of the RCA from the RV branches (Figure 2-4A). When wiring the RCA, an LAO view would help to direct the

wire from the catheter into the proximal segment. It also helps to appropriately select the different PDAs in the distal segment. However, because of the presence of a few marginal branches, an RAO view would help to direct the wire at the mid-segment, avoid the marginal branches, and advance it easily to the distal segment.

SAPHENOUS VEIN GRAFT

The principle of selecting the best angle for a venous or arterial graft is to angle a 90° view from direction of the bypass graft. In the case of the saphenous vein graft (SVG) to the OM, in the RAO caudal view, the direction of the SVG is up-to-down (or cranial to caudal), then the best view is the RAO caudal view because the RAO caudal forms a 90° angle with the SVG on the vertical plane and with the OM on the horizontal plane. The best view for the ostium or body of the SVG to the LAD and diagonals is the LAO view, the LAO cranial view for the distal PDA, and the LAO cranial and RAO cranial for the LAD or the diagonals.

INTERNAL MAMMARY ARTERIES

Usually the left internal mammary artery (LIMA) graft is inserted into the LAD, so the basic views of the LAD and its insertion site should be the LAO cranial or the RAO cranial views. The insertion site of the LIMA to the LAD can also be seen well in the lateral view because in this view the x-ray will arrive at the LIMA insertion site at a 90° angle with the distal end of the LIMA graft on the vertical plane while at 90° with the LAD on the horizontal plane.

Though the LIMA catheter is usually engaged on the AP view, to check the position of the catheter tip in relation to the ostium, the best views are the 60° LAO or the RAO 45°. These angulations would elongate the aortic arch and separate the subclavian artery to identify clearly the LIMA ostium.

In rare case there is difficulty engaging the left and right internal mammary arteries, a nonselective angiogram with the tip of the catheter just in the vicinity of the ostia can be done. Complete opacification of the vessel can be achieved with 10 cc manual injection with a blood pressure cuff inflated 10 mmHg above the systolic pressure on the ipsilateral arm [8].

TROUBLE-SHOOTING TIPS

****How to Select the Angulation:** There are a few rules how to select the angulations mentioned earlier. The main concerns are how to have the x-ray pictures showing the problematic areas with the least number of x-ray pictures required, the least amount of contrast agent and with the least radiation exposure to the pat-ient, operators and staff. How to angle the camera is summarized in Table 2-5.

Table 2-5 How to Select the Angle for the Camera

Problems	Solutions
The LAD is not central in the LAO cranial view	Move camera tube more LAO
The LAD is too tortuous or foreshortened in the LAO cranial view	Move camera more or less cranial
If LCX overlaps LAD in the RAO cranial view	Move the camera more cranial and deeper inspiration to lift the LCX more above the LAD or change to AP cranial view
If the LCX is clearly below the LAD in the AP view	RAO caudal view with deep inspiration
If the proximal LCX is too tortuous in the AP view	Deep inpiration and take RAO caudal view
If the proximal LCX is foreshortened in the AP view	Make view more caudal with deep inspiration
If there is difficulty in cannulating the RCA	Take an RAO view to check co-axial position of the guide

CAVEAT

Deceiving Angiographic Views: There are angiographic views that minimize the severity of an angulated segment or the severity of a lesion. The most common situation is the RAO caudal view for a lesion in the LCX. This view foreshortens the proximal segment of the LCX so the ostial lesion of the LCX can be missed and the lesions in the proximal segment can be overlooked. In the RAO cranial or LAO cranial views, the lesion in the distal LM can also be missed; if there is a problem advancing the device or thrombus formation after manipulation of interventional hardware, then the severity of the lesion is much more appreciated. In the LAO cranial view, the lesion in the proximal LAD can be missed, because it is foreshortened and it can be seen better in the RAO cranial view or AP cranial view. During a procedure in a RCA lesion, the guide is thought to be coaxial in the LAO view; however, after failing to advance the interventional devices or difficulty in withdrawing them, it is found that the guide is not coaxial in the RAO view (Table 2-6).

Table 2-6 Suboptimal and Deceiving Angiographic Views

1. RAO caudal views for the ostial and proximal LCX. **Better view:** AP caudal with deep inspiration (or vice versa)
2. LAO view of the proximal or ostial RCA. **Better view:** LAO caudal to have better delineation of the ostium. RAO view to check coaxial position
3. LAO view for origin of distal PDA. **Better view:** LAO cranial or AP cranial view with deep inspiration in order to depress the diaphragm further
4. AP view of the distal LM. **Better view:** LAO caudal, (spider view) or cranial angulation
5. LAO cranial view for the proximal LAD. **Better view:** AP cranial

CAVEAT

Missing Lesions: Coronary angiography or "luminography" is well known to miss severe lesions especially the short, napkin ring lesion or short aorto–ostial lesions. The reason is that when the lesion is viewed from an angled projection, the lesion is not seen because the adjacent contrast-filled vessel segments are projected over the short and diseased segment and mask it. In the case of ostial lesion, the tip of a small catheter can be engaged too deeply without causing venticularization of blood pressure and spill-over contrast in the aorto–ostial area would mask the short, severe ostial lesion. This is the same problem of PCI in ostial lesion where it is difficult to position the proximal end of the stent because an angiogram will spill contrast over the ostial area.

MAGNIFICATION ARTIFACTS FOR BALLOON OR STENT SIZING

In the RAO caudal view, the size of the tip of the guide is projected smaller than the projected size of the LCX, OM or distal RCA because the LCX, OM, distal RCA is more posterior so it is more enlarged then the tip of the guide on the image intensifier. We encounter the same problem measuring the size of the distal LAD in the RAO cranial view. In all circumstances, the image intensifier should be as close to the patient's chest as possible. Caution should be exercised when measuring the size of the arteries (Table 2-7).

CAVEAT

OVERSIZING the Vessel in Mid and Distal Segment: In many patients undergoing PCI in the left circumflex, the reference size of the mid-segment of the LCX is measured on the RAO caudal view. In this view, the tip of the guide at the LM ostium is more anterior while the mid-segment of the LCX is more posterior, at the level of the aorta, so the mid-segment of the LCX (and the shaft of the guide compared with its tip) projected bigger on the camera screen. This is why the size of LCX as measured by quantitative coronary angiography (QCA) can be quite deceptive. This is the cause of balloon or stent oversizing in PCI of LCX. The same problem happens with the distal RCA in the AP cranial view and the mid and distal segment of the LAD in the RAO cranial view (Table 2-7; Figure 2-5).

Table 2-7 Best Views for Balloon or Stent Sizing

The Left Anterior Descending Artery	
Segment	*Best View*
Proximal or Mid LAD	RAO or AP cranial
Distal LAD	RAO cranial (caution for magnification artifact)
The Left Circumflex Artery	
Segment	*Best View*
Proximal LCX	RAO caudal and AP caudal
Distal LCX or OM	RAO caudal (caution for magnification artifact)
The Right Coronary Artery	
Segment	*Best View*
Proximal, mid-RCA	RAO, LAO
Distal RCA, PDA, PLB	AP, LAO cranial (caution for magnification artifact)

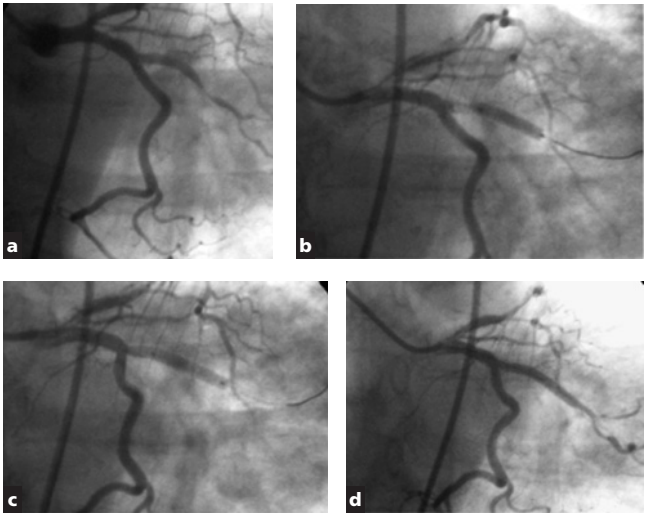

Figure 2-5 False magnification of the LCX. (a) With the size of the guide tip as reference, the OM was measured as 3.8 mm proximally to the lesion and 3.3 mm distally to the lesion. Therefore, a 3.25 mm balloon was selected for predilation. (b) During inflation, an angiogram showed total occlusion of the artery, so the balloon fits well. (c) The body of the guide looked bigger than the tip, so a 3.0 mm stent was selected and deployed. The angiogram during inflation also showed the same size between the proximal segment and the stent size. (d) The post-stenting angiogram showed there was no discrepancy between the diameter of lumen in the stented area and its proximal segment. The real diameter of the artery was around 3.00 mm not 3.8 mm as measured with the tip of the guide as reference. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

CAVEAT

UNDERSIZING the Vessel: In the LAO cranial view, the mid LAD looks smaller because the tip of the guide is behind and the artery is near the image intensifier so the guide is projected larger on the screen while the LAD looks smaller. By that, the size of the balloon or the stent looks smaller than in reality.

Radiation Exposure to the Operators: The operator should be cautious in using the views in order to protect him or herself, and the staff, from radiation exposure.

CAVEAT

Angulations that Gives the Most Radiation to Operators: The steep LAO cranial angulation is the view that causes the most radiation exposure for operator using the right femoral approach. It is due to radiation from the x-ray generator below the table and the x-ray beam is redirected and scattered off when meeting the patient. This radiation scatters toward the operator, and there is increased scatter produced by the higher kVp level required for hemiaxial angulation [9]. During PCI of obese patients, in order to permit adequate x-ray penetration, avoid deep angulation, especially caudal angulation. The image magnification should be also lower, to reduce patient and operator radiation exposure and limit the amplitude of table panning, thus reducing motion artifacts. In selected suspicious areas, the areas will be re-imaged with higher magnifications.

CORONARY ARTERY ANOMALIES

The most common anomaly is the variation of coronary artery origin from the aorta. Usually, they are of no clinical significance, except in the case of origin of the LAD from the right sinus or the RCA from the left sinus that is compressed resulting in ischemia and sudden death [10,11]. When the LCX originates from the RCA or right sinus, usually it takes the retroaortic course to supply the lateral wall of the ventricle and is benign. The left or right coronary artery can originate from the posterior sinus (very rare) or from the ascending aorta like a bypass graft [12]. Besides an ectopic origin, their anatomical course is usually normal. These anomalies are considered benign.

When the LCA or RCA originate from the opposite sinus, there are four pathways. The rare form is the interarterial course and the most common is the septal course. The other two forms are the retroaortic and the anterior courses. The interarterial course is the most serious one because it can cause ischemia, leading to sudden death.

TECHNICAL TIPS

****The Dots and the Eyes:** The course of an anomalous coronary artery is confirmed by the filming of the pathway in the 30° RAO view. In this visualization, a dot representing the artery seen on end is noted. The most severe one, the interarterial pathway of an anomalous LM crossing between the aorta and the pulmonary artery, is recognized by the position of the "dot" anterior to the aorta. If the "dot" is behind the aorta, this is the retroaortic benign pathway [13]. The septal pathway is recognized by the fish hook picture in the RAO view, because the LM goes down to the septum then comes up to the epicardium, making a picture of a fish hook. Then the LCX would curve backward and form the "eye" with the LCX as the upper border [13]. In the anterior (pathway) the LM is in front of the pulmonary artery. This pathway is recognized by the "eye" with the LM as the upper border and the LCX as the inferior border (Figure 2-6).

The Single Coronary Artery: Defined as an artery that arises from an arterial trunk and nourishes the entire myocardium, single coronary artery (SCA) is rare.

The Left Circumflex Artery from the Right Sinus: The second most common coronary anomaly is the LCX arising from the proximal RCA. This variant is benign. When the LCX arises from the right coronary cusp or the proximal RCA, it invariably follows a retroactive course, with the LCX passing posteriorly around the aortic root to its normal location. On the LAO, the LCX is seen originated from the proximal RCA. The LAD is a large one without LCX. In a 30° RAO view, the LCX will be seen curving in the posterior area and is seen head-on, as a dot, posterior to the aorta [13] (Figure 2-7). When the LCX originates from the proximal RCA, near the ostium, if the catheter tip is engaged too deeply, it can pass the ostium of the anomalous LCX and miss opacifying the LCX.

The Right Coronary Anomalies

Anterior Position of the Ostium: If the origin of the RCA is minimally displaced anteriorly, at that time, the tip of the right Judkins catheter may not be directed to the right, but rather looks foreshortened in the familiar LAO view. Directing the tip to the right in the usual fashion using the LAO view permits easy cannulation of the anteriorly directed RCA orifice [14]. In the RAO view, there would be an angle between the catheter tip and the ostium with the tip pointing toward the left (see Figure 2-4B).

Anomalous Origin of the RCA from the Left Sinus: When the RCA arises from the left sinus or from the proximal LM in the RAO view, the RCA will be seen head-on, as a dot anterior to the aorta [13]. Figure 2-8 shows a middle-aged nurse with acute myocardial infarction. Two years later her son had an angiogram that showed exactly the same anomaly (Figure 2-8).

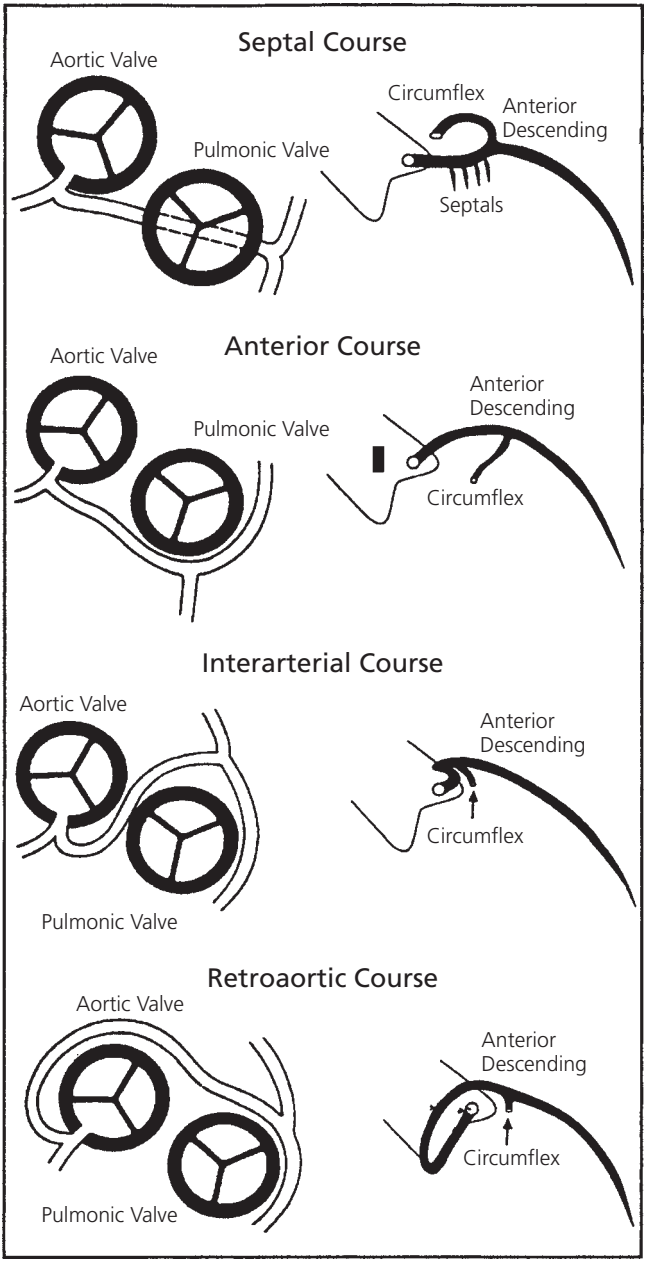

Figure 2-6 General view of coronary anomalies. (Adapted from Serota H, Barth III CW, Seuc CA, *et al.* [13].)

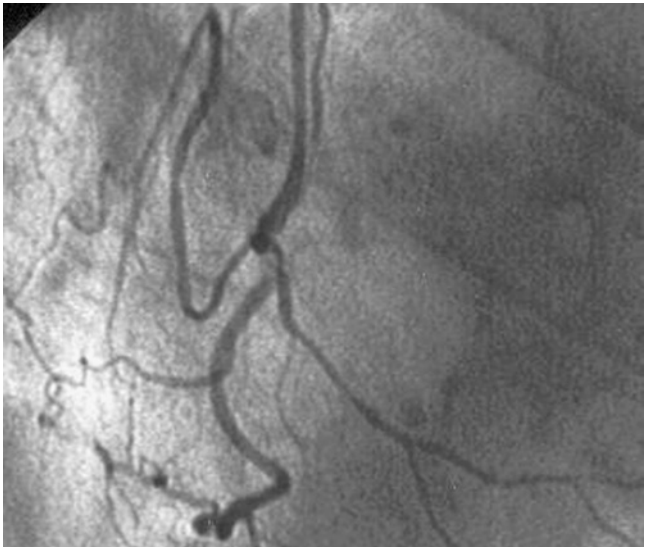

Figure 2-7 In this RAO view, the LCX that is originated from the RCA is seen in a retroaortic pathway as the dot is seen behind the aorta and the artery curves posteriorly. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

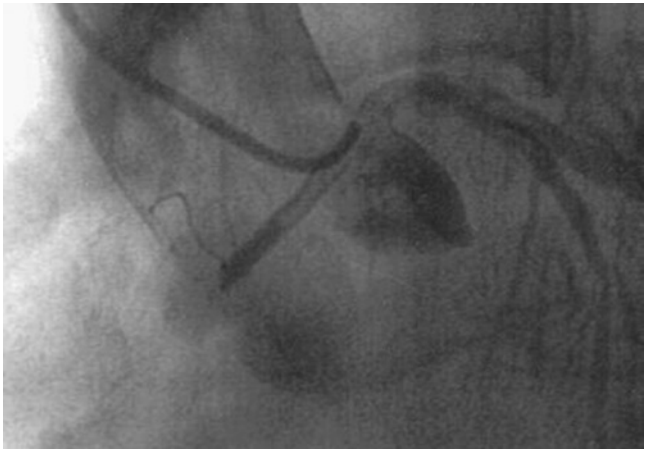

Figure 2-8 In this left coronary injection, an anomalous RCA originating from the left sinus was seen. It was occluded because of AMI. It was then successfully opened. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

The Left Anterior Descending Artery Anomalies

LAD from the RCA or Right Sinus: Anterior Free Wall Course: The LAD crosses the anterior free wall of the right ventricle in front of the pulmonary artery, then at the mid-septum turns toward the apex. On the 30° RAO view, the LAD will pass to the left and upward before turning to the apex. This coronary anomaly is benign [13].

LAD from the RCA or Right Sinus: Septal Course: The LAD runs an intramuscular course through the septum along the floor of the RV outflow tract. It then surfaces at the mid-septum and turns toward the apex. In the RAO view, the LAD will pass to the left and downward before turning toward the apex. This type of coronary anomaly is considered benign without ischemia [13].

The Left Main Coronary Artery (LM) Anomalies: The incidence of LM originating from the right sinus is very low (1.3%) [15]. The artery, seen in the RAO view, may course in front of the pulmonary artery (anterior course), through the septum (septal course), between the aorta and the pulmonary artery trunk (interarterial course), or behind the aorta (retroaortic course) (see Figure 2-6). Accurate diagnosis is prognostically important because of fatal events associated with the interarterial pathway [16].

The Septal Course: The LM runs an intramuscular course through the septum along the floor of the RV outflow tract. It then surfaces at the mid-septum where it bifurcates into the LAD and LCX. Because the artery divides at the mid-septum, the initial portion of the LCX curves above the LM toward the aorta (the normal position of the LAD) and forms an ellipse with the LM (similar to the shape of an eye, with the LM as the inferior border) seen best at the 30° RAO view. The LAD is relatively short because only the mid- and distal LADs are present. One or more septal vessels can originate from the LM. This type of coronary anomaly is considered benign without ischemia [13] (Figure 2-9).

The Anterior Free Wall Course: In the anterior course, the LM crosses the free wall of the right ventricle, in front of the pulmonary artery, and divides into the LAD and LCX at the mid-septum. The LCX would curve back toward the aorta (the position of the normal LAD). On the 30° RAO view, the circumflex forms an ellipse ("eye") with the LM on the superior border. There is no myocardial ischemia associated with this coronary anomaly [13].

The Retroaortic Course: In this type, the LM goes around the aortic root to its normal position on the anterior surface of the heart. It divides into the LAD and LCX at its normal point so the LAD and LCX have normal length and course. In the RAO view, the LM is seen head-on, as a circle, posterior to the aorta. This retroaortic dot is diagnostic of a posteriorly coursing artery. There are only rare cases of ischemia reported with this type of anomaly [13].

The Interarterial Course: In this type, the LM courses between the aorta and the pulmonary artery to its normal position on the anterior

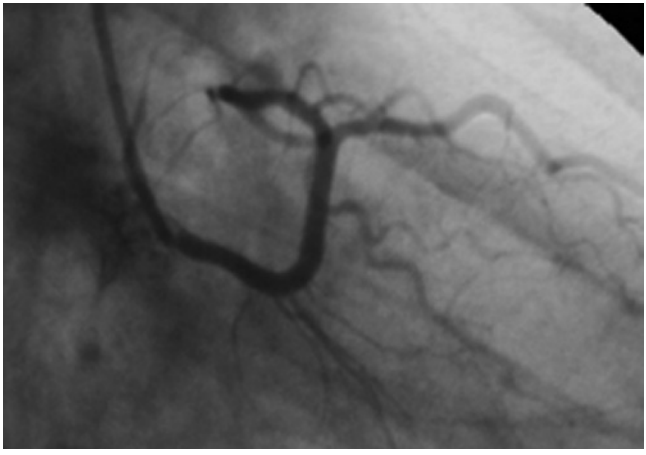

Figure 2-9 The LM from the right sinus by the septal course. The LM forms the inferior border of the eye while the LCX forms the superior border of the eye. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart, IN.)

surface of the heart. In the RAO view, the LM is seen head-on, as a dot, on the anterior aspect of the aorta [12]. The circumflex arises with a caudal orientation. This type of anomaly is associated with exertional angina, syncope, and sudden death at young age [13].

Anatomic Consideration of the Ostial Segment: Not every anomaly has a wide ostium that the tip of the guide can hook onto, or a narrowing at the opening that needs to be stented. There have been several reports that an anomalous RCA from the left coronary artery can leave the aorta in oblique fashion, so the ostium has a slit-like configuration formed by flaps of aortic and coronary tissues. During exercise, the aorta can expand its part of the flap, narrowing farther the slit-like opening and causing ischemia [17].

Mechanism of Ischemia due to Anomalous Pathway: If an anomalous artery has to course between the aorta and the pulmonary artery, the expansion of the aorta during exercise can cause narrowing of the mid-segment and subsequent ischemia. If it happens in young patients, there is indication for corrective surgery. If the anomaly is found incidentally in asymptomatic elderly patients, surgery is indicated only if objective signs of ischemia can be demonstrated. The reason is that the hardened aorta in older patients does not expand much any more, so it does not cause as much exercise-induced ischemia as in young patients [18].

Some anomalous coronary arteries with an intramural course may adhere to the wall of the aorta, and can even share a common media with the aorta without intervening adventitious [19].

Left Main from the Posterior Sinus: In the AP view, the noncoronary cusp is on the right side and inferior to the left aortic sinus. However, it is seen best in the RAO view, in its posterior location, and identified by the catheter tip in the posterior direction. An injection in the sinus would outline the artery and the posterior wall of the aorta [20].

Right Coronary Artery from the Pulmonary Trunk: This anomaly is very rare. The RCA is originated from the pulmonary trunk. Because the low pulmonary resistance, so the fully oxygenated blood arriving in the anomalous coronary artery, via collaterals from the normal coronary artery, is stolen by the pulmonary trunk, resulting in myocardial ischemia. The treatment includes surgical ligation of the RCA and bypass or re-implantation of the RCA [21].

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Chapter 3
Guides

Thach N. Nguyen, Tan Huay Cheem, Abhiram Prasad

*Basic; **Advanced; ***Rare, exotic, or investigational. **$** <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

An optimal guide provides a stable platform for the operator to advance devices to the coronary ostium, through tortuous arterial segments, and across tight lesions. The guide is selected according to the size of the ascending aorta, the location of the ostia to be cannulated, and the degree of tortuosity and calcification of the coronary artery segment proximal to the target area. Once engaged in the ostial segment, its soft tip is to be positioned with atraumatic coaxial alignment. Compared to diagnostic catheters, guides have a stiffer shaft, larger internal diameter, shorter and more angulated tip (110º vs 90º) and re-enforced construction (3 vs 2 layers).

As a device is pushed forwards, any guide with a tip held still, not being displaced, will be the ideal guide for the procedure. In a simple case with easy access, the Judkins guide, even in a relaxed position in the aortic sinus, can provide an adequate platform to advance the device. It is the ideal guide position in aorto-ostial lesion stenting. In complex cases, where more resistance is encountered, any selected guide with its secondary curve well positioned and standing firm against the opposite aortic wall would provide the strong and stable platform needed for device advancement.

Practical Analysis of Guide Design: The most commonly used guides are the Judkins, Amplatz, and Extra-back-up guides. The others that have a niche in various situations include the Multipurpose for the right coronary artery (RCA) bypass or a high left main (LM) takeoff, and the left internal mammary artery (LIMA) catheter for the superiorly oriented graft and the right and left coronary bypass graft. In the literature there is discussion about guides with passive or active support. Passive support is the strong support given by the inherent design of a guide with good back-up against the opposite aortic wall and stiffness from manufactured material. Additional manipulation is generally not required. Active support is typically achieved by either manipulation of the guide into a configuration conforming the aortic root, or by subselective intubation with deep engagement of the guide into the coronary vessels [1].

The Judkins Guide: The Judkins left (JL) guide is designed for coronary angiography with its primary (90°), secondary (180°), and tertiary (35°) curves fitting the aortic root anatomy so it can engage the LM ostium without much manipulation. It knows where to go unless thwarted by the operator [2]. Because of the 90° bend at its tip, it does not make perfect coaxial alignment. On many occasions, even when the secondary curve does not sit well on the opposite aortic wall or coronary sinus, diagnostic angiography can still be performed satisfactorily while there is no adequate support for advancement of interventional devices during percutaneous coronary interventions (PCI) [1].

The Amplatz Guide: The left Amplatz guide is designed with its secondary curve resting against the noncoronary posterior aortic cusp, while in the right Amplatz guide, the secondary curve rests against the left aortic cusp [3]. This guide offers a firm platform for advancement of device. It is best in the case of a short LM, with downgoing left circumflex artery (LCX) or RCA or in superior take-off. However, because its tip is slightly pointing downward, there is higher danger of ostial injury causing dissection.

The Multipurpose Guide: This guide is straight with a single minor bend at the tip. With the exception of a few cases of high LM takeoff or downward RCA, which can be cannulated well with this Multipurpose guide, it is not routinely used for native coronary PCI.

The Extra-Back-Up Guide: The names of these Extra-back-up (EBU) guides vary (Voda or XB, EB, C, Q, or Geometric curve guides) according to manufacturers. The common design is that their long tip forms a fairly straight line with the LM axis or the proximal ostial RCA, so they can provide a better transition angle with less local friction. They have a long secondary curve which is designed to abut the opposite aortic wall, so the tip in the coronary artery is not easily displaced and hence provide a very stable platform [4].

GUIDE MANIPULATIONS

Safety Measures: In any situation, the basic safety measures should be applied rigorously when manipulating guides. They are listed in Table 3-1 [5].

TECHNICAL TIPS

***Advancement Through Tortuous Iliac Artery:** Because of excessive tortuosity of the iliac artery, rotations at the proximal end do not transmit similar motion to the distal tip. If not constantly watched, the guide can twist on itself. Simple gentle movement of the guide in and out, often over a very short distance, transmits torque to the tip [6]. In these situations, a 23 cm sheath may help to overcome the problem of iliac tortuosity. In the rare case of a patient with abdominal aortic aneurysm (AAA), a 40 cm sheath is needed. A more simple technique is by torquing a guide still cannulated inside by a stiff 0.38″

Table 3-1 Standard safety techniques

1	Aspirate the guide vigorously after it is inserted into the ascending aorta for any thrombus or atheromatous debris floating into the guide
2	Insist on generous bleed back and introduce devices into the Y adapter on flush to avoid air embolism
3	Flush frequently to avoid stagnation of blood and thrombus formation inside the guide
4	Constantly watch the tip when withdraw interventional device from coronary artery and especially in patients with ostial or proximal plaques
5	Watch the blood pressure curve for dampening to avoid inadvertent deep engagement of the tip
6	During injection, keep the tip of the syringe pointed down so any air bubbles will float up and are not injected into the coronary system

wire inserted through a Y adapter. Manipulate the tip near the ostium, remove the stiff wire, flush the guide, and then engage the tip to the ostium [6].

***Dampening of Arterial Pressure:** The guide can cause fall of diastolic pressure (ventricularization) or fall of both systolic and diastolic pressure (dampened pressure). The causes can be due to significant lesion in the ostium, coronary spasm, non-coaxial alignment of the guide, or mismatch between diameter of the guide and of the arterial lumen. When dampening of the aortic pressure is caused by a small coronary artery, the guide can be exchanged to one with side holes, which allows passive blood flow into the distal coronary artery. The drawbacks include suboptimal opacification of the artery because contrast escapes through side holes, and very rarely, decreased backup support due to weakened guide shaft and kinking of the guide at the side holes, if the guide is excessively manipulated.

However, the most common cause of ventricularization is ostial lesion. If the blood pressure is dampened – not due to LM lesion while a balloon, stent or wire is already in the coronary artery – then advancing any of these devices farther will back out the guide and restore the normal blood pressure tracing.

***Checking Stability and Potential of Backup Capability:** Under fluoroscopic guidance, forward advancement of the guide should demonstrate a tendency to further intubate the coronary artery rather than prolapse into the aoric root. If the tip slips out, it means that the guide does not provide sufficient backup. It may need to be changed for another with better support. Active intubation of the guide may be tried if its tip is soft, if the artery is large enough to accommodate the guide and there is no ostial or proximal lesions. This active support position is needed temporarily in order to advance the device across the lesion [7]. Once the device is positioned, the guide is then withdrawn to outside or stays at the ostium.

****Simple Coaxial Position or Active Support Position?** Coaxial guide alignment with the ostium is more important than an active support or "power position" because it allows the operator to gently advance and retract the guide as needed, ensuring proper stent position and contrast opacification. Because almost all interventional devices (stent, cutting balloon, directional, rotational ablative, thrombectomy or distal protection devices etc.) are rigid and of large profile, a non-coaxial alignment of the guide may lead to injury, endothelial denudation causing thrombus or dissection of the ostium of the coronary vessel. Aggressive guide intubation may prevent stent deployment at an aorto-ostial lesion [1].

JUDKINS GUIDE

A Judkins guide is selected according to the width of the ascending aorta, the location of the ostia to be cannulated, and the orientation of the coronary artery segment proximal to the target lesion. The segment between the primary and secondary curve of the Judkins left guide should fit the width of ascending aorta: 3.5 cm, 4 cm, 4.5 cm, 5 cm, 6 cm, etc. The locations of the ostia can be low, high or more anteriorly or posteriorly oriented. The ostial or proximal segment can be pointed upwards, downwards or horizontally. For the average American patients, a 4 cm Judkins left guide is often adequate. For Asian patients, a 3.5 cm Judkins left guide usually fits well. In patients with a very superior direction of the LAD or in those with narrow aortic root, a smaller size guide with a tip more anteriorly pointed will provide a coaxial position of the tip. In patients with horizontal or wide aortic root (e.g., chronic aortic insufficiency or uncontrolled high blood pressure), a Judkins left guide with long secondary curve (size 5 or 6) will fit well the width of the ascending aorta. Once in the left coronary sinus, gentle counter-clockwise rotation of the guide will frequently direct it anteriorly and enter it into the LM.

TECHNICAL TIPS

****Non-coaxial Position of a Small Judkins Guide:** If a small Judkins left guide is chosen, with its tip not coaxial to the LM, that tip will point superiorly to the wall. In that position, even though there is no dampening of aortic pressure, an injection of contrast agent in young patients may not cause dissection, but in elderly patients with many unsuspected plaques, it can cause a localized dissection [8].

***Guide That is Too Large:** The left Judkins tip points in a cranial direction, depending on the length between the primary and secondary curves and how far the heel or secondary curve is advanced into the aortic root. As a guide is advanced down the aortic sinuses, if its tip remains in the vertical axis of the ascending aorta and does not curve upward to reach the left ostium, then this catheter is too large. It should be changed for a smaller one [9].

***Guide That is Too Small:** If the Judkins left guide is smaller than needed, or the distance between the primary and secondary curves is too short, the guide would be advanced too far into the aortic root. It would double back on itself inside the sinuses of Valsava [9].

***Engagement of a Judkins Right Guide:** The basic maneuver for cannulation of the RCA is by advancing the guide into the aortic root, then rotating the shaft clockwise while gently withdrawing it, so its tip can select the RCA ostium.

When the RCA arises more anteriorly or above the right cusp, the tip of the Judkins right guide will not stay coaxial inside the right ostium. The coaxial position can best be appreciated by viewing the tip of the guide as a ring in a head-on position with the RAO 30 view (see Figure 2-4a).

AMPLATZ GUIDE

Selection of the proper size for an Amplatz guide is essential. Size 1 is for the smallest aortic root, size 2 for normal, and size 3 for large roots. Attempts to force engagement of a preformed Amplatz guide that does not conform to a particular aorta, aortic root, or aortic sinus only waste time and increase risk of complication [10]. If the tip does not reach the ostium and keep lying below it, the guide is too small. If the tip lies above the ostium, or the loop cannot be opened, the guide is too large. When the right coronary ostium is very high, then the left Amplatz guide may be used to engage the right ostium. For arteries that lie in the mid-portion of the right sinus or lower, a right Amplatz with a much smaller hook should be used.

The guide is advanced into the ascending aorta behind the long soft distal segment of the wire, with the tip pointed toward the patient's left until the guide lies on the posterior or noncoronary sinus. After being flushed well, the guide is then advanced slowly with the tip pointing upward and anteriorly. The guide should be torqued counterclockwise in order to point the tip more anteriorly while being pushed in order to point more superiorly. The tip is rotated, and retracted until it engages the LM ostium.

TECHNICAL TIPS

****Optimal Position of an Amplatz Guide:** Once the tip of the Amplatz is inside an LM or RCA ostium, the primary and secondary curves of the guide should form a closed loop with the tip coaxial to the ostial segment. This is the appropriate guide position. If the guide is pulled back, its tip could dip farther into the LM and increases the risk of LM dissection. Under fluoroscopy, while the guide is in a relaxed mode, the undesired position is a more open loop with the tip pointing down the inferior wall of the ostial segment [11].

***Withdrawal of an Amplatz Guide:** Amplatz catheters must be carefully disengaged from the coronary artery. A simple withdrawal from the vessel in a manner similar to Judkins catheter can cause the tip to advance farther into the vessel and cause dissection. To disengage

the Amplatz catheter, first advance the guide slightly under fluoroscopy to prolapse the tip out of the ostium, then rotate the guide so its tip is totally out of the ostium before withdrawing it [9].

****Withdrawal of an Amplatz Guide After Balloon Inflation:** After angioplasty or deployment of a stent, the balloon is deflated. If this latter is pulled out, the tip of the Amplatz (or any) guide would have the tendency to be sucked in deeper. This is a situation to avoid. The first best technique is to pull the balloon out while simultaneously pushing the guide in to prolapse the guide out. The procedure has to be done under fluoroscopy to monitor the calculated movement of the guide tip.

If the above maneuver fails, then the second technique can be used. The deflated balloon should be advanced slowly to back out of the guide. As the guide stops backing out, then the guide is withdrawn slowly, while watching the tip in order to avoid scratching the inferior aspect of the ostial segment. Once the tip is sensed to point unsafely down the ostial segment, then the balloon is advanced again to lift the tip and back out of the guide farther. This maneuver is repeated until the tip of the guide is totally out of the ostium. Then the guide and the interventional device can be retracted as needed. The tip is less likely to cause damage if retracted over the wire or the shaft of a device catheter.

MULTIPURPOSE GUIDE

Most operators starts by placing the tip of the multipurpose (MP) guide at the posterior sinus or noncoronary cusp in the 30° RAO position. The guide is advanced with the tip pointed toward the spine until the guide begins to buckle. When a loop is formed, slight clockwise rotation flips the tip to the left cusp. Withdrawal of the guide at the moment of that flip is usually required to maintain the correct position. If the tip does not enter the left coronary artery directly, advancing the tip by gentle counter-clockwise rotation will cannulate the LM ostium. The RCA is approached in the 45° LAO position. From the left cusp, the tip is directed anteriorly and to the patient's right. Then the guide is rotated clockwise, and then slightly withdrawn to engage the right ostium [10].

EXTRA-BACK-UP GUIDE

Most operators advocate the advancement of the tip of the guide with a wire protruding into the ascending aorta, at the aortic valve sinus, below the coronary ostium. Then the wire is removed. The guide is then flushed, advanced or withdrawn gently while torquing clockwise to point it up then it can be torqued clockwise or counter-clockwise, like an Amplatz guide, until it enters the LM or RCA.

DIAGNOSTIC CATHETER FOR LEFT MAIN LESIONS

A significant lesion in the LM can be suspected when there is: (1) typical angina at low level of activity or exercise testing; (2) typical angina

at rest; (3) typical angina after a large meal; (4) significant diffuse ST-T segment depression at low level of exercise testing; and (5) no increase of blood pressure or decrease of blood pressure upon exercise stress testing.

TECHNICAL TIPS

***Catheter Position in Suspected LM:** Once a LM lesion is suspected, a short-tip Judkins left catheter should be chosen. The catheter is positioned below the LM ostium, beneath the cusp where an injection of 10 cc contrast may opacify the cusp and give a general assessment of the LM segment. Then the tip of the catheter is manipulated to slowly engage the LM ostium, avoiding the uncontrolled jump into the artery, due to its preshaped configuration. If there is no dampening or ventricularization of the aortic pressure, then a small amount, 2–3 cc, of contrast is injected in the AP, or shallow RAO, or shallow LAO with caudal tilt (spider view) views to detect the severity of the LM.

****How Many Views Do You Need After Detecting a LM Lesion?** Once a LM lesion is confirmed, only two more views are needed: The AP cranial and the AP caudal. These two views give the full information of the LAD and LCX needed for bypass surgery. If LM PCI is contemplated then the LAD and LCX need to be visualized more, especially the ostial segment of both branches.

****Dampening Pressure:** Dampening of the aortic pressure can be due to an LM lesion, and more frequently, due to mismatch between the large-size catheter and a small coronary ostium. Gradual repositioning and withdrawal of the catheter may eliminate pressure dampening [12]. A few senior angiographers suggest a small injection of contrast with quick removal of the tip of the catheter (hit and run) technique. It is not wise to do so because the tip of the catheter may lie under a plaque and this is the possible cause of dampening of the aortic pressure. An injection of contrast agent, even a small amount, can further lift the plaque and really cause a dissection that can become disastrous.

GUIDE FOR LAD LESIONS

In order to reach a left anterior descending artery (LAD) lesion, the guide has to engage the LM ostium. The LM is located in superior and anterior position, so any guide with a tip pointing superiorly, such as the Judkins left guide, would provide a stable and coaxial alignment. In patients with a very superior direction of the LM or in those with narrow aortic root, a smaller size guide will point the tip more anteriorly or the EBU guide would help to provide stronger backup. In the case of high coronary takeoff, a Multipurpose guide or an Amplatz would easily cannulate the LM ostium. In patients with horizontal or wide aortic root, a Judkins guide with long secondary curve (size 5 or 6), or a Left Amplatz-type guide may be needed [9].

GUIDE FOR LCX LESIONS

Cannulation of the LM for PCI in the LCX usually can be achieved with the Judkins Left guide. In the case of high coronary takeoff, use a Multipurpose or an Amplatz guide. In patients with horizontal or wide aortic root, a Judkins guide with long secondary curve (size 5 or 6) or a Left Amplatz-type guide may be needed. Because the tip of the Judkins guide points superiorly, better axial support for LCX lesions can be obtained using an Amplatz, or extra-backup guides.

TECHNICAL TIPS

***Pointing Towards the LCX:** In the case of short LM or separate ostium of the LCX, if the tip of the first Judkins guide does not point toward the LCX, slightly withdraw the guide and turn clockwise. The tip will point posteriorly, toward the LCX. If this maneuver does not achieve satisfactory results, change to a larger size or to an Amplatz-type guide with a tip pointing down. If the LM is very short, a size 1.5 Amplatz will allow acceptable cannulation without over engagement. However, watch for dissection caused by a too-engaging tip.

****Selection of Guides:** If the LM is short and there is no acute angle at the bifurcation with the LCX, a left Judkins may be the first best choice. If the LM is long and the angle between the LM and LCX is acute, an extra-backup guide should be chosen. The rationale for this choice is that the tip of the guide is very close to the ostium of the LCX so the acuity of the LM and LCX angle is nullified making smoother the transition between the LM and LCX. The second reason is the secondary curve of the guide lies on the opposite aortic wall to provide firm backup in order to cross any tight lesion.

*****Rotational Amplatz Maneuver:** To enhance the support role of a Judkins left guide (active support or "power position"), if the length of the guide is appropriate, with the tip hooked in the LM, the Judkins left guide is gently pushed down until the whole curve sits well in the left sinus while torqued gently clockwise so the tip still pointing to the LM and the whole guide simulating the position achieved by an Amplatz guide. The guide should be torqued over the shaft of an interventional device (stent, balloon, intravascular ultrasound, etc.). This is called rotational Amplatz maneuver [9]. The operator should not feel any resistance when attempting this maneuver. After the interventional device is advanced and positioned in place, then the guide is withdrawn from the artery by reverse the earlier torquing energy: gentle clockwise rotation so the guide can untwist itself while pulling the guide back slowly. This technique should be performed with soft-tip guide in coronary artery large enough to accommodate the guide. There should not be disease at the ostium or proximal segment. However an alternative way is to exchange the Judkins left guide for an Amplatz or extra-backup guide that can provide stronger (passive) support, less friction at the LM–LCX bifurcation and safer advancement

of interventional hardware without excessive manipulation resulting in unintended or unexpected complications.

GUIDE FOR RCA LESIONS

The RCA usually arises anterolaterally from the right coronary cusp. In the large majority of cases, its proximal segment has a horizontal configuration and forms a 90° angle with lateral border of the aorta. In the case of an acutely angled takeoff, the "shepherd's crook", the angle is smaller than 90°. When the RCA is directed caudally, the downward angle is more than 90°. However, there are other minor variations, including the slightly anterior or posterior placed ostium or the one with anomalous origins that can make cannulation or alignment of guides difficult [13].

TECHNICAL TIPS

****Selection of Guides for Horizontal Takeoff Angle:** In the majority of cases of RCA with horizontal takeoff, a Judkins right 4 guide can easily engage the ostium. When a Judkins right fails to cannulate the right ostium, a right Amplatz would be the next option. If it fails, a left Amplatz with backup from the opposite wall of the aorta will usually achieve cannulation of the ostium and provide the required backup [13].

****Selection of Guides for Superiorly Oriented Takeoff Angle:** When the shepherd's crook or a markedly superior orientation of the RCA is encountered, guides with the tip pointing in a cranial direction are necessary. The Judkins right guide, which is effective in diagnostic angiography, does not provide sufficient backup; therefore the Amplatz left guide is usually selected. Other guides with a superiorly directed tip, such as the hockey stick, the left venous bypass, and the internal mammary artery guides, can cannulate the vessel, although they offer poor backup support. These preshaped guides may eliminate the need of extensive torquing and are particularly useful in elderly patients or in patients with very tortuous iliac arteries, which make some times guide manipulation very difficult [13].

****Selection of Guides for Inferiorly Oriented Takeoff Angle:** In this orientation of the proximal segment of the RCA, aggressive engagement of the tip from a regular JR tip can abut the lateral wall and cause dissection. The guides with inferiorly directed tips, such as the right venous bypass, Multipurpose, and Amplatz guides, may achieve more effective coaxial alignment with the proximal vessel segment [13].

****Avoiding Selective Entry of the Conus Branch:** If the guide keeps entering the conus artery, do one of two things: (1) change the guide for a larger one; or (2) approach the RCA from a posterior direction – position the guide above the sinus, rotate the guide counterclockwise to enter the main RCA first [14].

****Deep-Seating a RCA guide:** In a non-coaxial situation, backup support will not be adequate for advancement of interventional devices. Thus, the guide should be better aligned by additional clockwise rotation to allow the tip to engage deeper into the ostium. This maneuver is performed in the LAO view. When the interventional device is advanced into the coronary artery by the right hand, additional pressure should be put on the guide by the left hand placed firmly on the patient's thigh near the femoral sheath so the guide does not back out. While the device catheter is advanced, the assistant should pull the wire back slowly to decrease friction inside the device catheter, thus facilitating its advancement. If the guide needs to be deep-seated then it is advanced over an interventional device (stent, balloon catheter, etc.) while applying clockwise torque. Once the guide is deep-seated, the interventional device is advanced and positioned. After achieving the position needed, the guide is withdrawn with gentle counter-clockwise rotation to outside the coronary ostium. This procedure should only be attempted if the artery is large enough to accommodate the guide, if there is no ostial or proximal lesion, and the guide tip is soft.

****Rotational Amplatz Maneuver for the RCA:** To enhance the support role of a Judkins guide (active support or "power position"), the guide is torqued counter-clockwise, and simultaneously pushed down gently to make a loop in the coronary sinus in such a manner it takes a 90º bend on its shaft. The original secondary curve is hence obliterated, and in fact displaced proximally to obtain direct support from the opposite aortic valve. This maneuver is distinct from deep-seating of the guide where no support is derived from the opposite aortic wall. This can be done with small and soft guide (6Fr). If the guide is stiff, it will tend to prolapse into the ventricle with the wire pulled back into the aorta. Such catastrophe can be avoided by carefully monitoring the shape and the position of the guide as the maneuver is carried out. Having rotated the catheter in a counter-clockwise direction while advancing it, it is essential to have the distal part of the guide on a plane parallel to the aortic valve. If the catheter moves downward, toward the aortic valve, further advancement will result in prolapse of the guide into the ventricle. At that point, the guide is to be gently pulled back and rotated further counter-clockwise prior to its advancement. If prolapse tends to recur, this maneuver should be abandoned [15]. It is also very important to avoid excessive rotation that may lead to kinking of the guide and impede and/or dislodge stent passage. This maneuver is also not useful when a Judkins right cannot engage a RCA because of ostial lesion. However, an Amplatz or EBU guide could provide the same support without excessive manipulation of the guide and unexpected complications.

GUIDE FOR SAPHENOUS VEIN GRAFT

Selection and manipulation of guides for saphenous vein graft (SVG) and internal mammary grafts is discussed in detail in Chapter 13.

GUIDE FOR AORTIC ANEURYSM AND DISSECTIONS

When performing procedures in patients with aneurysm in the ascending aorta, the technical problems could be loss of catheter control or inadequate catheter length to reach the coronary arteries. In the case of aortic dissection, the arterial entry route chosen may not allow access to the true aortic lumen. Then other risks include extending a dissection plane by advancement of the guide or wire, perforation of the aorta by manipulation or injection in a false lumen, or displacement of thrombotic material from an aneurysm [16]. For these reasons, a careful discussion of the goals of angiography should be carried out with the surgeon. The role of aortic angiography is to visualize the origin and flow into the coronary arteries. Many surgeons do not require extensive angiography when multi-slice computed tomography (MSCT) or magnetic resonance angiography (MRA) can confirm BETTER the size and the extent of the dissection in the aortic arch, and the need for angiography can be avoided.

TECHNICAL TIPS

****Simple Maneuver to Determine Supra-Aortic Vessel Involvement:** When planning to perform angiography for a patient with suspected ascending aortic dissection, the difference in blood pressure between the two arms will pinpoint the involvement of the supra-aortic arch vessel. If the blood pressure in the right arm is higher than the one in the left arm, then there is possibility of the dissection involving the left subclavian artery without involving (or distal to) the innominate artery, so the right radial or brachial approach is preferable.

****Which Approach for Catheterization?** In cases of aneurysm or dissection limited to the thoracoabdominal aorta, the radial or brachial approach is preferred. When the CT scan shows involvement of the great vessels or carotids, the radial or brachial approach should be avoided. When there is involvement of lower extremities, access from the involved limb is avoided. When extensive ascending and thoraco–abdominal aneurysmal disease is present, the femoral approach is chosen because of greater ease of catheter exchange and manipulation [17].

****Is the Catheter in the True Lumen?** In patients with aortic dissection requiring ascending aortography, at first an attempt is made to enter the left ventricle directly with a pigtail catheter. After pressure measurement is made, the catheter is pulled back and the aortography is performed. In this way, one can be assured of being in the true aortic lumen. It is risky to attempt to cross the aortic valve against resistance. A straight-type catheter with a blunt tip like the Sones or the Multipurpose should be used cautiously in known or suspected aortic dissection due to the possibility of advancing it into the false lumen [17]. Since the majority of the dissection occurs in the lateral wall of the aorta, the pigtail can be positioned in the true lumen

by advancing while hugging the medial aspect of the aortic arch in a shallow (anterior-posterior) AP view. In the true lumen, selective cannulation of the coronary artery is possible as is direct entry into the LV.

****Ascending Aortogram:** An ascending aortogram in the left anterior oblique (LAO) projection is obtained with 60 cc of contrast with a flow rate 25–40 cc per second. The aortogram is frequently helpful in defining the shape and size of the aorta, showing the position and orientation of the coronary ostia, and in choosing appropriate coronary catheters. Injection is never made if the aortic pressure is damped or there is no brisk blood return through the catheter. If the test injection showed delayed washout or swirling of contrast, it is assumed that the catheter is in the false lumen. It is withdrawn and redirected into the true lumen with a 0.035" high torque floppy wire [17].

****Engagement of the Coronary Catheter:** When the aortic root is horizontal, the Judkins left 6 guide is often successful in cannulating the LM. Most often it has to be "pulled into" the LM by a combination of advancement of the catheter below the ostium with simultaneous retraction of the wire which is curled up into the left sinus. Due to frequent prolapse of the catheter, this maneuver often needs to be repeated many times before successful engagement is achieved. Thus a 0.038" wire is inserted through the Y adapter and left ready in the guide, so the above maneuver can be repeated quickly if needed.

When the aortic root is vertical, the Amplatz left 4 is more frequently successful in engaging the LM. The guide is engaged by curling the wire well up the left sinus and tracking the guide up just below the LM. The wire is then retracted and the guide is gently advanced up into the LM [17].

The engagement of the RCA is frequently problematic because its origin is often distorted. Usually it is displaced low in the floor of the right sinus of Valsalva (particularly in a horizontal root) but its origin may occasionally be abnormally high. In many cases, the dissecting plane begins above the RCA. Therefore, the aortic diameter may be normal at the level of the RCA which is usually easily engaged with the standard Judkins right 4 or 5 catheters [17].

In contrast to the patient with aneurysm, the aorta diameter in dissection may be narrower due to systolic compression of the true lumen by the hematoma. One particular problem is a lack of support from the dissecting aortic wall to the Judkins catheter. The Amplatz guides require the support from the aortic valve cups for manipulation, and due to the weakening of the aortic apparatus by the dissection, the guides are more difficult to use, prolapsing frequently into the left ventricle, during attempted engagement [17].

GUIDES FOR CORONARY ANOMALIES

Regardless of the rarity, an experienced interventionalist should be aware of all variations of coronary anomalies and systematically search in other aortic sinuses when the vessel in question does not arise

from its usual location [18]. For PCI, the location of the ostium of the anomalous artery and the geometry of the proximal segment should be the prime determinants dictating selection of a specific guide [18]. An RCA with a long horizontal segment in the LAO view may appear to have an angle of proximal vessel orientation favorable for use of a Judkins curve, but the long segment usually represents an ectopic origin, appreciated more readily in the RAO projection. Coaxial engagement of these arteries may be more difficult and require considerable manipulations. To cannulate the anomalous artery from the right sinus, the best guides are the left, right Amplatz and the Multipurpose guides. For the artery originating from the left sinus, the best guides are the larger left Judkins, left Amplatz, and the Multipurpose guides [19]. In some very unusual anomalies, "trial and error" guides selection or reshaping guides may be necessary [19]. Approach from the radial artery may offer a better chance of success.

CAVEAT

Anatomic Anomalies at the Ostial Segment: Not every anomaly has a wide ostium that the tip of the guide can hook onto, or a narrowing at the opening that needs to be stented. An anomalous RCA from the left sinus or a LM from the right sinus can leave the aorta in oblique fashion, so the ostium has a slit-like configuration formed by flaps of aortic and coronary tissues. During exercise, the aorta can expand its part of the flap, narrowing farther the slit-like opening and causing ischemia [20].

TECHNICAL TIPS

***Guides for Right Aortic Arch:** In patients with right aortic arch, dextrocardia, or corrected transposition of situs inversus, a left coronary catheter may be used to cannulate any artery originating from the right aortic sinus. A right coronary catheter will be used for an artery originating from the left sinus. The catheter is torqued in a counter-clockwise fashion rather than the usual clockwise one, and is based on mirror-image angles [21].

***Guides for Anomalous Coronary Arteries Arising Above the Sinotubular Ridge in the Ascending Aorta:** Patients can have coronary arteries arising above the sinotubular ridge. In this situation, the best guide would be an Amplatz-type guide. A Multipurpose guide could help if the ostium is not situated too high [22,23].

***Guides for Anomalous Coronary Arteries Arising from the Left Sinus:** When the RCA arises from the left cusp, usually it is anterior and cephalad to the LM, so in principle, it can

also be cannulated by a Judkins left with the secondary curve one size larger than the one used for the patient's LM. This larger Judkins should be pushed deep in the left sinus of Valsava, causing it to make an anterior and cephalad pointing U-turn. The larger curve will prevent the guide to engage the patient's LM [24]. In the same principle, a left Amplatz 2 with a tip pointed more anteriorly, would help to cannulate the artery, by being torqued counterclockwise while being pushed gently [20]. Others reported the use of a Judkins left 4 with an eccentric tip to cannulate the anomalous RCA from the left sinus. The primary curve of the type G catheter is out of plane to the remainder of the catheter in an anterior orientation, therefore avoiding the normal left coronary ostium [25].

*****Guides for Anomalous Coronary Arteries Arising from the Right Sinus:** This artery usually arises from the very proximal RCA or from a separate orifice in the right cusp. An Amplatz left guide is well suited for cannulating this vessel and will do so selectively rather than entering the RCA. Others have suggested a Judkins right with a posteriorly directed tip or a Judkins right with its tip shortened by 2 mm and smoothed with a sterilized emery board [25]. When the Judkins right could not provide a stable platform to advance the hardware, then an Amplatz right can cannulate the ostium easily [25].

When the origin of the artery is in the base of the aortic sinus, then deep engagement of the guide is key. Contralateral wall support is not always feasible due to the downward orientation of the ostium. A multipurpose guide should be used in this situation. Alternatives to this guide could be the Amplatz left 1 or 0.75 and maybe the Amplatz right 1 or 2 [26]. In case the anomalous RCA ostium is anterior and superior in location, the standard size left Judkins catheter may be most suitable to engage the ostium, especially in patients with small diameter aortic roots. If the Amplatz guides fail, early consideration should be made for using standard 7Fr left Judkins guides positioned with clockwise torque in order to provide a stable platform for intervening upon complex lesions of an anomalous RCA with high anterior takeoff [27].

*****Guide for Coronary Arteries Arising from the Posterior Sinus:** The most common anomaly is an anomalous LCX originated from the right sinus. A Judkins right 4 can cannulate the artery by torquing the guide clockwise so its tip will point more posteriorly. A left Amplatz guide can be cannulated too and its tip should be oriented posteriorly by torquing clockwise in order to successfully engage the ostium of the anomalous LCX [28].

****Guide for Missing Arteries:** When there is missing LCX or RCA, possible abnormal locations are suggested. Then a few guidelines with guide selection may help to pinpoint where the missing arteries can be and their ostia to be engaged (Table 3-2).

Table 3-2 Guides for Missing Arteries

Missing arteries	Guide selection
Missing LCX due to very short LM	Use large guide with short tip and turn clockwise to point the tip more posteriorly
Missing LCX originated from RCA	Guide with short tip
No RCA	In right sinus: Amplatz left pointing antero-superior to the RCA ostium
No RCA	In left sinus, Judkins left one side larger, pointing antero-superior to the LM ostium
No LM	In right sinus: Amplatz left pointing antero-superior to the RCA ostium

TAKE HOME MESSAGE

Guides for Coronary Anomalies: Coronary arteries arising from the left sinus of Valsalva, be they normal or anomalous, are likely to be successfully cannulated with a left Judkins, a left Amplatz, or a left EBU. Like normal arteries arising from the right sinus of Valsalva, anomalous arteries arising there are likely to be cannulated without difficulty using a right Judkins, a right or left Amplatz, or a multipurpose catheter. Although a right Judkins guide has been used successfully in a patient with an anomalous left circumflex arising from the right sinus of Valsalva, because of the inferior take-off of the artery a multipurpose, a right Amplatz, or a right bypass graft guide might be a better choice [29].

MANIPULATION OF THE GUIDE DURING PCI

TECHNICAL TIPS

****Stabilizing a Guide with the "Buddy" Wire Technique:** When working with an unstable guide, after unsuccessful advancement of interventional hardware, a second angioplasty wire can be advanced parallel to the first one. It straightens the tortuous vessel and provides better support for device tracking.

A second wire in a side branch can be very useful in "anchoring" the guide (e.g., second wire in LCX when dilating LAD lesion). This provides for better "backup" and allows retraction of the guide when necessary, without loss of position. It also prevents the guide from being "sucked in" beyond the LM when pulling back high profile, poorly rewrapped balloon catheters following stent deployments or post-stent dilations. However, a second wire in a non-diseased branch would cause unnecessary denudation of endothelium in that vessel. If one extra wire does not help then maybe two or three buddy wires may help to advance any devices.

****Stabilizing a Guide with Anchoring Balloon:** When working with an unstable guide, a second small balloon (1.5–2 mm diameter) can be inserted in a small proximal branch and inflated at 2 ATM, in order to anchor the guide (without letting the guide back out).

****Stabilizing a Guide With a Long Sheath:** When working with an unstable guide, a long sheath can stiffen and support the guide, depending on how close it is to the tip of the guide. The closer it is, the more supportive the system becomes. At first, the sheath tip is positioned high in the ascending aorta. If further back-up is required, then the sheath can be advanced further. As the sheath advances over the guide, it straightens the secondary and tertiary curves of the latter causing the tip of the guide to move forward. Therefore, the guides with relatively simple curves (Amplatz, Multipurpose, EBU) are probably safer and better suited for this technique. In order to avoid proximal coronary dissection, instead of fixing the guide in place as the sheath is advanced, a gentle reverse traction on it is advised, so that the guide tip does not move forward. The operator should watch the guide tip continuously on fluoroscopy during this maneuver and should ensure coaxiality of the guide in two orthogonal views. Also, disengaging the guide from the coronary ostium should be performed only after the sheath is retrieved away from it, probably to the descending aorta, with the guide fixed in place. After PCI, it is possible that by just pulling the sheath away from the ostium, the guide will disengage because of reconstitution of its curves. A larger diameter sheath can be used if even more support is deemed necessary. The sheath size can be selected depending on the amount of support needed (larger Fr size will give more support) and the height of the patient (taller patients will require longer sheaths) [30].

****Strengthening the Guide with Another Guide or Catheter:** In a case report by Saito *et al.* the 5Fr Heartrail straight guide is 120 cm in length, whereas the 6Fr guide is 100 cm. The 5Fr Heartrail guide has a very soft 13 cm end portion. This soft end portion can easily negotiate the tortuous coronary artery with the minimal damage and then it can be inserted more deeply into the artery. The inner lumen of the 5Fr Heartrail catheter is 0.059″ in diameter; it can accept normal balloons or stent delivery systems less than 4.0 mm in diameter. The inner lumen of the outer 6Fr catheter needs to be more than 0.071″ in diameter to accommodate the 5Fr Heartrail catheter; Launcher (Medtronic), Heartrail, and Radiguide (Terumo) guides have this large inner lumen diameter. When a lesion could not be crossed by a balloon or a stent in the regular 6Fr system, the five-in-six system could be tried. First, the balloon or the stent is removed from the 6Fr guide, while the wire and the 6Fr guide remained in place. Next, a 5Fr guide is inserted over the wire inside the 6Fr guide. At this point, the 5Fr guide should not protrude out of the tip of the 6Fr guide. Finally, the Y-connector is connected to the 5Fr guide and PCI could be restarted. Before the 5Fr guide is advanced into the target artery, a balloon catheter is advanced

near the target lesion in the artery. Keeping a slight tension on the balloon catheter, the 5Fr guide is pushed out slowly in order to avoid the possible injury to the coronary artery by the tip of the 5Fr guide [31].

TACTICAL MOVE

Which Technique is BEST in Stabilizing a Guide?

Three factors were found to be associated with increased backup of a guide. (1) The size (larger is stronger, if the same material in the construction of the shaft). (2) The angle between the wall of the ascending aorta and the segment of the guide spanning the aortic root. This segment is the long tip of the EBU or MP or Amplatz guide or the segment between the primary and secondary curve in the Judkins left. The maximal angle is 90° (perpendicular to the opposite wall of the ascending aorta). In a relaxed position, the backup force of a Judkins left is weak. However, as the guide is deep-seated, this angle changes and becomes bigger so the backup force is better. (3) The aortic wall area which the secondary curve rests on (the larger the better (up to 25 cm). Between all the three criteria, the EBU guide fares the best . The Amplatz design shows a very long line resting on the opposite wall of the ascending aorta and this is the mechanism of strong back-up of the Amplatz left [32] (Figure 7-9). In summary, the guide size, the angle between the guide and the ascending aorta, as well as the contact area with the ascending aortic wall with the guide have been found to be associated with increased back-up force.

In order to advance interventional devices to the intended position, a guide needs to give enough back-up support. If it fails, other techniques to stabilize the guide are discussed below.

Advancing an extra wire along with the first wire is to straighten the guide, straighten the artery, modify the contact surface of the wire to the arterial wall (wire bias) and hopefully the interventional device can be advanced further. This mechanism is further reinforced if more than one wire is inserted, with a second wire being anchored in a different branch.

The balloon anchoring technique (inflating at low pressure a second small balloon on a proximal branch) works by preventing the guide to (slide) back out. The drawbacks are that (1) the balloon can damage the endothelium of the side branch, (2) if there is no accessible side-branch to anchor the balloon, and (3) in case of chronic total occlusion (CTO), the balloon could be anchored in a proximal branch which provide antegrade collaterals. By that, during procedure, no opacification of the distal segment can be performed.

The long sheath technique works by stiffening (armor) the guide and preventing the guide from backing out of the ostium.

Which technique will work BEST? The criteria to judge any new technique, technical tip or equipment are: it has to be (1) simple, (2) cost-effective (no need for extra-equipment), (3) if new equipment is needed, cheaper and user-friendly devices are suggested, and (4) time-effective.

TACTICAL MOVE

BEST Technique in Strengthening a Guide

1 **$ FIRST Best maneuver:** Add a second stiffer wire
2 **$ ⌛ SECOND Best maneuver:** Change to stronger guide
3 **$$ ⌛ ♦ THIRD Best maneuver:** Advance a small balloon to a side branch and inflate the balloon to prevent the guide from backing out
4 **$ ⌛** Change the current sheath to a very long sheath
5 **$ ⌛** Double guide technique: insert a smaller guide in current guide

TECHNICAL TIPS

****When Should a Guide with Sidehole be Used?** When there is ventricularization or dampening of aortic pressure, corrective measure by using guide with side holes can give false security because the tip of the guide could be located under a plaque and manipulation or injection of contrast could cause severe dissection. A guide with side holes could be used ideally in PCI of chronic total occlusions of the RCA in case of present anterograde collaterals. This generally guarantees anterograde flow even during deep guide intubation, which permits distal opacification during contrast injections but avoids possible ischemia [33].

****Selection of Guides According to Inner Diameter:** It is important for the guides to have sufficient inner diameter to accommodate various single or multiple devices while allowing adequate vessel visualization. During intervention with the double balloon technique, the minimal required lumen diameter is calculated by adding 0.006″ to the combined diameter of the largest portions of the two balloon catheters. As the lumen size becomes larger due to innovations from manufacturers, the selection of any guide is up to the size of the lumen and its accommodating capacity (Table 3-3).

Table 3-3 Inner Lumen Size and Accommodating Capacity

Size (inches)	Accommodating Capacity
5F (0.058–0.059)	Balloon angioplasty
5F guide (wide lumen)	Some stent and cutting balloon if <2.5 mm
6F (0.070–0.073)	Standard angioplasty and stenting, AngioJet catheter
6F guide (wide lumen)	Some bifurcation angioplasty (including kissing balloons), IVUS catheters
7F (0.078–0.081)	2.0-mm Rotablator burrs 2 rapid-exchange balloon catheters
8F (0.080–0.090)	2 over-the-wire balloon catheters 2.25 mm Rotoblator burrs Directional coronary atherectomy
9F (0.098–0.101)	Maximum Rotoblator burr: 2.5 mm

Table 3-4 Direction of Torquing During Deep-Seating Maneuver

1	Toward the LAD	Counter-clockwise rotation
2	Toward the LCX	Clockwise rotation
3	In the RCA	Clockwise rotation

TECHNICAL TIPS

****Deep-Seating Maneuver:** To provide further support for an interventional device to cross a tight lesion, some operators suggest deeply engaging the tip of the guide into the ostium. For the RCA, the interventional device is retracted as the guide is advanced over the wire and gently rotated clockwise. For the LAD, counter-clockwise rotation while advancing the guide provides the best deep-seating. To point the guide toward the LCX, clockwise rotation is suggested (Table 3-4) [34].

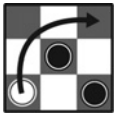
TROUBLE SHOOTING TIPS

*****Difficult Engagement of a Guide While Easy Engagement by a Diagnostic Catheter:** Sometimes a diagnostic catheter can engage an artery easily but it is very difficult with an interventional guide. After the diagnostic catheter engages the artery, a long 0.0142″ wire is advanced into the artery and then the wire is replaced with the guide. In a similar situation, when there is difficulty in deeply engaging a guide, a 0.14″ wire is advanced into the artery and as a rail for tracking the guide. The wire with gradual tip transition should be selected to avoid prolapsing at its point of transition and disengagement of the guide.

****Changing a Guide with Wire Across Lesion (Japanese Blowing Technique):** Changing a guide is more difficult if the lesion has already been crossed by a wire. If a wire placement was difficult, it is desirable not to recross the lesion. Techniques have been developed to allow exchange of guides over regular length angioplasty wires. The use of exchange wires with long radiopaque tips facilitates this procedure by minimizing the chance of failing to notice a redundant loop of radiolucent wire in the aortic root.

First withdraw the guide a few centimeters without losing position of the wire across the lesion. When the proximal end of the guide meets the proximal end of the wire, attach a syringe full of fluid into the guide. Under fluoroscope, withdraw the guide while keeping the wire immobile by continuing to inject the fluid into the guide from the syringe. By that, the guide is removed slowly [35]. Once the guide is out of the sheath, the new guide is inserted over the two wires: the angioplasty wire across the lesion and a 0.035″ wire. When the guide is at the ascending aorta level, the 0.035″ wire is removed. A balloon catheter is inserted. Once the balloon catheter is at the ostium of the coronary artery, the new guide is then advanced over the shaft of the balloon catheter which can provide a better rail for the guide than the wire alone would [36].

TACTICAL MOVE



BEST Technique for Changing a Guide With Wire Across Lesion

1 $ ⌛ **FIRST Best technique for American operators:** Change the guide through the extension of the existing angioplasty wire
2 **No extra cost FIRST Best technique for European and Asian operators:** Remove the guide while injecting contrast from a syringe and insert the new guide through a second stiff 0.035″ wire while the angioplasty wire is kept immobile across the lesion [36]. Reinsert the guide through a balloon catheter
3 $ ⌛⌛ **Third option:** Remove the whole system, change the guide, and recross the lesion

TECHNICAL TIPS

****How to Untwist a Twisted Guide:** When the iliac artery is very tortuous, torquing maneuver can twist the guide at its proximal segment. Usually the pressure curve would disappear on the screen. The patient can complain of pain in the lower quadrant area because when the guide becomes twisted, forms a sharp bend pointing laterally and can cause perforation. When seeing a twisted guide, the first thing to do is to move the twisted segment to a large area by advancing it into to the aorta rather than leave it in the iliac artery. Then cannulate the guide with a 0.035″ wire, move its tip to the twisted area. Next try to untwist the guide by torquing in the opposite

direction. If you turned the guide clockwise more in the last few minutes, then try counter-clockwise now (or vice-versa): work on a trial and error basis. However, as you torque, slowly advance the wire to secure the segment you just untwisted. If you see on fluoroscope that the guide becomes more tortuous, that means you are making the situation worse. Try the opposite direction. Advance the wire gently as you torque. In a matter of less than one or two minutes, the damaged guide should be entangled, straightened and removed.

*****If the Guide is Too Long:** In cases of interventions through long SVG or IMA grafts, care must be taken to ensure that there is sufficient guide length to reach distal sites with extra-long (145 cm) balloon catheters or short guides (80 cm), or the guide can be shortened and capped with a flared, short sheath one size smaller [37].

TECHNIQUE How To Shorten a Guide: First cut the guide with a scalpel. While the guide is still engaging the artery with a wire across the lesion, the guide should be clamped by a hemostat to prevent blood loss during the shortening procedure. Care must be taken that the scalpel does not damage the wire. Next, a standard sheath – that is 1French size smaller than the guide – is cut with 2 cm of sheath attached to the hub. The newly shortened sheath tip is then "flared" with a vessel dilator that is 1French size larger than the sheath. This is accomplished by inserting the tapered end of the dilator retrograde into the sheath tip.

Insert a dilator into the shortened sheath and then thread the sheath on the in-dwelling wire through the dilator. Remove the dilator. Finally, the flared end of the sheath stub is advanced over the cut end of the guide with a firm friction fit. The hemostatic clamp is removed and the side port is attached to the manifold. The newly assembled system is carefully aspirated through the manifold to assure there is no air trapped within it [37].

TECHNICAL TIPS

*****Guides for Unusually Wide Ascending Aorta:** In extremely enlarged aortas, due to long-standing aortic insufficiency or hypertension, the usual guides do not fit the large size to cannulate the ostium. If the guide has a Judkins shape, the primary curve is prolonged to 7, 8, or 9 cm length. For the Amplatz catheter, it is like the Amplatz left 5 or Amplatz left 6. However, the radius of the curvature is increased so the tip remains lower. For the guide with the EBU curve, the length of the distal tip is prolonged to fit the extra-wide aortic sinus [38].

TECHNIQUE Shaping the Guide: Stainless steel wires (0.035") are mounted into the desired shapes and kept in sterile conditions. 6Fr Multipurpose guides are mounted on the preformed wires and heated using an industrial warm air blower. Then the guides are immersed in cold saline and the wire removed. However, it is best accomplished with polyethylene catheters, which are quite rare today [38].

TECHNICAL TIPS

****How to Stabilize a Guide with Ostial Stented Lesion?** Often it is difficult to selectively intubate the ostium of a coronary artery with prior stenting and now having in-stent restenosis. The ostial stent could extend into the aortic sinus and has various degrees of in-stent restenosis. Not every guide could engage the ostium, so a wire could be passed into the stent via a lower stent strut and then is used as a support to lift the guide and make it more coaxial with the stent ostium. A second working wire could be advanced through the central lumen into the distal vessel. Before dilatation of the balloon, the uninflated balloon is to be advanced and retracted multiple times through the stent to confirm that it is inside the main lumen and not through side stent struts. An IVUS interrogation could help to confirm the intraluminal position of the wire before PCI. However, at first the IVUS tip should positioned not to far across the stent, because of fear of being trapped in a side strut [39].

*****How to Extend the Tip of a Guide:** In extremely enlarged aorta, the tip of a guide could be extended with the help of a catheter. First, a coronary guide with a large bend, usually the Amplatz left is advanced into the ascending aorta. A 6Fr size is sufficient since the inner lumen is 0.070″ (1.78 mm). Inside this guide, a longer (125 cm) 4Fr multipurpose diagnostic catheter (MP A1, Cordis/Johnson and Johnson) is advanced. The diagnostic catheter is longer than the guide and can serve as an extended tip to the guide. The inner catheter can be rotated independently of the guide and thus it is possible to adjust the orientation of the system and intubate the coronary arteries [31]

****Is It Safe to Use a Large Guide?** When using a large diameter guide, there is often a significant step-off/transition between the inner diameter of the guide and the outer diameter of the wire. This transition may act like a wood-plane or cheese grater damaging endothelium and collecting debris as the guide is advanced. A smaller diagnostic catheter within the guide may smooth the transition from wire/diagnostic catheter/guide for advancement (4Fr within a 6Fr Guide, or a 5Fr within a 7Fr Guide). The diagnostic catheter and wire may then be removed and the guide applied to pressurized continuous flush [31].

*****Double Guides for Complex PCI:** If there is a need for two guides, in complex PCI of vessels with large ostium, the two smaller guides can be used. The disadvantages are that there are two punctures, two sets of guides. However, with two guides, there is more room for contrast injection so the visualization of the coronary arteries during PCI can be better (rather than one large guide cramped with many devices inside). With two guides, the movements of interventional (stent, balloon), diagnostic (IVUS) or distal protective (filter) devices can be better. If the sheath can be changed to a larger one, two diagnostic catheters can be inserted into one 8Fr sheath or one

guide and one diagnostic catheter can be inserted into a 9Fr or 10Fr sheath without the need for a new puncture [31].

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Chapter 4
Wires

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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Pacing

Technique: How to pace with an angioplasty wire?

Take-home message: Complex wiring

GENERAL OVERVIEW

A wire consists of two main components: (1) a central shaft of stainless steel or nitinol, and (2) a distal flexible tip shaped as a spring coil made with platinum or tungsten. Wires with a nitinol core are kink-resistant while those with stainless steel are more susceptible to kinking.

The usual wire is the flexible tip 0.014″ diameter light weight wire, while the larger size wires (0.16–0.18″) or the much stiffer wire are used to straighten tortuous coronary segments and to provide more support for device tracking and when the support offered by a guide needs bolstering. In general, the flexible wires are less steerable and the stiffer wire offers more torque control [1]. However, since the wires are stiff, they can straighten the curved segment, change the vessel shape and cause wrinkles or pseudolesions. Removing the stiff part of the wire while leaving the radiopaque flexible end across the pseudolesions, exchanging for a more flexible wire or a flexible transit catheter or pulling the wire completely out, would abolish these pseudolesions. Other disadvantages of stiff wire include difficulty of tracking stents because of the wire bias inducing coronary spasm, even obstruction of flow induced by vessel kinking, and wire buckling due to sharp transitions.

The reasons that a wire is stiff or flexible is down to its intrinsic design and material composition. A core that extends to the distal tip provide extra support and torque transmission, however, it is stiff. The nitinol core increases wire trackability, including the ability to traverse acute artery angulations without wire prolapse. Nitinol wire might be more capable of entering a retroflexed circumflex takeoff than a stainless steel core wire. The limitation of nitinol core wire is that it tends to store, rather than to transmit and torque [2].

Sometimes, stiff wires can act as a "cheese cutter" when the wire is so straight and stiff that it cuts though the curve of a vessel. In these situations, the wire should be exchanged and use of atherectomy devices avoided because of excessive wire bias.

A wire is advanced gently and should pass smoothly through the stenosis. Withdrawal and reorientation of the wire is required when buckling occurs. It should not be forcefully jammed, because it can disrupt the plaque, cause thrombus formation and ultimately acute occlusion. Repeated rotation of 180° in clockwise and counterclockwise directions also seems to aid wire advancement and reduces subselection of unwanted small branches [1]. Never rotate the wires through 360°. This may result in entanglement with a second wire or tip fracture if it is caught into a small branch. In any case, the tip of the wire has to be placed as distally as possible, so the stiff part of the

wire is across the lesion where the stent or other interventional devices are to be tracked.

When selection or entry to a side branch is desired, it is important to make the radius of the curve on the tip of the wire match the diameter of the main vessel proximal to the origin of that branch. A double bend can be useful if there are two different angulated segments to cross [1].

Wiring the LAD: The left anterior descending artery (LAD) typically has little tortuosity; therefore, the wire transition point between the flexible tip and the more rigid body is usually not a major problem.

Wiring the LCX: For left circumflex artery (LCX) interventions, the wire needs to pass the left main (LM), turn into the LCX, then move forward to cross the lesion. Sometimes it requires a broad curve to successfully enter the LCX and a smaller curve to cross into the obtuse marginal. Wires with gradual transition from the body to the spring tip are preferred to avoid continuous prolapsing into the LAD [1].

Wiring the right coronary artery (RCA): When the origin of the right coronary artery (RCA) is relatively normal, a conventional soft wire with good steerability to avoid side branches is usually chosen first. When the RCA arises anteriorly, the wire sometimes may be required as an aid to guide placement. In this situation, wires with improved tip transition are selected to avoid prolapsing at the point of transition and disengagement of the guide [1].

ADVANCING A WIRE

In order to enter a tortuous proximal LAD, the best view is the left anterior oblique (LAO) caudal view (spider view). Once the wire is far enough in the LAD, the angle is changed to the right anterior oblique (RAO) cranial view so the wire can be moved to the mid and then the distal LAD. If the LAD is entered from the LAO cranial view, the wire should be pointed to the right of the patient and advanced. If the LAD is entered in the RAO cranial position and the wire is pointing downward, most likely, it enters the LCX. If the wire moves widely, then it may enter the ramus intermedius which buckles with contraction of the LV like the diagonals. Usually it is worthwhile to place a little extra curve at the tip of the wire because there is more angulation at the take-off of the LAD than is apparent in the RAO projection [1].

TECHNICAL TIPS

***Better Torque Control:** When a wire is more difficult to manipulate after it passes through too many curves, advancement of the balloon catheter near the wire tip will improve wire support, torque control, and steerability. Other options include use of stiffer wire or hydrophilic wires, which are very sleek and kink-resistant. However,

since they are so smooth, the operator has little tactile feedback, they can easily go subintimally or cause distal perforation if inadvertently advanced into a small and short branch. So, when manipulating a hydrophylic wire, always watch the distal tip, to avoid inadvertent migration and perforation.

****Wire Prolapsing:** When navigating a curve in order to enter an artery, (e.g. from the LM into the LCX), a floppy wire may keep prolapsing into a nonintended artery (e.g. LAD). The reason is the abrupt transition between the short tip and the main shaft. The way to resolve this is to change to a wire with a gradually tapered core so that as the tip is deeply advanced, it stabilizes the wire and the stiffer shaft can negotiate the angle better, without prolapsing into an unintended area (or LAD) [1]. Once the soft part of the tip passes the acute corner, torque the wire slowly while advancing the wire. The rotational energy will advance the wire distally.

****Enter the LCX Without Changing Wire:** When navigating the LM in order to enter a sharp bifurcation of the LCX, there are two maneuvers in order to enter the LCX:

1. Apply clockwise torque on the guide so its tip will point toward the LCX ostium, especially if the LM is short.
2. Ask the patient to take a deep breath that elongates the heart and straightens the angle between the LM and LCX. In this short window of opportunity, advance the wire into the LCX.

If these two maneuvers are unsuccessful then remove the wire. Shape the tip to conform with the entry angle of the LM and LCX.

****Advancing a Wire Through Severely Angulated Segment by Pulling it Back:** In rare instances, a wire has to enter a very severely angulated segment. The tip of the wire should be curved to form a large diameter curve. Once the tip enters the branch, the wire is withdrawn to prolapse the tip into the intended branch. Then rotate the wire toward the main lumen, clockwise if the tip was pointing towards the left of the patient, and counterclockwise if the tip was pointing towards the right of the patient. If there is enough stiff segment inside the side-branch (not just the soft tip), then the wire will advance further, without prolapsing back (Figure 4-1).

****Directing the Wire When Navigating the LAD:** In case of navigating the LAD, at first at the LAO caudal view, the wire should point to the right on the screen. The left is toward the diagonal. Once inside the proximal segment the better view is the LAO cranial view. Here the wire should move downward. Any stray to the left will point to the diagonal, and to the right will point to the septals.

****Crossing a Stent:** If a stent needs to be recrossed, the tip of the guidewire should be curved well into a wide J and the whole wire can be advanced while being rotated. This maneuver will help to avoid the inadvertent migration of the tip of the catheter under a strut, changing the direction of the whole wire to outside the stent. If there is

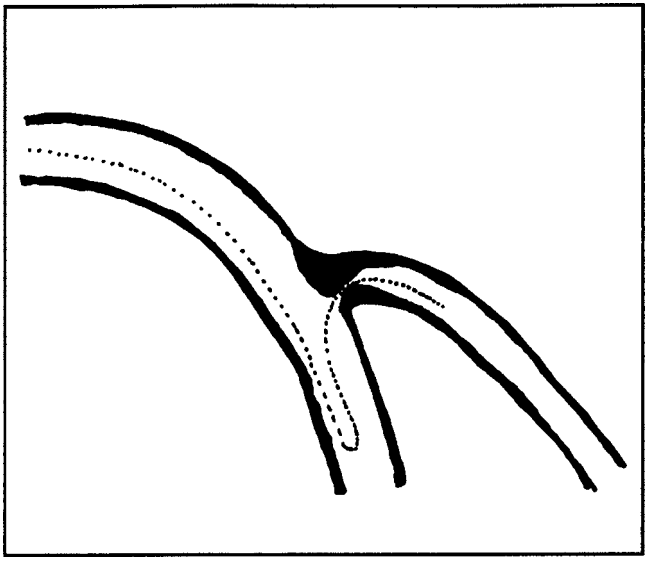

Figure 4-1 Pulling the wire to advance it. A wire is inserted and manipulated to enter an angulated tortuous branch. In order to advance the tip further, the technique is to pull the wire back to prolapse the tip deeper into the branch.

subtle resistance, wire exit through or behind the struts is suspected. If the stented area has sudden acute thrombosis and a curved tip fails to cross the stent, then an intermediate wire with a mildly bent tip can be manipulated to cross the stent. Try to have the pictures of the segments in two orthogonal views so the wire can be advanced inside the lumen as best as possible.

****Measure the Length of a Lesion with a Wire:** It is not easy to guess accurately the length of a lesion if some segments are foreshortened because of tortuosity. When a vessel bends in more than one plane, no single angiographic view can overcome multiple foreshortenings. Most radiolucent wires have a 20–30 mm radiopaque distal end. Position this radiopaque segment across the lesion so the length of the lesion can be estimated. Another way is to measure the lesion length with a balloon that has markers at its two ends.

****Manipulating the Hydrophilic Wire:** The hydrophilic wires such as the Glidewire (Meditech/Terumo Corp, Piscataway, NJ), Choice PT phus, Whisper wire etc. are kink-resistant, flexible wires covered with a hydrophilic polyurethane coating. The core is constructed with super-elastic titanium–nickel alloy that offers extreme flexibility and kink-resistance, thus optimizing pushability. A hydrophilic polymer coating results in low thrombogenicity and extreme lubricity when wet.

Because it is very slippery, it has to be handled with a plastic holding device at its proximal end to avoid slippage. Due to its alloy core

construction, its distal tip is not easily visible and it cannot be shaped, so it is more difficult to steer away from side branches [3].

****Parallel Wire Method (Seesaw Wiring) for Chronic Total Occlusion (Figures 4-2 and 4-3):** When the wire tip goes into subintimal space at the small branch or outside of the vessel, the

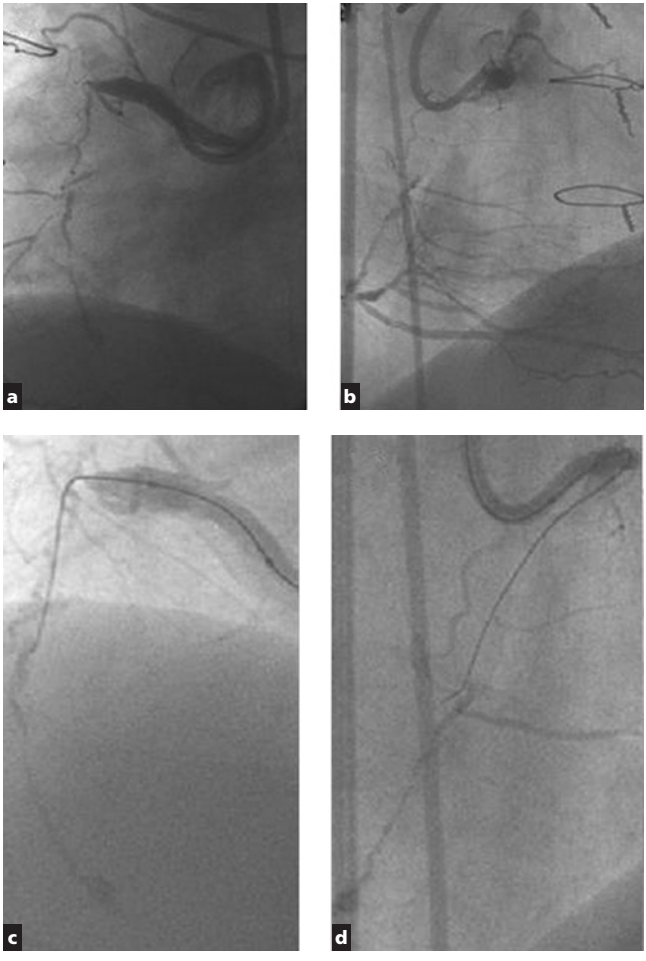

Figure 4-2 CTO lesion with bridging collateral in proximal RCA, shown in LAO (a), and LAO (b) views before intervention with seesaw wiring technique (see Figure 4-5). Parallel wire method of seesaw wiring (c) (LAO view), and (d) (RAO view) show that the wire is deviated from the true lumen; a little to the right in (c) and to the left in (d). Leaving the first wire in place as a landmark, a second wire is advanced and directed into the true lumen.

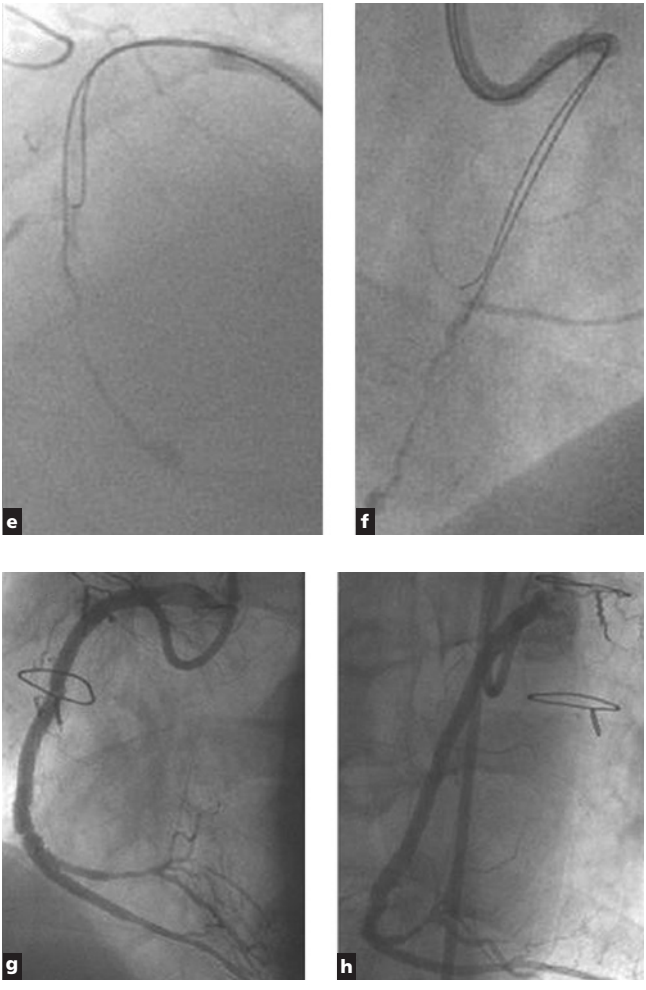

Figure 4-2 (Continued) The second wire is viewed at the left and right of the first wire, respectively in (e) (LAO view) and (f) (RAO view). (g) (LAO view) and (h) (RAO view) show post-stenting angiograms.

second wire is advanced whilst leaving the first wire in place (the parallel wire method). The first wire has two roles: the first is to obstruct the incorrect pathway; and the second is to mark the route to the true lumen during wire manipulation. With the existence of this landmark the operator can lead the wire tip more easily to the direction of the true lumen. In the parallel wire technique, if the operator intends to use only one support catheter, the support catheter should be pulled

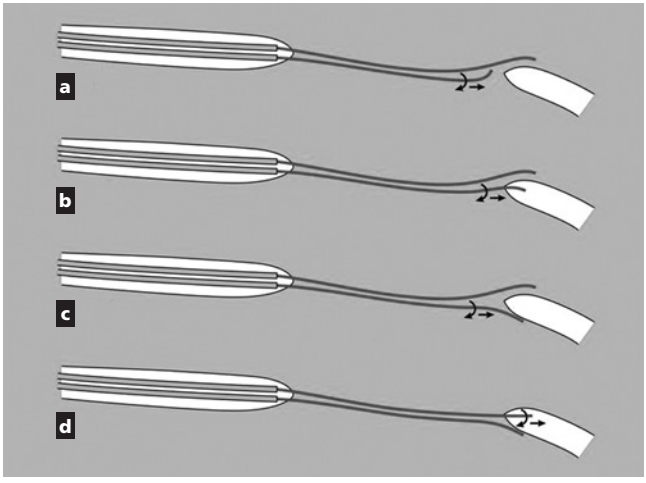

Figure 4-3 Seesaw wiring (parallel wire method with two support catheters). (a) When the first wire fails to cross into the distal true lumen, the first wire is left as a landmark, and the second wire is advanced into the occlusion site using the second support catheter. (b) The second wire is successfully manipulated into the true lumen. (c) Alternatively, the second wire fails to enter the true lumen. (d) Use of two support catheters facilitates role-exchange of the two wires. The first wire now is successfully engaged into the true lumen.

back and re-inserted into the target vessel again with the second wire. If the operator uses two support catheters at a time, the procedure becomes simpler. If it is difficult for the second wire to enter the true lumen, one can exchange the roles of two wires (Figure 4-2). Using the parallel wire method with two support catheters is called "Seesaw Wiring". The operator is able to move either of the two wires at any time. This method introduces the fluid (blood) into the waterless occlusion site, triggering the hydrophilic mechanism (slippery when wet) and thus preventing the hydrophilic wires from sticking to each other.

****Optimal projection angle:** The best projection angles differ according to the region of interest (ROI). The operator should explore the angles in which the stump can be seen most clearly, especially at the entry point. In general, it is desirable for the two projection angles be perpendicular to the vessel axis of the ROI (Figure 4-4), and also perpendicular each other (orthogonal projection). Because the summation of the blind area is smallest in the orthogonal projection (Figure 4-5), it will be easier to lead the wire tip to the true lumen. In many cases, a stiff wire with a sharp bend at the tip (e.g. the Conquest Pro or Miracle stiff wire) in a seesaw wiring technique, can be successfully steered to re-enter the true lumen (Figure 4-6).

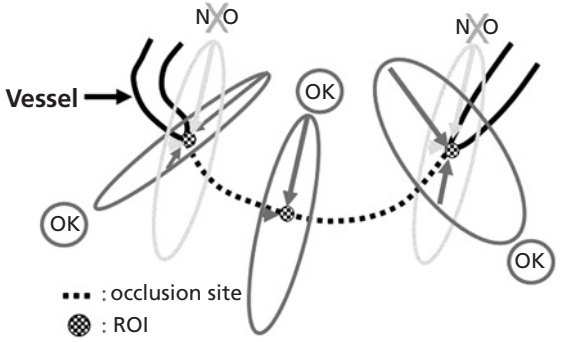

Figure 4-4 Required projection angles. Projections with perpendicular angles to the occlusion site of the vessel or guidewire tip site of the occluded part are recommended. Each ROI (region of interest) has its suitable projection angle (O). The angle optimal for the central ROI is not always suitable for the other ROI (X). For example, for viewing a proximal RCA site, the LAO/caudal or RAO/cranial is best. For the mid-RCA site, it is the straight LAO or RAO view. For the distal RCA site, the LAO/cranial or RAO/caudal projections are the most suitable.

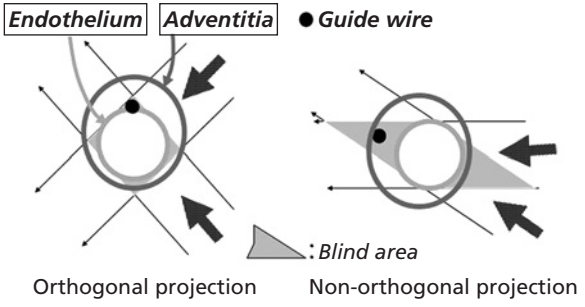

Figure 4-5 Orthogonal projections. Because the summation of the blind area, not projected by each view, is smallest in the orthogonal projections, it is very useful for leading the wire into the true lumen and to confirm successful wire entry in the true lumen.

CAVEAT

Dissection by a Wire: A wire should be advanced smoothly without any resistance and tip buckling. Otherwise, it can go through soft plaque, under the intima, and cause dissection. In case of a perfusion balloon, a wire can exit through a proximal side hole instead of the main distal lumen tip. When the perfusion catheter is deflated and readvanced with the sharp metallic wire entangled on its side, the balloon can dissect the artery. Therefore, it is important to advance the wire while the perfusion balloon remains inflated and to watch its course under fluoroscopy [4].

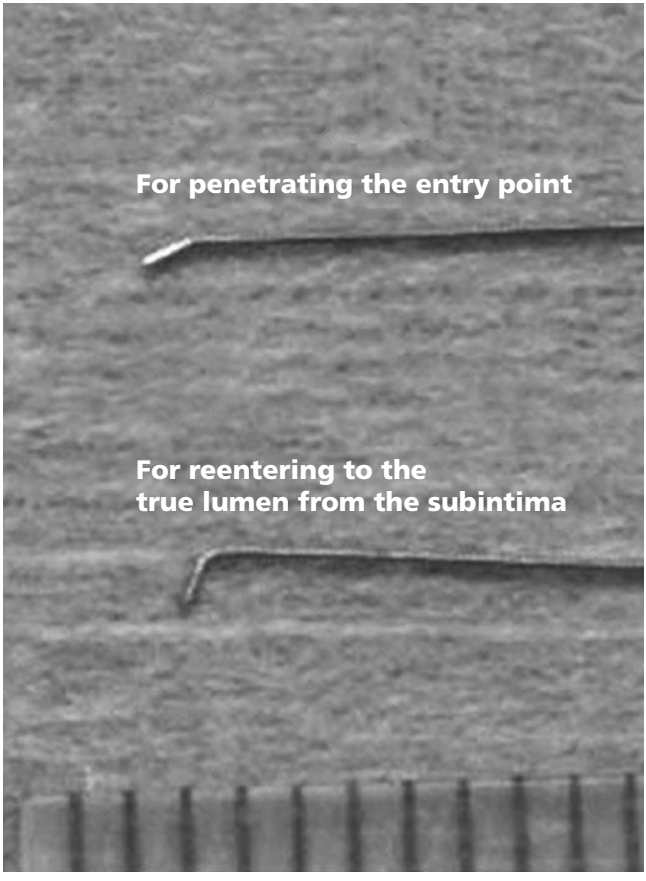

Figure 4-6 Typical tip shapes of the tapered-tip wire (Conquest Pro™ or Cross It™). For penetrating entry point, it's 1.5–2.0 mm tip is bent at a curve of about 15–30° (upper panel). For re-entering the true lumen from the subintima, an almost 90° bend is required (lower panel).

TROUBLE-SHOOTING TIPS

****Failure to Cross a Lesion:** When a wire does not seem to be able to cross a lesion, the most appropriate step is to check the wire position in a second orthogonal view. Maybe the tip of the wire is in a side branch or has migrated outside the true lumen. Once the wire is sure to be inside the true lumen, other strategies include changing to a stiffer wire, a smaller wire or a hydrophilic wire, or passing a balloon to near the tip to increase support to the wire.

****Directing the Tip of the Wire with a Transport Catheter:** Sometimes it is difficult to curve a wire into a branch of a large artery (such as from the LM to the LCX then to the obtuse marginal (OM)) because of the short stem of the second main branch. Despite persistent manipulation, the wire keeps buckling under pressure and prolapsing into the main brain (LAD). A small transport catheter may be inserted through the wire into the proximal LCX. The new catheter prevents buckling of the wire and helps the wire cross the tight lesion by shortening the distance between the tip of the catheter and the lesion, nullifying the acuteness of the angle between the LM and LCX [5].

*****Deflecting the Tip of a Wire by a Distal Inflated Balloon:** Sometimes, it is difficult to curve a wire into a sidebranch with extreme angle take-off. In a case report by Gershony *et al.*, a 74-year-old patient with unstable angina underwent a coronary angiogram, which showed severe ostial lesion of an OM. This OM originated from the LCX at an extreme angle. Multiple attempts to wire the OM were unsuccessful because of repeated prolapse of the wire in the main LCX. A small balloon was then advanced to the distal LCX and inflated with its proximal end right beyond the ostium of the diseased OM. A new wire was inserted and successfully entered the ostium of the OM, steering it towards the opening of the side branch with extreme angle take-off. In this case, the inflated balloon prevented the continuing prolapse of the wire and deflected the tip to the desired retrograde direction [6].

*****Withdrawal of an Uncoiling Ribbon of Wire:** After excessive manipulation of a wire (more than a 180º turn), its distal segment can become uncoiled. This is detected as the distal tip shows a radiolucent segment. Instead of pulling the wire in an effort to remove it from the coronary system, the best technique involves proper seating of the guide, then advancing an over-the-wire balloon or a transport catheter over the whole wire including the uncoiled segment, if it tracks easily. After the radiolucent segment is advanced over by the balloon or a transport catheter, the whole system, guide, catheter, and wire, is removed as a unit [3]. If the balloon catheter does not track easily over the floppy tip, it may dissect the artery. In this case, it may be better to simply pull the wire and all the devices as a unit.

*****Entangled Wires with the IVUS Catheter:** In complex percutaneous coronary interventions (PCI) with advanced physiologic or imaging studies by pressure flow wire (PW) and intravascular ultrasound (IVUS), there is a possibility of entanglement of the wires. In a caseload of 704 patients who had IVUS, 0.5% had entanglement with angioplasty wire, while it happened in 13% with the PW (Radi Medical System, Upsala, Sweden). Besides some minor difficulties in advancement of either wire, the problem began to unfold when the IVUS catheter was withdrawn. The predisposing factor for these entanglements was most likely due to the short monorail segment of the IVUS catheter. In order to avoid entanglement of the wires, it is wise to ensure that the

short monorail segment of the IVUS seats near the stiff segment of the PW or angioplasty wire. If entanglement is noticed, attempt to advance the PW or the angioplasty wire further in order to separate its kinked part from the corresponding tip of the IVUS catheter. Another option is to slide the IVUS catheter distally over the kinked wire. However, over-manipulation may be hazardous and predispose to generation of loops and further kinking of the PW. Removal of the complete system as single unit may be a last resort and a pragmatic option [7].

***Avoiding Entangled IVUS Catheter and Wire:** When the proximal segment to be crossed is too tortuous and calcified, the advancement of the IVUS catheter is less than smooth. If too much force is inadvertently applied to advance the IVUS catheter, displacement of the catheter away from the wire could be induced and the wire could become kinked [8]. If the problem is not recognized early, advancing a kinked wire can cause perforation or dissection. Because the IVUS catheter slides on a short monorail distal segment, so torquing movement is not transmitted well to the tip. This hampers its capacity to negotiate distal tortuous segment or cross tight curves, lesions or acute angles.

CAVEAT

Inadvertent Jailing of Wire: Do not forget to pull a wire from a sidebranch before stenting in the crush stent technique. The non-radiopaque segment is not often seen and can be forgotten during the procedure so the wire can get trapped behind the stent.

EXCHANGING CATHETERS

***Advancement of an Over-the-Wire Balloon Catheter over a Regular Length Wire:** A regular-length angioplasty wire is inserted and manipulated to cross the lesion. Be sure that the wire tip is positioned at the most distal segment of the artery. An over-the-wire balloon catheter is then advanced over the wire without manual control of the wire. This is performed with fluoroscopic guidance until the proximal end of the wire reappears through the wire port of the balloon catheter. During the passage of the balloon catheter, extreme care is taken to make the movement as smooth as possible with no tactile evidence of any hindrance. An absolute requirement is to refrain from any forceful forward motion of the balloon catheter over the wire, if any resistance is felt. This precaution would limit any forward migration of the wire. Any resistance or evidence of ventricular ectopy should prompt immediate reassessment of the wire, balloon catheter, and guide positions under fluoroscopy. There is no reported damage to the coronary artery due to inadvertent advancement of the wire. With

forward motion of the balloon catheter, increased tension in the wire can back out the guide, so the tip of the guide needs to be watched [9].

This technique is cost-effective as only one wire is used, saving the cost of an extension. It is also advantageous when the proximal end of the wire is damaged and therefore cannot be extended, or if the wire is not constructed to be extended, or there is no extension wire available.

*****Exchanging the Balloon Catheter over a Regular-Length Wire Without Sacrificing the Wire Position:** There are many ways to exchange balloon catheters without sacrificing the wire position across the lesion.

TACTICAL MOVE

BEST Options in Exchanging Balloon Catheter Over a Regular-Length Wire

1. **No Added Cost FIRST Best maneuver:** Injecting normal saline through the central lumen during withdrawal of catheter
2. **$ ⌛ SECOND Best maneuver:** Using extension wire
3. **No Added Cost THIRD Best maneuver:** Using the back end of a regular wire

TECHNIQUE Exchanging Balloon Catheter over a Regular Length Wire: The balloon catheter is pulled back over the wire until the stiff end of this wire remains just within the hub of the balloon catheter. The stiff end of a second wire is then introduced into the catheter hub in end-to-end apposition to the first wire inside the catheter. This introduction can be facilitated by the use of a 20-gauge intravenous (IV) cannula with the tip placed inside the hub. The balloon catheter is then gradually pulled back over this combination while maintaining a forward push on the second wire. Be sure that this wire is compatible with the lumen size of the balloon catheter [10].

PACING

TECHNIQUE How To Pace with an Angioplasty Wire? After cannulation of the coronary artery ostium with a guide, a 300 cm, 0.014″ angioplasty wire is advanced into the distal aspect of the coronary artery. This is connected to an external pulse generator (Medtronic 5348, Medtronic, Minneapolis, MN) using an adaptive alligator clip (Medtronic 5833SL, Medtronic, Minneapolis, MN). This pulse generator provides a variable output current while maintaining a fixed pulse width. The negative pole (cathode) is attached to the end of the angioplasty wire and the positive pole (anode) to the patient, making the angioplasty wire function as a unipolar lead. Initially, the alligator clip for the anode is attached to the skin using a large skin surface electrode. However, the required current can be

unacceptably high using the skin electrode. Therefore, to improve tissue contact, the anode is attached to a steel monofilament suture (3–0 surgical steel monofilament B&S 30) anchored in the subcutaneous tissue near the access site. This steel monofilament suture is routinely used by cardiac surgeons to ground temporary epicardial pacing wires after cardiac surgery and allows lower pacemaker capture thresholds [11].

TAKE-HOME MESSAGE

Complex Wiring

Jailing Wire in Bifurcation Stenting: In bifurcation stenting, a second wire is used for side branch protection or side branch stenting. However, it should be reminded that jailing a hydrophilic coating wire should be avoided in the bifurcation crush technique due to increased incidence of tip rupture.

Double Wiring for Advancement of the Main Wire: The plugging wire technique: occasionally, a wire keeps persistently entering an unwanted side branch. Leave that wire in the unwanted branch, so a second wire can be advanced and engage the desired artery.

When it is difficult to steer a wire into a side branch with extreme angle take-off, advance a balloon catheter and its wire in the main artery and park it right beyond the origin of the side branch. Inflate the balloon. Try to advance a second wire into the side branch. The inflated balloon would deflect the tip of the second wire to enter the desired branch.

Side branch IVUS Guide in CTO Intervention: At times, if there is a sidebranch coming out next to the proximal end of a CTO lesion, an IVUS transducer can be placed in this proximal side branch for interrogation of the CTO lesion in the main artery (detailed discussion in Chapter 10). If the wire enters a false channel, an IVUS catheter can be advanced there and interrogate the proximal end of the CTO in order to look for a favorable entry location.

Anchoring Wire Technique: When the left main artery is short, a wire is inserted in the LAD and the LCX respectively, to avoid super-selective injection of contrast in either artery. Intervention of ostial RCA or left main lesion: to control and stabilize the tip position of a guide, a second wire is placed in the conus branch or right ventricular branch in case of RCA ostial stenting, or in the circumflex artery in left main ostial stenting.

Anchoring Balloon Technique: While a second wire with a balloon is placed in a side branch or main artery, the balloon is inflated to provide better guide support so the first wire can enter the distal segment.

Parallel Wire Technique: When there is difficulty in advancing a device, a second wire (an extra-support wire) is placed in an attempt to straighten the proximal tortuous coronary segments and to provide more support for device tracking.

Railing "Buddy Wire" Technique: When it is difficult to advance a device over a wire, advancing a second hydrophilic wire along the first one may help to advance the device. This technique may be applied to retrieving an entangled ruptured balloon.

When it is difficult for a catheter balloon to cross a stent, a second wire is inserted through the stent to facilitate balloon crossing. In this case, the tip of the second wire is curved so there is no migration through the side struts.

Lesion Modification: A buddy wire is inserted alongside the pre-placed balloon, and the balloon is inflated for the purpose of lesion modification. This is similar in concept to the use of a cutting balloon, rail-balloon or angiosculptor.

Retrieval of a Dislodged Stent: When a stent happens to be dislodged in the coronary artery, the wire should not be pulled back. A second wire is inserted with its tip placed beyond the first wire. After having wrapped the first wire and the stent together by rotating the second wire or both wires, the wires and the stent are removed as a unit.

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Chapter 5
Balloon Angioplasty

Thach N. Nguyen, Thomas Ischinger, Vo Thanh Nhan, Rajiv Kumar

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complication

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GENERAL OVERVIEW

Angioplasty balloons come in different diameters and are made from diverse materials (polyethylene PE, polyethylene terephthalate PET, etc.) to give exact or oversized diameters, with various inflation pressures and a degree of balloon hardness dependent on material characteristics (compliant or non-compliant). The catheter can be advanced over a wire that passes through the lumen inside the whole length of the shaft (over-the-wire system) or only inside the distal segment (monorail system) or without an indwelling wire at all (fixed wire). A perfusion balloon has extra lumen with antegrade flow while the balloon is in inflation. A cutting balloon has micro-blades arranged lengthwise along the sides of the balloon. Once inflated, the mechanism of acute lumen gain after plain balloon angioplasty (PTCA) in calcified lesions is dissection while it is plaque compression and vessel expansion in fibrotic lesion.

Ideally, a balloon has a very low profile in its undeployed state. However, for most lesions low profile is less important than trackability and pushability. Trackability is the ease of advancing a balloon over a wire through an angulated coronary artery segment. Pushability is defined as the ability to push a balloon through tortuous segments or across a lesion. In the monorail system, smaller guides can be used with better opacification and reduced fluoro time. However the wire is unable to be exchanged or reshaped without removing it, giving up position or use of a transport catheter. The over-the-wire balloon may be more trackable and the fixed-wire balloon has a much lower profile and can be useful in tortuous arteries and extremely tight lesions. Because of the excellent result of stents to seal off dissection, the need for perfusion balloon catheters disappears, except in the case of perforation which requires prolonged inflation or in the case of poor results of initial balloon but inability to stent.

SELECTION OF BALLOON

The choice of the exact balloon and wire system is less important than the operator's overall approach to the technique of dilation, the familiarity with the system chosen, the balloon size, and the capacity to treat possible complications [1]. The ratio of the balloon:artery should be approximately 1 : 1. A higher balloon:artery ratio has been shown to be associated with dissection and acute closure.

TECHNICAL TIPS

****Length of the Balloon:** In a long lesion with diffuse disease, long balloons can distribute inflation pressure more evenly across the

diseased segment, without the inconvenience of multiple short and overlapped inflations, or the danger of untreated plaques protruding into the lumen. The probability of dissection with a long balloon is thought by some operators to be lower because a short balloon can partially disrupt an atherosclerotic plaque and allow the blood to enter the channel created behind the newly ruptured plaque. The longer balloon also exerts less straightening force on the vessel, tending to maintain the curved configuration during balloon inflation [2]. When dilating a long lesion in an artery that tapers, the disadvantage of a long balloon is that it may underdilate the proximal segment and overdilate the distal segment.

****Compliant Balloons:** Compliant balloons are used routinely because of their low profile and good "re-wrapping" after deflation. They are also useful when vessel size is uncertain, such as an undersized vessel due to chronic low flow or total occlusion. Once the lesion is open and nitroglycerin is given, the vessel may show a larger lumen; then a second dilation at higher pressure further expands a compliant balloon (20% bigger in size). Similarly, when multiple lesions are targeted in different sized vessels, use of a compliant balloon may be more cost-effective. At first, the balloon is to dilate a lesion in a small diameter vessel, then it is moved to a larger vessel where the balloon can be inflated with higher pressure in order to achieve a higher diameter. Compliant balloon is made of polyolefin copolymer (POC).

****Noncompliant Balloons:** Noncompliant balloons are made with polyethylene terephtalate (PET) and are needed in different situations. When post-dilating a stent, a short noncompliant balloon may be needed to maximize the stent size without overstretching the vessel at the stent edges. In a long lesion, a non-compliant balloon will not overstretch the distal segment, which is frequently smaller than the proximal segment. In hard and calcified lesions, compliant balloons may preferentially overdilate the soft segments while being unable to break the plaque in the hard and calcified segment. The artery still looks bigger, thanks to the overstretched normal segment, which may recoil with time. In the same lesion, a non-compliant balloon would concentrate its dilating force more directly on the hard and calcified segment without stretching the adjacent normal segment. With high pressure, it would break the plaque and shift the atherosclerotic burden equally along the longitudinal axis, and reconstruct a bigger and more stable lumen. The semi-compliant balloons are made with polyether blockamide (PEBAX), nylon or polyurethane elastomer.

****Where Should the Marker Be?** A balloon with a marker in the middle should be chosen if the lesion is very tight. When the operator sees the marker at the middle of the lesion, then inflation can be started. When doing percutaneous coronary interventions (PCI) for a long lesion, then a balloon with markers at two ends will be needed in order to be sure that the whole length of the lesion is covered.

ADVANCEMENT, POSITIONING AND INFLATION OF BALLOON

The left hand of the operator may advance the balloon while the right hand or another operator keeps a gentle traction on the wire. The balloon should be advanced by constant pressure rather than by jerky movements. While the balloon is advanced, watch carefully the tip of the guide. As the balloon is being pushed harder, an unstable guide may back out and even become completely disengaged forfeiting the wire position. If resistance is encountered at the lesion, gentle forward pressure on the balloon catheter, while pulling back on the wire and deeply but gently seating the guide, will often cause the balloon to cross.

TECHNICAL TIPS

****Speed of Inflation Pressure:** Most operators inflate the balloon slowly until its waist disappears. This gradual inflation will slowly rearrange the atheromatous material in the plaque along the longitudinal and radial axis. By that, the vessel is being stretched and deformed in a more predictable fashion than with sudden inflations that are more likely to cause extensive tearing (dissection) of the vessel wall. No studies however have defined an optimal inflation strategy.

****Checking the Appropriate Size of the Balloon or Stent:** After successful inflation of the balloon, 5 seconds before deflating it, a small injection of contrast will verify the correct fitting of the balloon with the proximal segment of the dilated lesion. If contrast agent is seen flowing around the proximal segment of the inflated balloon, the balloon is too small for the artery. This is only a rough assessment for the appropriate size of a balloon or stent, when intravascular ultrasound (IVUS) is not available. If the balloon is inflated in a proximal segment without a side branch, there may not be enough flow to opacify the artery, so in this situation it is not possible to evaluate the balloon size with the above technique (Figure 5-1).

****Looking for Collaterals:** Collaterals seen during the diagnostic study or appearing with a contrast medium during balloon occlusion, project a low-risk profile for the patient and reduce the stress for the operator. They can be looked for by injecting contrast while the balloon is about to be inflated. If the contrast caught in the distal vasculature the moment the balloon is fully inflated is washed out promptly, good collaterals are documented. If it stays put, the territory is devoid of collateral blood flow in the event of a later vessel occlusion [3].

TROUBLE-SHOOTING TIPS

****Positioning a Balloon When the Blood Flow is Cut Off:** In many cases, just advancing across a severe lesion cuts off the coronary flow. Once the balloon is in place, there is no way to check the location of the balloon before inflation. Select a balloon whose length well covers the lesion. Inject the contrast agent while advancing the

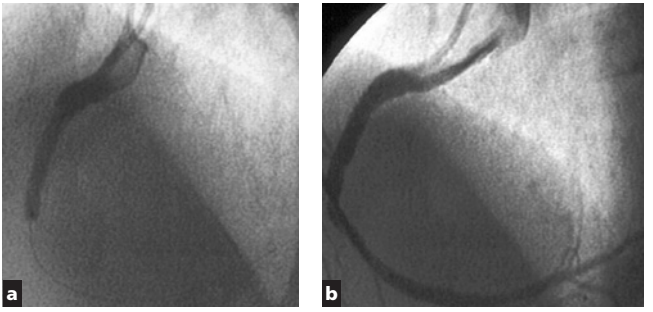


Figure 5-1 Assuring the adequate the size of the balloon and stent. (a) A balloon was inflated. Five seconds before deflation, inject contrast agent. Compare the size of the balloon and the proximal segment. In this case, the size of the balloon is clearly smaller than the proximal arterial segment. (b) A larger stent was selected and deployed. A post-stenting angiogram showed larger size of the stented area compared with the proximal arterial segment. (Courtesy of the Cardiac Catheterization Laboratories of Community Heathcare System, St Mary Medical Center, Hobart, IN. Reprinted with permission.)

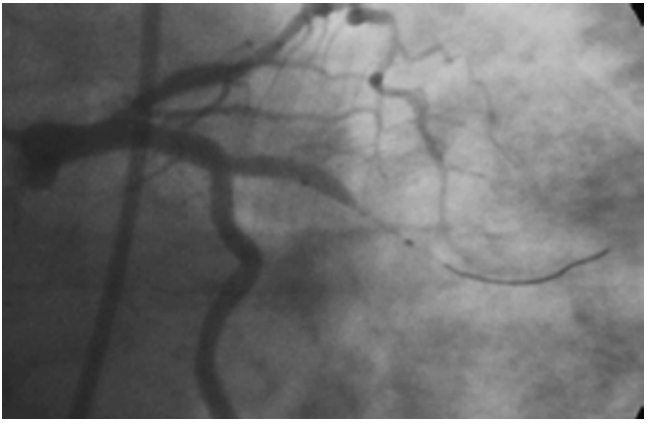


Figure 5-2 There was a tight lesion at the obtuse marginal. When the balloon was across the lesion, it obstructed the flow so a coronary angiogram could not be done and check the exact location of the balloon. Injecting the contrast while advancing the balloon will trap the contrast exactly at the center of the lesion. (Courtesy of the Cardiac Catheterization Laboratories of Community Heathcare System, St Mary Medical Center, Hobart, IN. Reprinted with permission).

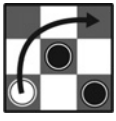
balloon. The balloon traps the contrast and shows exactly the critical point of the lesion. If the balloon totally covers the lesion, then it can be inflated (Figure 5-2).

****Failure to Reach or Cross a Lesion:** The causes of failure to pass a balloon to or across a lesion are multiple. If the lesion is too

severe, the balloon tip will not cross, and the guide would back out; then the guide should be held steady, engaged deeper or be replaced. If there is excessive tortuosity of the arterial segment proximal to the lesion, the solutions are: (1) to secure a more stable position of the guide; (2) to use a stiffer wire for the balloon to be tracked on; (3) to straighten the artery by asking the patient to take a deep breath; or (4) using a "buddy" wire, placed adjacent to the primary wire. A smaller balloon with a lower profile or length may also succeed in crossing a tight lesion. Once inflated, it creates a channel sufficient for the optimally sized balloon to enter. The pros and cons of each method of advancing a balloon across a tight lesion are discussed below.

Failure to Cross a Lesion: Should we use an over-the-wire (OTW) balloon or should we use a monorail balloon of the same size? The OTW balloon has a wire inside its core shaft so its pushability is better. The monorail balloon has only the wire at the distal segment of the shaft so its pushability is lower. The proximal and mid segment of the shaft of the monorail balloon is made either of a stainless steel hypotube or of a polyamide or plastic core shaft. The core of the wire shaft is flexible, however its profile is bigger than the hypotube shaft [4].

TACTICAL MOVE



BEST Maneuver for Advancing a Balloon across a Tight Lesion

1 **No Added Cost FIRST Best maneuver:** Check the guide position, optimize co-axial alignment, deep-seat the guide if needed, so the guide can provide sufficient support for advancing the balloon
2 **No Added Cost SECOND Best maneuver:** Ask patient to take a deep breath in order to elongate the heart and the artery (less tortuosiy). During this short window of opportunity, advance the balloon
3 **No Added Cost THIRD Best maneuver:** PARADOXICAL MOVE – constant pressure to advance the balloon while pulling the wire so the balloon can cross the lesion. This technique is to decrease the friction between the wire and the lumen of the balloon catheter. It also helps to keep the wire straight and taut so the balloon catheter can slide on it
4 **$ ⌛ FOURTH Best maneuver:** Add a stiffer wire so the balloon can slide on it. The guide can become stiffer and cannot slide back out. The arterial segments are also straightened (less wire bias) and the balloon can be tracked on more easily
5 **$ ⌛ FIFTH Best maneuver:** Change to a lower profile balloon (monorail, compliant balloon material, center marker)

Failure to Dilate a Lesion: A rigid lesion with heavy calcification may prevent the full expansion of a balloon, even the balloon is inflated to near the rupture pressure. It may be successful but it exposes the patient to the risk of dissection or balloon rupture. The BEST first move is to use force-focused angioplasty by adding an (or two or three) extra-wire(s) besides the inflated balloon. The next best move is to exchange for a non-compliant balloon so much higher pressure inflations can be achieved. In lesions with heavy superficial calcium, the problem can be resolved with debulking by rotational atherectomy, followed by drug eluting stent (DES) stenting. The cutting balloon is the best option in PCI of the undilatable fibrotic lesion. Different options are listed below.

TACTICAL MOVE

BEST Options for Dilating Undilatable Lesions

1 $ **FIRST Best move:** Add another wire and perform force focused angioplasty
2 $$ **SECOND Best move:** Change to a non-compliant balloon and perform higher pressure inflation
3 $$ **THIRD Best move** Cutting balloon angioplasty for non-calcified and calcified lesion
4 $$ **FOURTH Best move** Rotational atherectomy for calcified lesion

TECHNICAL TIPS

****Force-Focused Angioplasty:** If the balloon fails to break a plaque, it is withdrawn into the guide. A second wire is advanced beyond the lesion. The balloon is readvanced, positioned across the lesion, and inflated as usual. With the wire across the lesion, the pressure is then focused on the wire, which acts as a cutting wire to selectively put pressure and crack the plaque. Complications include dissection, which can be treated by stenting. It is best done with an undersized noncompliant balloon that allows the operator to go to high pressures without concerns of balloon oversize relative to vessel size or balloon rupture.

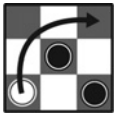
****Exchanging an OTW Balloon Catheter over a Regular-Length Wire:** It is simple to exchange a balloon catheter over the monorail system. When exchanging a balloon on the OTW system, the indwelling wire must be extended (docked) to a second wire, or a long exchange (300 cm) wire is needed. However an OTW balloon catheter can be exchanged without the need of another wire. With the indwelling wire held immobile, the balloon catheter is removed slowly until its proximal hub meets the proximal tip of the wire. Attach a 5-cc syringe of contrast to the central lumen of the balloon catheter. Persistently inject the contrast while simultaneously withdrawing the balloon.

This persistent injection will move the wire forward while the balloon is removed slowly. To successfully accomplish this exchange, the wire should not be bent and the catheter well flushed before starting the procedure. The Y-Touhy should be open. The catheter should be positioned on a straight line, to minimize any friction with the wire [5].

****Balloon Angioplasty of Large Vessels:** Current maximum balloon size is 4 mm in diameter, so when the coronary artery or the saphenous vein graft (SVG) is larger than 4 mm, the hugging balloon technique or use of a peripheral balloon is suggested. The two balloons are positioned side by side and inflated simultaneously. The combined diameter will be 70% of the sum of each balloon alone and the cross-section area would be oval rather than round [5–7].

Failure to Deflate the Balloon: Inability to deflate the balloon is a rare occurrence. Possible causes are excessive twisting: more than 360° in order to cross a distal lesion [8], or entrapment in the distal portion by a tight lesion. Comparative usual maneuvers to deflate the balloon are listed below.

TACTICAL MOVE



BEST Options When the Balloon Fails to Deflate

1. **No Added Cost FIRST Best option:** Deflate the balloon with the inflation device
2. **No Added Cost SECOND Best option:** Deflate the balloon with a 50 cc syringe directly at the inflation port
3. ♦♦ As a **last resort**: Inflate the balloon to rupture it. Prepare for damage control from dissection or coronary perforation [9]
4. ⧖ ♦♦ Rupture the balloon with the back end of a wire
5. **$$** ⧖⧖ ♦♦ Surgical removal of balloon

TECHNICAL TIPS

*****How to Puncture an Undeflatable Balloon?** After exhausting all maneuvers without success, one rarely used measure is to advance a small new OTW balloon immediately next to the proximal end of the entrapped and still inflated balloon. Remove the wire of this OTW balloon, reinsert the wire back, with the stiff BACK END in first. Inflate the new balloon at low pressure to position the sharp tip of the wire at the center of the vessel lumen. Try to puncture the trapped inflated balloon with the back end of a 190 cm wire which has a tapered back end. Although there is a risk of coronary perforation, the hole would be quite small and unlikely to cause any significant complication. In addition, vessel trauma from balloon rupture can be much more extensive and more uncontrolled than a single pinhole puncture [10].

*****Impending Rupture Due to Material Fatigue:** Besides rupture due to excessive inflation or calcified plaque, another cause of

rupture is due to material fatigue [11]. Balloon fatigue generally occurs after numerous inflations and deflations of a re-used balloon seen frequently outside the US. As the balloon material undergoes fatigue, a focal bulging in the balloon during inflation may be observed. It is suggested that even when faced with an unyielding stenosis, inflation pressure above the rupture level marked by the manufacturer should be avoided [12].

Entrapment of Deflated Balloon During Withdrawal: Even though the incidence of entrapment of a deflated balloon is low, once it happens it is quite traumatic to the patient, operator, and the interventional team. The entrapment can happen in any unpredicted way. Different options for management are listed below. There are no best options. Different modalities of treatment can be tried on a trial-and-error basis.

TACTICAL MOVE

BEST Options for Freeing an Entrapped Balloon

1. **No Added Cost FIRST Best maneuver:** Push the balloon forward then pull it back
2. **No Added Cost SECOND Best maneuver:** Twist the balloon in an attempt to rewrap the balloon before pulling back [12]
3. **$** ⌛ Insert a stiffer wire alongside the entrapped balloon before pulling the balloon back so that the artery is straighter
4. **$$** ⌛ Advance a second wire distally, then insert an OTW balloon alongside the entrapped balloon and inflate the new balloon at low pressure to free the entrapped balloon [13]
5. **$$** ⌛ If the OTW balloon cannot be advanced, then advance a balloon on a wire alongside the entrapped balloon and inflate it to free the entrapped balloon
6. **$$** ⌛ Advance a commercial microsnare and tighten the loop as near to the balloon as possible, then pull the balloon back as you would any embolized material [14]

TECHNICAL TIPS

***Using a Commercial Snare to Remove a Balloon:** Cut the proximal end of the balloon catheter. Advance the snare using the balloon catheter as a wire. Arriving at the entrapped balloon site, loop the snare around the balloon, tighten the loop by advancing the transport catheter, and pull the snare and the catheter end to free the balloon [14]. Be prepared to unwrap the snare and pull it back alone if it is not possible to remove the trapped balloon.

***Why There is Repeated Balloon Rupture:** Balloon rupture can happen repeatedly as in a case reported by Gilutz *et al.* In a patient with in-stent restenosis (ISR), three consecutive balloons were ruptured during inflation. IVUS study showed a ridge of calcium protruding into

the lumen. Management of this problem includes use of stronger balloon, rotational atherectomy which can be problematic because it can ablate the metallic stent struts or as in this case report, coronary artery bypass graft (CABG) [15].

*****Damage Control for Balloon Rupture:** Balloon rupture is seen under fluoroscope as a quick dispersion of contrast agent from the balloon, with short contrast opacification of the vessel or decrease in the inflation pressure. When this occurs, slowly withdraw the balloon proximal to the lesion and inject some contrast to detect if there is perforation. The balloon is then removed if not entrapped in the lesion. Stenting should be performed if there is dissection.

ANGIOPLASTY WITH CUTTING BALLOON

Manipulating the Cutting Balloon: Because of the presence of the microblades at its side, the cutting balloon is quite stiff, and is difficult to curve around sharp bends. To overcome this problem, the cutting balloon is designed with very short length. While dilating the cutting balloon, a slow inflation strategy is used. There should be a 3–5 seconds interval between each atmosphere increase, to ensure that the peripheral balloon wings unfold slowly first around the blades, before inflation of the central core of the balloon. Rapid inflation could result in puncturing a hole in the balloon by the blades. The manufacturer's guidelines for balloon inflation should be adhered to (1 atm inflation every 5 seconds; maximum 6–8 atm). Some operators also recommend deflation of the balloon at the same gradual rate. Finally, withdrawal of the cutting balloon should not be attempted until an adequate time interval has elapsed to allow full rewrapping of the balloon.

TROUBLE SHOOTING TIPS

*****Extraction of Stent by Cutting Balloon:** The cutting balloon has its blades mounted along its length. During inflation, the blades are protruded outwards and exposed. Then during deflation there is a mechanism of rewrapping the balloons with multiple wings. During this process of rewrapping, there is a possibility of the creation of an anchor formed by the balloon and the blades, or just because it is a higher profile balloon strengthened with the blades. This can get stuck into the stent struts and prevent withdrawal of the cutting balloon. If the cutting balloon is pulled hard enough, it could pull with it the stent or part of the stent. Because the lumen of the artery is removed of its stent, the lumen can become avulsed and suffer acute occlusion [16,17].

The causes of cutting balloon entrapment can be related to unsuspected passage of the coronary wire through a stent cell which then is followed by a cutting balloon, due to locking of the microblades in the stent struts, with subsequent avulsion of the struts on attempted withdrawal, or in association with fracture of the microblade. If difficulty is encountered advancing the cutting balloon, the preliminary

presumption is that there is a possibility of wire passage through stent struts. Excessive resistance to advancement of the balloon should be interpreted as an indication of this, and rewiring considered. Where doubt exists, intravascular ultrasound can be used to confirm that the wire is within the stent lumen throughout its course. A number of strategies can be used to remove an entrapped cutting balloon. It should be inflated and deflated in an effort to encase the proximal portion of the microtome within the balloon [18].

TACTICAL MOVE

BEST Strategy for Freeing an Entrapped Cutting Blade

1 **No added Cost** **Push/Pull:** An avulsed longitudinal atherotome may be trapped between the stent strut and the vessel wall. The entrapped cutting balloon should first be advanced forward and rotated either clockwise or counter-clockwise in order to unhook the protruding microtome.

2 **$$** ⧖ ♦ **Buddy Balloon:** If the atherotome has become deformed in its mid-portion that has snagged a stent strut, a second balloon can be introduced alongside the entrapped cutting balloon and inflated in order to change the confirmation of the cutting balloon and release a stent strut. Although the "buddy balloon" technique may be useful in changing the configuration of the entrapped cutting balloon, the risk of balloon rupture from a protruding microtome is highly possible.

3 **$** ⧖ ♦♦ **Guide "Sheath":** With the extreme longitudinal arterial force sometimes required to remove an entrapped cutting balloon, the proximal vessel can sustain tractional injury, resulting in proximal coronary dissection. With some 6Fr guides, the guide can be deep-seated within the coronary artery, and the retraction force can be applied at the orifice of the guide. If the cutting balloon has been dilated through a stent strut, this maneuver is unlikely to be successful, but will lessen the degree of traction injury to the vessel.

4 **$$** ⧖ **Snare:** If a large (≥8Fr) guide has been used (or a smaller guide can be exchanged for a larger one), the hub of the cutting balloon can be cut and a snare can be placed over the shaft of the cutting balloon and its wire. The snare can then be advanced through the guide to the cutting balloon allowing for greater traction force to be directly applied to the entrapped device.

5 **$$$** ⧖⧖⧖ ♦♦ **CABG:** If the entrapped cutting balloon cannot be retracted despite these maneuvers, or if anterograde flow cannot be sustained, surgical consultation for CABG and removal of the entrapped cutting balloon should be undertaken. Although this therapy is a last resort, it is required in some cases with refractory cutting balloon entrapment [19].

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Chapter 6
Stenting

Thach N. Nguyen, Pham Manh Hung, Dinh Duc Huy, Nguyen Huynh Khuong, Nikola Bakraceski

General Overview
- Easy delivery
- High longitudinal flexibility + low profile
- Perfect deployment = great radial strength and curve conformity

Engineering Criteria For Stent Evaluation
- Longitudinal flexibility
- Radial strength

Practical Clinical Evaluation of a Stent
- Is this stent flexible? What is the radial strength?
- Does this stent brace itself against the wall with a strong strut network?
- Is the stent user-friendly?

Advancing a Stent
- **Strategic mapping**
- **Technical tips**
- **Testing the road
- **The "buddy wire(s)" technique
- ***The distal buddy balloon technique
- ***The proximal deflecting balloon technique
- ***Partial inflation of a stent in order to cross of tortuous segment
- **Tactical move:** Best maneuvers when a stent fails to advance

Deploying a Stent
- Direct stenting
- Predilating balloon angioplasty
- High-pressure post dilation of DES
- **Technical tips**
- **Deployment of a stent in a tortuous artery
- **Appropriate sizing for tapering artery
- **Deploying a stent without angiogram
- ***Post stent deployment balloon inflation

Recrossing a Stented Area
- **Caveat:** During PCI near a previously deployed stent
- **Technical tips**
- **Dottering the stented area
- **Steer the wire to a new branch

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

As a clinical cardiologist must know the pharmacological properties of a new cardiovascular drug or the mechanism of a new therapeutic device, interventional cardiologists must understand the basic physical or bioengineering principles of stenting so that the most suitable stent for a given lesion can be selected.

Easy Delivery: High Longitudinal Flexibility + Low Profile: A stent mounted on a delivery balloon should be able to easily negotiate the tortuous segments proximal to the target site without injuring the intima or eliciting spasm. This smooth delivery is termed high trackability as a stent (with excellent longitudinal flexibility) is passed easily over a wire. The two properties, high longitudinal flexibility and low profile, help to bring the stent to the target site within an allotted time frame and with minimal manipulation.

A stent can easily cross an angulated segment if its length can fit in the widest interval at the curve. If it is longer, it can be advanced as long as it can be bent or curved, or the arterial segment is not too calcified to relax and compliantly accommodate the stent.

Perfect Deployment = Great Radial Strength and Curve Conformity: Once deployed, a stent must have sufficient radial strength to resist the elastic recoil of the media and of the shifting plaque. In an emergency situation, it has to be strong to seal the entry of a dissection, patch a dissecting flap, or brace against the persistent compression of a growing intramural hematoma. In non-emergency situation, an expanded stent should be able to sustain its frame against the systolic compression of a myocardial bridge. In addition, an adaptively deployed stent would mold its shape along the contour of a curved segment rather straighten it and still provide a large desired lumen. These two properties of a deployed stent, great radial strength and curve conformity, would give an instant perfect angiographic result. Following deployment, the struts should be well embedded into the arterial wall and stop any systolic contraction or diastolic relaxation. Therefore, they effectively immobilize the stented arterial segment and prevent any ongoing injury to the intima, which is the nidus for any endothelial thrombotic formation. Excellent apposition of the struts on the vessel wall will guarantee the delivery of any drug from a drug eluting stent (DES) preventing intimal hyperplasia and ultimately in-stent-restenosis.

ENGINEERING CRITERIA FOR STENT EVALUATION

Longitudinal Flexibility: At the beginning of stent design and manufacture, in the coil design a single wire is coiled in different curves and crimped tightly around a deflated balloon. It has no longitudinal strut so it uses the delivery balloon as a platform for its struts to be positioned on the longitudinal plane. Without stiff longitudinal shafts, the coil stent is very flexible, however, the radial strength is minimal. For this reason coil design became obsolete. In the tubular design, the primary mechanism for flexibility is that the longitudinal struts should be rather short and interrupted while the circular struts should be bent or folded and positioned sidewise along the longitudinal axis, before deployment. This arrangement of struts makes a stent highly flexible during delivery while providing adequate radial strength.

Radial Strength: At the target site, the balloon is inflated to deploy the stent. In the tubular design, the struts that were previously longitudinal or folded along the length of the stent rotate outward away from the long axis, and become the circumferential struts. In the coil design, the circumferential loops are just stretched wider to attain the desired diameter. Because they are incomplete loops, their radial strength is lower as evidenced by a 15–20% loss of achieved diameter due to intrinsic recoil.

In general, a stent has higher radial strength if its longitudinal struts rotate more circumferentially during deployment and it has more struts that are thicker and wider. Thus, the majority of coil stents have thicker struts (0.12–0.20 mm) to increase radial strength while the stents of other design have thinner struts (0.05–0.12 mm) to increase longitudinal flexibility.

PRACTICAL CLINICAL EVALUATION OF A STENT

Is This Stent Flexible? In general, if a stent has no stiff longitudinal shaft along its length, it will be quite flexible. This is well evidenced in any coil stent design. With better supportive equipment to advance a stent (stiffer wire, more stable guide), or in the case of minimal tortuosity, the flexibility of a stent is not a major concern in today's busy cardiac catheterization laboratories. However, at this present time, the majority of patients undergoing percutaneous coronary interventions (PCI) are older and have more complex lesions: more tortuous, more calcified, located in distal position; so the flexibility of a stent is still a major concern in selection, advancement and deployment.

What is the Radial Strength? Most currently available stents have adequate radial strength. However, the most important concern is the even and reliable distribution of their struts or radial strength. In the left main trunk, at the anastomotic site of a saphenous vein graft, or in the lesions of elderly patients, the lesions are composed of extensive fibrotic tissue and have significant recoil pressure. In these situations, stents with high radial strength are particularly needed. It is also required for the long-term success in stenting of the carotid, femoral, popliteal, or tibial arteries, which may be subjected to external compression.

Does this Stent Brace Itself Against the Wall With a Strong Strut Network? intravascular ultrasound (IVUS) has demonstrated that stenting a lesion shifts the mass of atheromatous material along the longitudinal and radial axes. To achieve the best luminal diameter, a just-deployed stent has to provide a strong network of struts to fence off the recoiling atheromatous mass and provide a controlled shifting of the plaque burden along the longitudinal axis. It has to be able to prevent any intraluminal herniation of the plaque through its struts and any possible distal embolization.

Is the Stent User-Friendly? In general, an elective stenting procedure should not require more than 30 minutes if the equipment is reliable and user-friendly. Every step of the procedure should be achieved on a first try. Besides a strong guide support, the success of delivery depends on the size, flexibility of the stent–balloon complex, and the compliance of the arterial segments proximal to the target site and the adequate opening of the lesion site.

A flexible and small stent can slide on a floppy wire while a stiffer wire is needed to track a bulky, stiffer stent. A short stent can easily negotiate a sharp bend, while some longer stents cannot. During delivery, a stent should hold well to the balloon, thus avoiding the risk of inadvertent embolization. In case of failure of delivery, while attempting to withdraw an undeployed stent into the guide, there should be no feeling of resistance and the stent should be watched carefully to assure it does not slip off the balloon. If this occurs, the guide, stent, and wire should be withdrawn en-bloc. In all situations, the choice of stent depends on the operator's preference, experience

with a particular design, and critical evaluation of different structural features to maximize benefits.

ADVANCING A STENT

STRATEGIC MAPPING

When a stent fails to move forward, it is imperative to assess the stability of the guide position, the stiffness of the wire, and the tortuosity and calcification of the vessel and the flexibility and length of the stent.

Guide: The tip of the guide should be co-axial with the ostium and advancement of the device does not back out the guide. If it does, the guide is not strong enough and should be deep seated. If the guide is deeply seated and the stent does not advance then change of guide shape may not have necessarily proved successful. For a guide to be stiffer, it has to have larger lumen, a large area in contact with the aorta or be encased in a long (70 cm) sheath.

Wire: If the guide position is fairly stable and the artery is more or less tortuous and calcified then an extra support (as stiff as, or stiffer than, the first) wire may compensate enough for this mildly deficient guide back-up. The mechanisms of success of this extra wire are by making the guide stiffer (with an extra wire inside), straightening the arterial segment proximal to the lesion (so the stent can slide easier) and hopefully decreasing wire bias at the curve by projecting the wire away from the inner curve and being positioned more at the center of the lumen. However, if the guide is so unstable, (e.g. a Judkins left for PCI of the left circumflex artery (LCX)) then an extra wire is not sufficient to correct the problem.

Artery: If the artery is moderately calcified and tortuous, then the problem is clearly not from the guide, or the wire, but it is from the artery. Is there a lesion in the proximal segment that impedes the advancement of the stent? If there is, then dilation of the proximal segment (with later stenting) is required. On rare occasions, the problem encountered is due to a hard eccentric plaque with the friction on the calcified and rough surface of the plaque too resistive for the stent to pass. The additional extra-support wire was not strong enough to divert the stent away from the plaque surface. Plaque modification with rotational atherectomy should have solved this problem easily. However, it adds additional costs and procedure time as well as its associated potential complications.

Stent: The most important features are the stent flexibility and the smoothness of the stent surface over the balloon. Stents that have minimal flare-up of the stent struts upon bending typically perform best in such cases. Longer stents have a tendency to "lock" over

bends, particularly if flaring up of the stent struts occurs while the stent bends. This "locking" can be released by gentle motion and steady gentle force on the stent. Sometimes pulling the wire slightly can lead to release of the lock. Shorter stents generally behave more favorably over longer stents in tortuous vessels. DES lack the advanced mechanical properties shared by some of the modern bare-metal stents [1].

TECHNICAL TIPS

****Testing the Road:** After the lesion is predilated with a balloon, a stent is prepared to be advanced. If the proximal segment is tortuous, the question is whether the stent can succeed in arriving at the lesion site. One way to test the possibility is to advance the deflated balloon, with its wings still out, to recross the lesion. If the balloon can recross the tortuous segment and lesion, then there is higher chance (>50%) a stent can do too.

****The "Buddy Wire(s)" Technique:** The "buddy wire" approach requires one extra-support or heavy-duty wire to straighten the artery. As the stent cannot be advanced through a first wire, advance a second heavier wire across the lesion, then advance the stent as usual. Once the stent is positioned across the lesion, the buddy wire is removed and the stent deployed [2]. If one extra wire does not work, an additional third wire may help.

*****The Distal Buddy Balloon Technique:** On many occasions, because of poor support from the guide, a stent cannot be advanced to the intended area. After exhausting all the technical tricks (the buddy wire, bubble stent, bending the stent), an operator advances an over-the-wire balloon beyond the target lesion in the distal segment. There he inflates the balloon to entrap the wire. While pulling the entrapped wire to keep tension on it, the stent is advanced successfully on this taut wire. However, the balloon should now be removed, if not it will be stuck by the deployed stent. The mechanisms for success of this technique are explained in Table 6-1 [3]. The negative sides to this trick are: (1) need of an extra balloon; (2) inflation of a distal balloon which can cause endothelial denudation which is the initial lesion of the restenosis process; (3) rupture of a new plaque causing thrombosis or acute occlusion; and (4) the balloon will get stuck if it is forgotten to be removed before stent deployment.

Table 6-1 Mechanisms of the Distal Buddy Balloon Technique

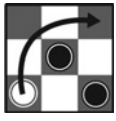
1	Pulling on the wire will seat the guide more deeply and firmly
2	Trapped by the balloon, the first wire can be pulled and become a stiff rail over which the stent is easily tracked
3	Straightening the proximal segment of the vessel causes less (or more) (and hopefully) wire bias

***The Proximal Deflecting Balloon Technique:** A small balloon is advanced near the questionable area and occupies the eccentric dead-end space just proximal to the lesion, and is strong enough to direct the second balloon/stent over to the lesion surface smoothly. Such a technique can also be applied in other circumstances such as difficulty in passing a balloon catheter into a stent for postdeployment high-pressure dilatation, and difficulty in passing a retrieval catheter or aspiration catheter through a stent at the end of the procedure after using distal protection devices. In a case report by Li *et al.*, because of wire bias caused by mildly calcified plaque, even after the use of a second buddy wire, the distal edge of the stent continued to get caught on the plaque, preventing it from further advancement. Gentle inflation of the buddy balloon to 2 atm provided a platform for the stent to be further deflected from the plaque, allowing it to advance to the desired position. However, there are limitations to this technique. A larger guide is needed and the vessel size needs to be large enough to accommodate two balloon catheters. Manipulation of multiple wires and balloon catheters may also induce trauma to the proximal parts of the vessel [4].

***Partial Inflation of a Stent in Order to Cross of Tortuous Segment:** In a case report of Fernandes *et al.*, a stent failed to cross a sharp bend despite many manipulations. Then the authors inflated the balloon–stent with 2–3 atm and all stents were successfully delivered and deployed. Partial balloon inflation also makes the balloon–stent complex stiffer, straighter and more co-axial with the lumen thereby eliminating local wire-bias. Of the angiograms illustrated in this case report, there is one common factor: the angle at the bend is very acute. A stiff and sharp straight stent would point towards the wall and can get stuck by a calcified plaque. Further pushing could have perforated the wall. So a round tip of a partially inflated balloon will smooth the tip and makes it less sharp so able to curve (or to float) along and around the bend [5]. What happens if the half-(heartedly) inflated balloon does not cross the lesion? The balloon cannot be deflated again: the stent would slide off the balloon quickly. So before trying this trick, the operator should think twice about the next move after this step.

As there are many options to overcome the difficulty of stent delivery, which one is best for the situation? This decision requires critical thinking so the procedure can be finished on time, without equipment wasted. The list of preferential best options is shown below.

TACTICAL MOVE



BEST Maneuvers When a Stent Fails to Advance

1 **No Added Cost FIRST Best maneuver:** Secure a more stable guide position or, if possible, the guide can be deep-seated safely

2 **No Added Cost SECOND Best maneuver:** 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 while asking the patient to take a deep breath in order to elongate and straighten the artery

3 $ ⌛ **THIRD Best maneuver:** Advance a second stiffer wire to straighten the artery (the buddy wire technique). Advance the stent on the second stiffer buddy wire; occasionally stents may actually advance more easily over a softer wire

4 $ ⌛**:** Change to larger guide or with different curve to achieve better backup and less friction at the ostium

5 $$ ⌛⌛ ♦**:** Dilate the proximal segment or plaque removal to facilitate stent advancement

Other exotic manipulations can be done without guaranteed success. They are listed below.

1 Partial stent–balloon inflation (rare) to deflect the stent away from an obstructive plaque or stent strut.
2 Inflate tip slightly (bubble stent).
3 Change the stent to a shorter one, if the problem is due to tortuosity of the proximal segment.
4 Select a different type of stent with better flexibility.
5 Bend the stent to conform the stent along the curve of the artery.
6 The buddy balloon technique: advance a second balloon beyond the deployment area, inflate the balloon to hold the first wire steady, pull the first wire to keep tension on it, and slide the stent on this taut wire.

DEPLOYING A STENT

As more PCIs are done without formal surgical backup, many lesions are strategically underpredilated to be stented immediately at standard size and higher pressure. Other operators suggest direct stenting without prior balloon inflation.

Direct Stenting: Direct stenting is a feasible and safe technique when used in selected coronary lesions, without significant calcifications and/or angulation. The degree of stenosis is not an important limitation, particularly in unstable angina where thrombus plays an important role. In the case of a type A lesion, there is not much difficulty in measuring the reference diameter for accurately sizing a stent. In a lesion with chronic distal vasoconstriction due to low flow, the angiographic distal reference diameter may be smaller. Sometimes the strategy of direct stenting backfires because of potential for only partial stent deployment (e.g. due to lesion fibrosis, calcification or balloon rupture), risk of stent loss and difficult stent retrieval, potential for inaccurate stent placement if poor distal vessel opacification. Therefore, it is important to check for the presence of heavy calcium at the lesion and in the proximal segment prior to angioplasty and

Table 6-2 Factors Favoring Successful Direct Stenting

1	Young age: <70
2	No calcium at the target and other coronary vessels
3	No severe proximal tortuosity
4	Not a LCX lesion
5	Not a too distal location

stenting for possible rotational debulking (if still available in your cardiac catheterization laboratory (CCL). Fluoroscopy alone is not sensitive enough to detect superficial calcium. Do use a moderate push to attempt the passage of the stent to the desired position at the lesion site. Avoid prolonged or forceful manipulation to cross the lesion because the stent can be stripped off the balloon and embolized distally.

The factors favoring successful direct stenting, and the contraindications, are listed in Table 6-2 [6].

Predilating Balloon Angioplasty: The goal of predilation is not to achieve a perfect result, but it is to facilitate the positioning of the stent. Perfect angioplasty may eliminate the angiographic landmark of the lesion and make the location of stent deployment uncertain and full coverage of injured segment doubtful. However, if the lesion is not expected to be heavily calcified and fibrotic, and is not fully dilated, then when deploying a stent the balloon may not be able to be fully dilated, leaving a half open stent which is the nidus for subacte thrombosis.

High-Pressure Post Dilation of DES: As a bigger lumen is found to have a better restenosis rate, there was a trend of post-dilating the stent with high pressure in order to achieve the largest lumen possible. A shorter noncompliant balloon is used so there is no stent edge dissection. However, with high-pressure post dilation, there was more plaque extrusion on the longitudinal axis, even exceeding the stent margins [7]. This observation explains the minimal haziness on the proximal or distal stent edges after deployment or the transient worsening of the ostial size of a side branch in the vicinity of the stented area and more distal embolization causing distal low flow.

Because of the new appearance of atheromatous material at the proximal or distal edge of the newly stented area (step up, step down), there is more turbulent flow in these areas. This increased shear stress is the most likely cause of proximal edge restenosis after DES. Therefore, when deploying a DES, the operator should avoid causing endothelial denudation in the proximal segment while advancing the stent towards the index lesion, avoid having different flow pattern at the entry and exit of the stented area (no step up, step down) and try to cover the DES on all intrumented (possibly future restenotic) areas.

TECHNICAL TIPS

****Deployment of a Stent in a Tortuous Artery:** When a stent is deployed in a very tortuous arterial segment, the vessel wall forms many invaginations beyond the struts rather than being well stretched. To maximize the length of the stented segment along the natural curve of the tortuous artery and to ensure that the struts are well apposed to the vessel wall, the stent is deployed while the patient takes a deep breath. Deep inspiration would make the heart more vertical, elongate the artery, and in that short window of opportunity, the stent is deployed.

****Appropriate Sizing for Tapering Artery:** After successful inflation of the balloon, 5 seconds before deflating it a small injection of contrast agent will verify the correct size of the balloon with the proximal segment of the dilated lesion. The same maneuver after deploying a stent will verify the correct size of the stent with the proximal segment of the dilated area. If contrast agent is seen flowing around the proximal segment of the inflated stent–balloon complex, the stent needs to be inflated at higher pressure and/or larger balloons are needed. If the stent is underdilated, the now deflated balloon is pulled back a few millimeters in order to avoid overdilating the distal end, and is inflated again with higher pressure to achieve greater size. This is only a rough assessment of the appropriate size of the balloon or stent, when IVUS is not available (see Figure 5-1).

****Deploying a Stent Without Angiogram:** In a case report by Hussain *et al.*, of stenting the mid left anterior descending artery (LAD) beyond the left internal mammary artery (LIMA) insertion, balloon angioplasty and stenting has to be done through the native artery using anatomic landmark (e.g. ribs, clips, side branches etc.). The reason is that no contrast injection could be done through the native LAD, while the flow through the LIMA was too brisk for visualization. The severe tortuosity in the LIMA prevented equipment passage. In such a case, balloon inflation and stent deployment was done through anatomical landmarks without angiogram [8].

*****Post Stent Deployment Balloon Inflation:** After deployment of a stent, the post-dilation high-pressure balloon should be short and noncompliant. The balloon should be short so it can fit entirely inside the length of the stent, without causing any tear at the two edges. If the balloon is longer than the stent, then the segment of the balloon exceeding the length of the stent is positioned at the proximal end. This position would help to avoid the need of recrossing the stent, if there is a rupture-induced dissection in the proximal end rather than at the distal end. This position also helps to avoid overdilating the adjacent distal segment that is often smaller than the proximal reference segment. Moreover, placing excess balloon length proximal to the stent should decrease the chance of entrapment and tethering of the ruptured balloon on the distal end of the stent, which could make the retrieval of the balloon extremely difficult or impossible.

RECROSSING A STENTED AREA

Often during PCI near a previously stented area, there is a risk of dislodging or removing the stent by any interventional hardware (balloon, rotoblation, cutting balloon, IVUS, directional coronary atherectomy (DCA), AngioJet catheters, etc. [9]). The first event in a later chain of catastrophe is that a wire exits through stent struts. So inability to pass balloons, stents, etc. over the wire must be taken seriously as a clue to this possibility of wrong wire exit. The wire should be advanced with a wide J curve, or repositioned, avoiding the previously stented area. Angiographic views can be deceiving and misleading. Sometimes, there is no resistance when the wire exits through the struts [9]. Some caveats for PCI near a previously stented area are listed below.

CAVEAT

During PCI Near a Previously Deployed Stent

1. Review prior angiogram for stent position and type.
2. Resistance to crossing device suggests passage between or behind struts (some times there is no tactile feeling of resistance by hydrophilic wire even if it goes through a side strut).
3. Use two orthogonal views in order to assess access, avoiding damage to ostial stents by diagnostic catheter or guide.
4. Advance wire easily with tip in wide J curve. Tip should move freely.
5. IVUS of parent vessel only. Avoid inserting IVUS catheter through struts.

If the first stent is well dilated and deployed, then the best technique to advance the stent is to gently dottering it.

TECHNICAL TIPS

****Dottering the Stented Area:** Hold the interventional device (stent, balloon, cutting balloon, IVUS catheter, etc) and advance it by gently dottering it. By moving gently the device forward and backward, the indwelling wire is also bounced gently forwards and backwards. Because the tip of the wire cannot go farther, the forward energy will be changed to the up and down direction that bounces the wire up and down the whole diameter of the lumen. This would make more wire-centering and open a chance for the interventional device to enter the newly stented area. If the wire cannot be bounced up and down, because of small vessel diameter, because the wire is well encased in a tight area, or because the monorail segment of the device (e.g. the IVUS catheter) is so short and does not transmit the dottering movement, then this technique does not work (Table 6-3).

Table 6-3 Unfavorable Factors for the Dottering Technique

1	Suboptimal opening of stent
2	Wire encased in a tight area
3	Small vessel diameter
4	Too short monorail segment of the balloon or IVUS cathether

****Steer the Wire to a New Branch:** Steer the wire into a different direction, or to a different branch in order to lessen wire bias and increase more wire centering; hopefully, it will help to advance the stent.

*****Other Exotic Techniques:** When crossing a stented segment of a vessel, a short stent can cross more easily than a longer one. If the stent fails to cross a stented segment while being tracked on a soft wire, a stiffer wire with less wire bias can direct the balloon–stent complex more to the center of the stented segment and help the stent to cross. If the stent fails to pass with a stiff wire in place, changing to a softer one may help. If a balloon cannot cross a stent, the balloon-on-a-wire type can have a higher chance to cross the stent, because there is no "step up" from the wire to the balloon, and therefore no "lip" on the balloon's nose to get caught inside a stent strut. Unfortunately, these balloons cannot be inflated at high pressure to fully dilate a stent [10]. A new wire with a stiffer distal tip that is flexible as a whole in its radial axis (the wiggle wire) can move up and down the tip of the stent–balloon complex as it is advanced. Recrossing a stented area that was deployed a long time before can be easier probably due to endothelial coverage of the struts and any small gaps between them.

*****First Balloon Deflecting Second Balloon Away from Problematic Area:** In a case report, Abernethy *et al.* suggested positioning a balloon at the resistance site where the balloon could not enter the stented area. Inflate the balloon at 2 atm. Then advance a second balloon as the working balloon. The first balloon would deflect the second balloon away from the problematic area and allow the second balloon to enter the stented area [11].

****Recrossing a Stent with a Bent Stiff Wire:** Often there is a need to cross a stent with a balloon for high pressure post dilation, to do angioplasty in the distal area, or to patch a distal edge dissection or perform stenting in the distal segment. If a balloon fails to recross a stent, it is usually due to the nose of the balloon engaging the stented arterial segment eccentrically or nonaxially. In order to facilitate coaxial entry of the balloon within the deployed stent, a stiff guidewire may be shaped so that a bend on the wire directs the balloon tip into the center of the stented lumen, thus facilitating passage. Recrossing a deployed stent with a guidewire is sometimes required, creating a large exaggerated loop on the distal wire tip to avoid passage beneath

struts. Difficulty, however, can be encountered in steering such a curve through the proximal vessel, especially if the stent is placed distally in a tortuous artery.

****Recrossing a Stent With a Movable tip Wire:** The Steer-It wire (Cordis Corp., Miami, Florida) is a 0.014-inch diameter wire consisting of a thin filament affixed at its distal tip to that of a hypotube in which it is housed. A sliding component of the proximal handle allows the operator to variably deflect the curvature of the distal end *in vivo*. After exit from the guide, the curvature at the tip was put a minimal. Then at the proximal edge of the stent, the curvature is maximized to >90º, and the wire is advanced through the stent. The knuckle formed while crossing the stent prevents substrut passage. Thereafter, the tip is relaxed again to suit the tortuosity of the distal aviculture [12].

TACTICAL MOVE

BEST Maneuvers for Recrossing of a Stent

1 **No Added Cost FIRST Best Technique:** The technique is to gently bounce the device forwards and backwards and because these movements are limited, so the wire will bounce up and down (more wire-centering) and open a chance to enter the lumen
2 **No Added Cost SECOND Best Technique:** Steer the wire into a different direction, or to a different branch or side-branch in order to lessen wire bias and increase wire centering; hopefully, it will help to advance the stent
3 **No Added Cost THIRD Best Technique:** Engage the guide in a more stable position or deep-seat the guide to change the entry direction of the wire, and hopefully lessen wire bias
4 **$ ⌛ FOURTH Best technique:** Insert a second stiffer wire to straighten the vessel

Other exotic techniques can be tried, however the chance of success is lower and not warranted. They are listed below.

1 Change the current wire to a stiffer one.
2 Use a shorter balloon or stent.
3 Rotate the balloon catheter while advancing it and let the catheter enter the stent by itself through its rotational energy (like torquing the JR catheter to engage the right coronary artery (RCA) ostium).
4 Bend the wire and place the bent segment near the ostium of the stent to be crossed in order to position the wire more at the center of the entrance of the stented segment, and to decrease wire bias.
5 Use a newly designed wire that wiggles its long tip up and down along the radial axis so the balloon–stent complex enters the lumen at the center (wiggle wire).
6 Use a more flexible balloon or stent.
7 Use a fixed-wire balloon to cross the stent.

8 Use a fixed-wire balloon to track alongside a buddy wire.
9 Mount a stent on a balloon with the tip partially inflated.
10 Park a balloon at the resistance site, advance the second balloon as the working balloon. The first balloon would deflect the second balloon away from the problematic area.
11 If only the balloon needs to enter the stented segment, inflate the balloon with 1–2 atm so the balloon will center the wire at the lumen and facilitate the crossing of the wire and balloon.

SIDE BRANCH DILATION

In complex interventions, a stent has to be crossed on its side for side branch dilation. Inflation through the stent struts causes stent deformity that decreases the diameter of the main lumen [13]. Different problems related to the side opening, side struts, and the stent itself are discussed below.

Opening of a Stent at its Side by Balloon Inflation: The average profile of a balloon ranges between 0.024–0.028″ while the profile of a cutting balloon is 0.041–0.046″. The mean diameters of the side opening created by inflation of a 4.0 mm balloon are listed in Table 6-4 [14].

Main Lumen Distortion and Restoration Following Dilations: In all stents, side dilation produces narrowing of the main lumen immediately distal to the dilation site. The severity increases with larger balloon size, especially after inflation of the 4.0-mm balloon. These changes are mostly reversed after the stent is redilated through its main lumen, especially after the inflation of the balloons in the kissing balloon techniques. The marked distortion of a stent may not be recognized by angiography [13].

CAVEAT

Entrapment of a Balloon During Side Branch Dilation: In a case report by Hongo *et al.* in order to dilate a side branch, a balloon was advanced through the side struts of a stent. Only the distal tip of the balloon would pass through the stent. Following inflation, the balloon was neither able to be advanced nor withdrawn farther. After an unsuccessful trial with various retrieval devices, the balloon was then removed by alligator forceps [15]. Entrapment may occur due to "wedging" of the catheter between acutely angulated stent struts, strut fracture, or balloon "winging" beyond the confines of a stent. To avoid the entrapment of the balloon across the struts, a few measures are recommended and listed in Table 6-5 [16].

Table 6-4 Size of the Opening Cell by a 4 mm Balloon

Name of stent	Opened Cell Size
CoStar	3.5 × 2.7 mm
BxVelocity	3.1 × 3.5 mm
Select	3.6 × 2.6 mm
Liberte	3.6 × 3.7 mm
Driver	4.7 × 3.5 mm
Vision	3.6 × 3.8 mm

Table 6-5 Recommendations During Dilation of a Stent on its Side

1	Placement of only the distal tip through the stent struts
2	Use a low-profile, nonwinged, undersized, high-pressure balloon
3	Avoid excessive high pressure
4	Knowledge of stent design and engineering characteristics so appropriate balloon size can be selected and opening diameter can be achieved

CAVEAT

Entrapment of the Distal Tip of an IVUS Catheter: In a case report by Sasseen *et al.* an IVUS catheter got stuck at the distal edge of a stent after multiple runs. After many manipulations and pulls, the IVUS catheter was finally removed minus the distal 2 cm trapped by the stent [17].

REDEPLOYING A STENT

With the trend of primary stenting or lower pressure balloon predilation, on many occasions a stent cannot be fully expanded, due to unexpectedly severely calcified vessel wall. In other cases, a stent can be crushed by inadvertent insertion of the dilation balloon through a strut. How to redeploy the stent?

TECHNICAL TIPS

****How to Cross a Crushed Stent:** After failure to advance a wire through the main lumen of a crushed stent (Figure 6-1a–b), the next step would be to take another picture from another orthogonal angle (90° opposite to the first angle) in order to locate the exact position of the possible opening (Figure 6-1c). Then advance a balloon with marker in the middle of the balloon. As soon as the marker is seen in the middle of the lesion it is assured that the lesion is crossed (Figure 6-1d). From there the stent is redilated successfully (Figure 6-1e).

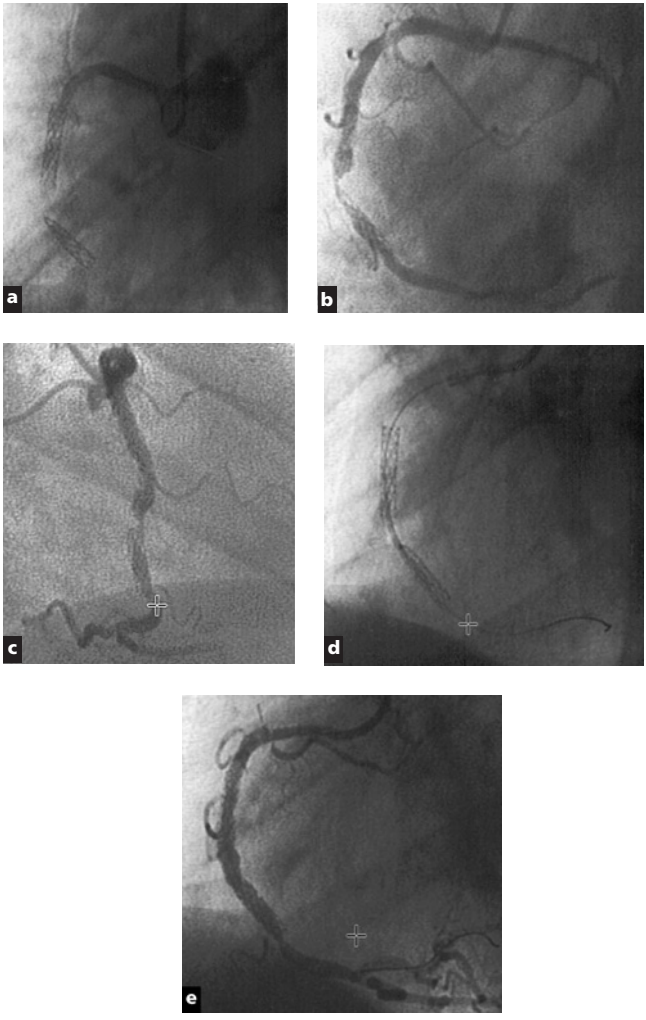


Figure 6-1 (a) The second stent is crushed at its opening. (b) The RCA in the LAO view. (c) Because the wire was unable to enter the stent ostium, another angiogram was done in the RAO view, (90° opposite) in order to locate the exact location of the true opening. (d) A balloon with marker in the middle of the balloon was advanced across the stent. (e) The stent was redilated successfully. (Courtesy of the Cardiac Catheterization Laboratories of Community Heathcare System, St Mary Medical Center, Hobart, IN. Reprinted with permission.)

Deploying a Stent After Failed Expansion by Balloon: In order to redeploy a stent, usually the operator would insert and inflate a noncompliant, high-pressure balloon. Even so, on occasion, very high-pressure balloon inflation fails to expand the stent.

Then conventional laser angioplasty in a blood medium would provoke local dissection behind the stent struts. The theory is that excimer laser irradiation of blood results in vapor bubble formation and acousto-mechanical trauma to the vessel wall, causing localized dissection. Usually, during regular laser angioplasty, intracoronary infusion of normal saline is required to displace the blood, and minimize the blood irradiation and consequent arterial wall damage. In case of failure deploying a stent due to unexpected heavy calcification, excimer laser irradiation would provoke local dissection, and weaken the vessel wall and allow full expansion of the stent [17].

Rotoblation through an unexpanded stent could be done in an exceptional case. However, what is the mechanism of enlarging the stent by rotational atherectomy? In a case report by Hadjimiltiades *et al*., a stent failed to expand because of unexpectedly heavy calcification at the lesion site. So after failure of all attempts to deploy the stent, the patient was prepared to have rotational atherectomy. It was performed with a 1.75 and 2.0 mm burr at 180.000 rpm through the unexpanded DES. Finally, post-dilation with a 4.0 mm Quantum Maverick balloon was successfully performed. The angiogram showed

TACTICAL MOVE

BEST Maneuver for Redeploying a Stent After Prior Expansion Failure

1 **No Added Cost FIRST Best maneuver:** Use the same balloon and increase inflation pressure to maximum. Be sure the proximal end is well inflated. Inflate the balloon with maximum possible high pressure so at least the proximal end is opened as much as possible in order to facilitate the reinsertion of a new balloon. It is not always easy to reinsert a new balloon inside an underdeployed stent. Be careful when moving the balloon because the stent is at (not all the time stuck to) the lesion and can easily be dislodged because the distal end is small and the proximal end is large. It is easier to dislodge it in a retrograde fashion with a large, winged and bulky poorly re-wrapped inflated balloon.
2 **$⧖ SECOND Best maneuver:** Change current balloon to a high-pressure and noncompliant balloon
3 **$$ ⧖⧖ ♦♦:** Laser angioplasty in a blood medium to cause local dissection behind the stent struts, then re-inflate the balloon to re-deploy the stent
4 **$$ ⧖⧖ ♦♦♦:** Rotablation (**DAYOR:** Do at your own risk)

an optimal result with a linear coronary dissection. IVUS showed an optimal expansion by ablating away some of the struts. Consequently, a second stent was implanted. The final IVUS showed two struts in the 80% of the stent circumference and one strut only where the first stent was damaged. The main concern is that the burr could get stuck in the stent. It may require surgery for its removal [18].

TROUBLE SHOOTING TIPS

*****Redeploying an Embolized Stent:** In case of inadvertent embolization of an undeployed or partially deployed stent, the best way to resolve the problem is to assure the wire stable inside the main lumen and to reinsert the same balloon and reinflate it (especially the proximal half so any further attempt to insert a new balloon will be facilitated). To insert a new noncompliant balloon into the stent is not easy. If successful, then deploy the stent with prolonged higher pressure. However, it is difficult and/or it will take a lot of time, patience, skill and luck to advance a new balloon across a partially deployed stent. If not successful, the stent has to be removed.

*****Management of Balloon Rupture:** Not infrequently after deployment of a stent, especially if it is a reused balloon, the balloon can rupture. Rarely, as proved by IVUS, an irregular jagged-appearing calcified lesion can penetrate into the lumen of the stented area and can cause repeated perforation and rupture of the balloon. In a case report by Zellner *et al*., while a balloon made with polyethylene terephtalate (PET) ruptured twice, the nylon material of a third balloon was able to withstand high pressure without being punctured [19]. If the heavy calcification was detected earlier, rotoblational atherectomy would have been helpful. However, once the lesion is stented, ablation with the burr is not an option as the stent abuts the calcified plaque.

*****Stent Deployment After Balloon Rupture:** When a balloon ruptures during stent deployment, withdrawal of a partially inflated balloon can dislodge the stent into the proximal segment. In order to deploy the stent, some experienced senior operators suggest: (1) keeping the ruptured balloon in place; (2) using a 20-cc syringe filled with contrast; and (3) injecting 2–3 cc very quickly to inflate the balloon in order to deploy the stent. Keelan *et al.* were able to partly deploy the stent using an automatic power injector. Using 50% diluted contrast at a rate of 20 cc/sec over 0.25 sec and a pressure limit of 200–400 psi, they found that 1 cc was injected before the pressure maximum was exceeded. The stent was sufficiently deployed with the damaged balloon to allow the removal of the balloon [20]. Many times, the balloon ruptures because of a tiny pinhole, so these quick injections can sufficiently inflate a balloon and partially deploy a stent; however, these injections can cause a jet injury at the arterial wall and may cause perforation.

COVERED STENT

Two polytetrafluoroethylene-covered stents are available on the market: the JoStent coronary graft (Jomed), in lengths 9–26 mm and a maximum achievable diameter of 5.0 mm, and Symbiot covered stent system (Boston Scientific), 20–45 mm in length and a maximum 5.0 mm in diameter. The JoStent has been constructed by sandwich technique: expandable polytetrafluoroethylene (PTFE) membrane, 50 μm thick, has been placed between two JoStent Flex stents made of 316L steel. The Symbiot stent is a self-expanding nitinol stent encased in a thin porous expanded PTFE (ePTFE) membrane. The JoStent GraftMaster (Jomed) can be delivered through 6Fr (inner diameter 0.068″) guide, while Symbiot stent requires 8Fr (inner diameter 0.086″) guide [21].

In the US, the Jomed stent (covered stent for coronary perforation) still needs to be hand-crimped. Most of the hand-crimping should be applied to the middle of the stent and not to the ends, to make sure the balloon material is not damaged. Always inflate the balloon first before mounting the stent, since the winged balloon material tends to hold the crimped stent in position more reliably than an uninflated balloon [21].

TECHNICAL TIPS

*****How to Check the Integrity of a Hand-Crimped Balloon Before Deployment:** To avoid crimping a stent on a ruptured balloon, three observations to confirm the integrity of the balloon should be checked prior to advancement (Table 6-6). In the case of a hand-crimped stent, the balloon-stent complex can be checked again when the stent is at the tip of the guide, before engaging the coronary artery, so there is still time to retrieve it if needed [22].

CASE REPORT Covered Stent in Aorto-Coronary Fistula: Successful occlusion of an iatrogenic aorto–coronary fistula by using percutaneous stenting of the great cardiac vein with covered stent via coronary sinus approach [23].

CASE REPORT Exclusion of Coronary Aneurysm With Covered Stent: The usual approach to exclude a coronary aneurysm is a technique that involves deployment of a covered stent to be anchored from the distal to the proximal nonaneurysmal segments of the artery.

Table 6-6 Checking the Integrity of a Balloon Before Advancing it into the Coronary Artery

1	No air bubble in the fluid inside the inflation device
2	No blood back inside the shaft of the balloon catheter lumen after the catheter is inserted into the guide
3	While the plunger of the inflation device is in the aspiration position, it does not return rapidly to the neutral position as it is released

In this specific case, the length of the segment to be covered with a stent was approximately 32.6 mm as measured by quantitative coronary angiography. Additionally, it might be difficult to advance a long PTFE-covered stent through the steep bend in the proximal right coronary artery (RCA). Therefore, sequential deployment of shorter stents was a reasonable choice. Stent deployment in the proximal part of RCA first could straighten the sharp bend and make distal stenting easier. Therefore, implantation of the stents from proximal to distal was attempted. A 2.5–5.0 × 26 mm JoStent Coronary Stent Graft (Jomed, Helsingborg, Sweden) was mounted and hand-crimped onto a 4.0 × 30 mm Maverick balloon catheter (Boston Scientific). The JoStent stent graft was positioned in the proximal portion of RCA to cover the proximal part of entry site of the aneurysm. The aneurysm was partially excluded from the coronary lumen.

To make passage of the second stent easier, the JoStent stent graft in RCA proximal was postdilated with a 4.5 × 20 mm Bypass Speedy balloon catheter (Boston Scientific) to 14 atm. A 3.5 19 mm premounted JoStent stent graft was positioned in the mid segment, partially overlapping the proximal JoStent stent graft, and deployed at 18 atm. Postdilatation of the overlapping segment between the two JoStent stent grafts was performed with a 4.5 20 mm Bypass Speedy balloon catheter (Boston Scientific) inflated to 12 atm [24].

TAKE HOME MESSAGE

With more improvements in technology and more operator experience, procedural success has been achieved in more than 99% of patients undergoing more complex PCI, with a low complication rate (less than 1–2%). To achieve these results, technical precautions and goals are listed in Table 6-7.

Table 6-7 Precautions and Goals in PCI

Basic Preparations
1 Perfect first try anterior wall puncture of the femoral artery so there is no posterior hematoma and no retroperitoneal hemorrhage. Perfect first try puncture of the radial artery
2 Optimal anticoagulation with active clotting time (ACT) of 250 to decrease bleeding complications
3 Prior administration of oral or intravenous antiplatelet agents to prevent thrombotic complications in the coronary artery system
4 Generous blood return after insertion of the guide to remove any atherosclerotic debris or thombus in order to avoid any emboli-related complications
5 Gentle and coaxial intubation of the coronary ostium, especially the left main, to avoid any ostial dissection

(*Continued*)

Table 6-7 (*Continued*)

Coronary Interventions

6 Gentle advancement of interventional hardware to avoid provoking any spasm or intimal injury, which are the nidus for thrombotic formation
7 Fully dilate the lesion and stent it with optimal pressure and dimension
8 The DES should encompass the lesion, the two ends of plaque shifting, and the area injured by the predilating balloon
9 The stent struts should be apposed well into the vessel wall so the drug can be absorbed and so inhibits the intimal hyperplasia
10 Avoid causing damage to the endothelium in the proximal segment by interventional hardware during transit
11 Avoid causing dissection and have all the equipment available if dissection happens. Prompt redilation with the balloon or sealing the entry and exit of the dissecting segment with stent
12 Achieve an optimal lumen reconstruction with a good TIMI-3Flow to have the lowest rate of rest enosis

Post-Procedure Care and Follow-Up

13 Femoral or radial sheath to be removed as soon as possible
14 Good groin compression or deploy vascular closure devices and follow-up to prevent any hematoma
15 Watch for late (24–48 hours) renal failure in elderly patients

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Chapter 7
Transradial Approach

Alexander Doganov, Valeri Gelev, Valentin Krastev, Phan Nam Hung, Shigeru Saito

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complication

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GENERAL OVERVIEW

In the last decade the scope of modern interventional cardiology has dramatically changed with new user-friendly devices and more complex interventions performed in high-risk patients. The default transfemoral approach (TFA) is burdened with relatively frequent access site bleeds (depending on definition) and rarer but potentially serious complications as retroperitoneal hematoma (RPH), arteriovenous fistula (AVF) and pseudoaneurysm (PA). The impact of access site complications may include increased morbidity, mortality, need for surgery, blood transfusions resulting in longer hospital stay, higher costs and worsened quality of life [1]. The recent advent of closure devices has lead to faster hemostasis and ambulation but they appear to have similar overall complication rates as compared to manual compression [2]. What we and our patients need is greater safety, lower costs and early return to prior level of activities. The transradial route achieves excellent procedural success rate and virtually eliminates access site complications, allows rapid ambulation and even to be performed in an outpatient setting [3,4]. This is due to the superficial location of the

radial artery, the extensive palmar collaterals, lack of adjacent veins or nerves and hence easy and safe hemostasis, no ischemic sequelae or injury of surrounding structures.

Patient Selection: Patient selection for transradial (TR) coronary angiography and intervention is accomplished by checking the pulse as well as by Allen's test or pulsoxymetry/plethysmography, which confirms patent collateral flow. Contraindications include negative bilateral palmar arch (type D according to G. Barbeau) and candidates for hemodialysis. At the beginning of the learning curve (first 500 to 1000 TR procedures), it seems reasonable to exclude patients with weak pulses, low body mass index (BMI) and complex interventions like percutaneous coronary interventions (PCI) for primary angioplasty, bifurcations and chronic total occlusions.

Radial Artery Puncture: Equipment and patient preparation is simple and similar to that for femoral approach. The hand is positioned along the body with the palm pointing upward and obliquely. After local skin anesthesia with 0.5 ml 1% lidocaine, puncture is performed with 21–19 G venous cannula or bare needle. Optimal puncture site and needle angulation are shown in Figure 7-1.

The puncture technique with IV cannula is illustrated in Figure 7-2. When blood appears in the hub (a), the cannula is advanced a few millimeters in order to transfix the artery and the needle is withdrawn. Then the cannula is gently drawn back (b) until backflow reappears, and then a 0.021–0.032 inch wire is introduced (c).

Radial Sheaths: The most commonly used sheaths are 5Fr for diagnostic angiography and 6Fr for intervention but sometimes 4Fr sheaths

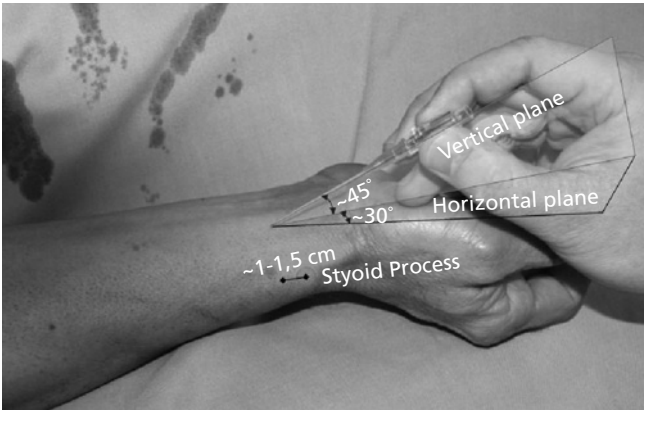

Figure 7-1 Angle of introduction for radial puncture.

are needed for coronary angiography via small radial arteries and 7Fr sheaths for complex interventions. The shorter sheaths seem preferable and hydrophilic coating reduces friction on insertion and removal by 70%. [5]. No skin incision is required with tapered sheaths. After insertion a cocktail of verapamil 3 mg and 3000–5000 units of heparin are injected through the sidearm. The operator can use single drug preparations or spasmolytic cocktails. Verapamil 3 mg has proved quite safe and effective. Previous radial catheterization does not diminish responsiveness to nitroglycerin and verapamil despite some reduction in the artery lumen diameter.

TECHNICAL TIPS

****Where is the Missing Artery?** In a case of «Atrophic» radial artery (radial artery is the terminal branch of the palmar arch), change the access site. Rarely, the «Radial» artery is not at the right place, it is on the lateral side of the wrist (very rare: 2/1000). So don't forget to check the pulse on the lateral side of the wrist.

Hemostasis: Hemostasis can be achieved simply with a roll of gauze and a couple of elastic strips (Figure 7-3) or with dedicated devices. One of the latter is transparent allowing visual control and has a marker ensuring selective graded compression of the radial artery without blocking blood return (Figure 7-4). Hemostasis usually takes 3 hours.

Right or Left Radial Approach: The choice between right and left radial approach is more or less related to operator preference. The left radial approach (LRA) requires a different table set-up and logistics. The right radial approach (RRA) is more nurse-friendly as it requires standard equipment. It is also more operator-friendly because, unlike the LRA, it doesn't require leaning over the patient in order to manipulate catheters, wires and devices. However, the LRA does seem preferable for slim females with small, spasm prone radial arteries, especially in the presence of subclavian and/or brachiocephalic tortuosity, and it is certainly the approach of choice when Allen's test is negative on the right side or when a left internal mammary artery angiography is indicated.

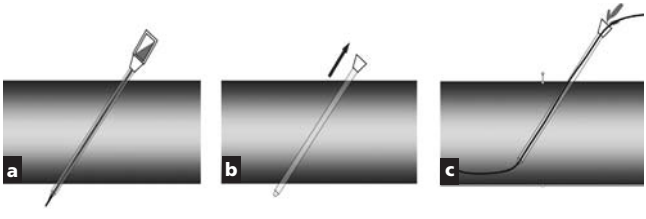

Figure 7-2 Technique for radial puncture.

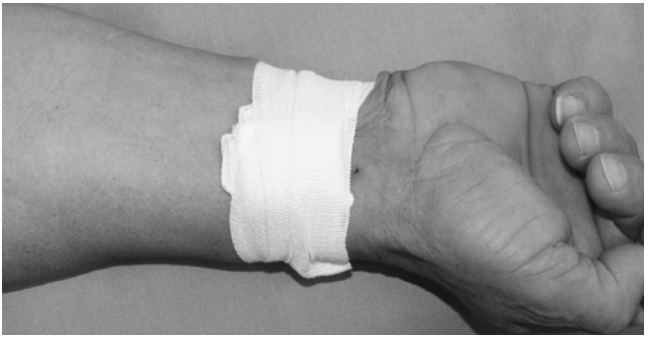

Figure 7-3 Radial artery dressing.

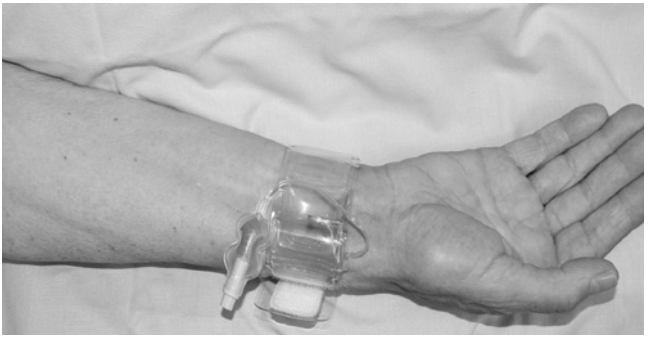

Figure 7-4 Radial artery pressure device.

Double Catheter Technique: Most operators still use the Judkins catheters for coronary angiography (M. Hamon – 93%) irrespective of the approach – right or left radial. This is probably due to the fact that most radial operators were originally trained to perform transfemoral angiography and feel comfortable with the Judkins catheters. In the Spaulding series of left transradial approach (TRA), there was high success rate for left coronary cannulation but a second catheter was needed for right coronary cannulation in every 10th patient [6]. Often successful left coronary artery (LCA) intubation is achieved with a Judkins left (JL) that is 0.5 size smaller than the one chosen for transfemoral angiography.

Single Catheter Techniques via RRA: Use of a single diagnostic catheter may minimize frequent exchange of hardware and thus may reduce the incidence of spasm and catheter embolism. This offers also a cost benefit – one catheter less is used. Several types of "multipurpose" catheters like the Kimny and Barbeau have been tested in this regard. Louvard *et al.*, who have had vast experience with the Amplatz

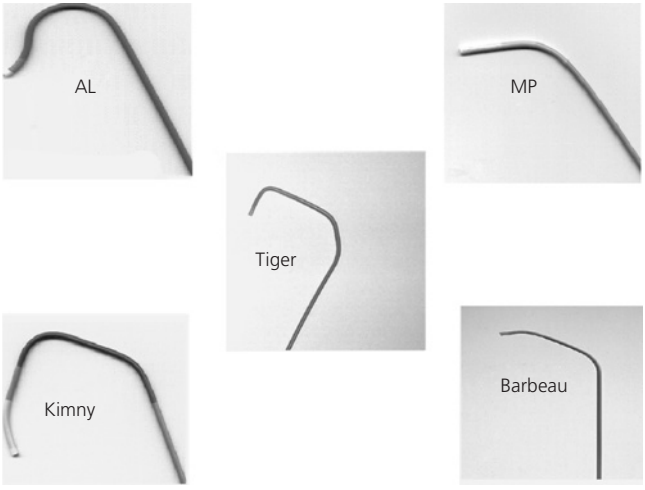

Figure 7-5 Catheters of different designs for TR coronary angiography.

left (AL) catheter with exceptionally high success rate, have advised the use of the AL for both coronary arteries via RRA [7] (Figure 7-5).

The TIGER Trial: This multicenter, randomized study compared the efficacy and safety of two "multipurpose" catheters for right TR coronary angiography. 194 patients were randomly assigned to the AL or Tiger 5Fr catheters at 4 European centers. Right radial artery access was successful in all patients (100%). Total procedural duration, x-ray time and dose, contrast medium volume, number of cine sequences and angiographic quality were similar. The study confirmed the very high success rate of transradial coronary angiography (99%). It showed a significantly favourable trend towards the use of the Tiger as a multipurpose catheter with a 95% success rate in both coronary arteries as opposed to 78% using the AL [8].

Angiography with the Tiger Catheter

LCA Cannulation: The catheter is advanced into the aortic root towards the left coronary sinus (LCS) in the anterior-posterior (AP) view, with the tip directed to the left of the screen. It is important that the tip is not deeply seated in the sinus. A slow and gentle clockwise rotation of the tip up to around 40–50° usually leads to engagement of the LCA ostium. If this fails, the catheter tip is rotated anticlockwise and slightly withdrawn until it eventually pops into the LCA ostium. Aimed to reduce catheter tension, this maneuver increases success rate to over 90%. In the event of a second failure, clockwise rotation from a higher sinus location and a slight withdrawal of the catheter usually enables successful cannulation of the LCA.

TECHNICAL TIPS

****Taming the Tiger:** In patients with a small ascending aorta and tip pointing downwards, the Tiger catheter has to be pushed down into the LCS and then be bent upwards while rotated counter-clockwise toward the LCA ostium. With a dilated ascending aorta, the catheter has to be rotated towards the LCA ostium with the wire tip between the primary and secondary curve of the Tiger catheter. This usually prevents the catheter bouncing upward into the dilated aorta. In cases of LCA cannulation failure, the Tiger catheter should be substituted for catheters with different curves (JL, AL).

Right Coronary Artery (RCA) Cannulation: Following LCA angiography, the catheter is withdrawn from the LCA ostium and rotated counter-clockwise in the left anterior oblique (LAO) view so that the perpendicular segment formed by the primary and secondary curve disappears on the screen. At this moment, a gentle push would direct the tip into the noncoronary sinus (NCS). Upon slight withdrawal and clockwise rotation, the catheter tip turns into the right coronary sinus (RCS) and engages the RCA ostium, in a similar fashion to the JR and AL technique.

TECHNICAL TIPS

****If the Tiger Keeps Going into the Conus?** If the catheter stubbornly enters the conus branch (CB), the maneuver can be repeated with a slighter and more discrete clockwise rotation accompanied by a short pullback, which prevents supraselective intubation of the CB. Alternatively, the curve could be straightened with a wire tip thus resembling the JR curve and/or the RCA ostium approached from a higher sinus position. Sometimes the problem can be solved with deep inspiration. If all that fails, the Tiger is to be exchanged for a JR catheter.

WIRES

Usually standard 0.025 or 0.035 (150–180 cm) J wires are employed to introduce catheters. The 0.025 wire can be used for the cannulation of the radial artery, while the J shaped 0.035 wire has the advantage of avoiding most side branches in the forearm and arm and providing better support. Advancing the wire around the shoulder of the patient should be done under fluoroscopy to ensure proper insertion into the ascending aorta without engaging side branches (carotids, vertebrals, mammary etc).

TECHNICAL TIPS

****Exchange of a Guide Through a Regular Length Wire:** Catheter exchange over a short wire resting in the ascending aorta can be easily accomplished under fluoroscopy by "blowing" the catheter off the wire with a syringe filled with saline. Do not use hydrophilic wires for this maneuver!

Radial Issues: If the wire or catheter does not progress easily, the operator should be aware of several possibilities: spasm, small radial artery, loops, high-take-off (sometimes as high as axillary origin), remnant artery, stenosis (radial, brachial, subclavian), wire in a side branch.

PROBLEMS IN THE FOREARM

Spasm: Radial artery spasm is the most frequent problem with transradial heart catheterization. It causes patient discomfort and reduces procedure success rate. Risk factors for spasm are patient- and operator-bound and include anxiety, age, female gender, improper sheath: lumen ratio, tortuosity, hematoma and repeated puncture. It is much more frequent at the beginning of the learning curve.

TECHNICAL TIPS

***How To Avoid Radial Spasm:** Give the patient adequate sedation. Keep the cardiac catheterization laboratory atmosphere quiet and peaceful. Cheerful attitude may help too. Use appropriate sheath size and by preference, the hydrophilic sheaths. Use fluoroscopy to see the problem each time resistance is encountered. Give generous dosage of vasodilators.

Forearm and Arm Loops, Remnants, High Take-off: Loops are relatively frequent and located in the forearm, arm and brachiocephalic segment. They represent different congenital and acquired anatomic conditions and most of them can be overcome with experience [9]. The really difficult ones are in the radial artery, sometimes with higher take-off (axillary, brachial) or with radio-ulnar anastomoses. The infrequent extreme loops that are generally accompanied by a remnant pose a real challenge to the operator and furthermore increase the risk of perforation.

TECHNICAL TIPS

****How To Handle Loops:** If you encounter a loop, do angiography through the sheath or catheter. Advance gently a hydrophilic 0.025″ J or 0.014″ PCI wire under angiographic control. This will usually solve the problem. Use a 4Fr relatively straight catheter (e.g. Multipurpose (MP); Figure 7-6).

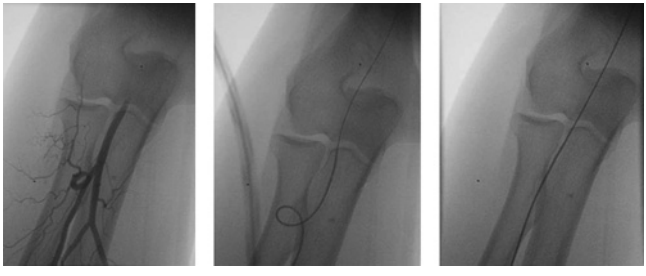

Figure 7-6 Loop in brachial artery straightened by wire.

Aorto-Subclavian Tortuosity, Loops: A tortuous right subclavian artery is encountered in about 10% of patients. Independent predictive factors are hypertension and age [9].

TECHNICAL TIPS

****What to do When Encountering Problem with Aorto-Subclavian Tortuosity? Don't panic. Use the MAGIC TRICK: Ask the Patient to Take a Deep Breath!** If the wire goes only in the descending aorta, push catheter in the descending aorta, pull wire, then slowly withdraw catheter and direct it into the ascending aorta with a counter-clockwise rotation.

Severe subclavian-innominate artery tortuosities and distal origin of the innominate artery result in a decreased forward force and increased friction during the progression of the wire or guide towards the ascending aorta. Use of a hydrophilic wire combined with deep breath frequently resolves the problem (Figure 7-7).

Stenosis: Dilate and stent if feasible or change access site.

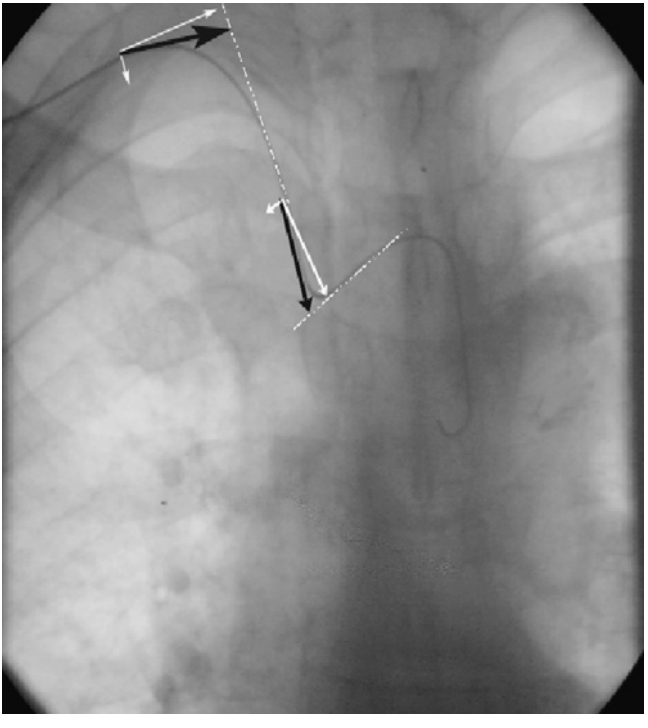

Figure 7-7 How to solve the problem with severe subclavian-innominate artery tortuosities.

Anatomical Variations: There are a number of variations in the origin and distribution of the brachiocephalic trunk including the bicarotid trunk and the rare retro-oesophageal right subclavian artery (arteria lusoria).

TECHNICAL TIPS

When viewing the arteria lusoria, use a specialized catheter with characteristic curve for arteria lusoria (Figure 7-8). If not sure, do an angiogram through the catheter.

Learning Curve: Limitations of the radial approach are the longer learning curve and lower rate of procedural success compared to the femoral approach. Proper patient selection is important at the beginning of the learning curve: it is preferable to start with large, easy to puncture arteries and simple cases, and gradualy progress to more difficult ones in terms of access and interventional complexity. Factors that predict TRA failure are female gender, low BMI, advanced age and lack of experience. After the inherent learning phase, TRA failure rate is very low (about 1%), the reasons being mainly anatomical difficulties [10].

When comparing the data for right TRA with the Tiger catheter for two periods: 1–500 TR procedures and 8500–9000 procedures, there was a clear trend toward procedural time reduction and increased success rate with mastering of the radial technique. An interesting trend towards shortening of the learning curve was observed for fellows who started their training in a well developed radial environment (Table 7-1; personal data).

TRANSRADIAL INTERVENTIONS

Guides: Guide selection in transfemoral coronary interventions (TFI) depends mainly on target lesion features, lesion location, the presence or absence of proximal tortuosity, size of the ascending aorta

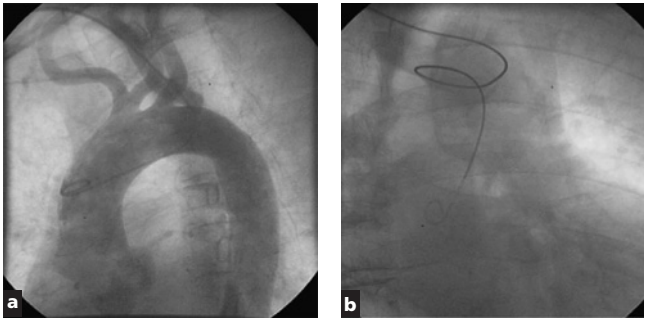

Figure 7-8 (a) Lusoria (retroesophageal course of subclavian artery). (b) Brachiocephalic tortuosity.

Table 7-1 Procedural time with Tiger catheter

	First 500 patients			Last 500 patients	
	All operators	Fellows	All operators	Fellows	Experienced operators
Puncture time (min)	4.9	5.6	2.6	3.3	1.9
Procedural time (min)	24.3	29.5	18.3	26.4	13.9
Fluoro time (min)	7.4	9.5	5.4	8.6	3.6
Success rate (SR)	92%	88%	96%	90%	99%
LCA-SR	95%	93%	93%	91%	96%
RCA-SR	81%	73%	89%	81%	92%
Both Tiger	80%	72%	88%	79%	92%

and location of the coronary ostium. During transradial intervention (TRI), besides the above, the operator should also take into consideration factors such as distal origin of the brachiocephal trunk from the aortic arch and subclavian-brachiocephalic tortuosities, which are frequently encountered in elderly and hypertensive patients, as well as the proper guide size, making sure that it is compatible with the radial artery diameter and the intended technique. Extensive manipulation of guides may provoke spasm, especially in anxious patients or those with small radial arteries or anatomical difficulties.

The vast majority of coronary interventions are performed through 6F guides, but in many patients 5Fr or 7Fr and 8Fr guides can be used if necessary [11]. Appropriate guide choice is even more critical with TRI than with TFA. Most difficulties, with proper selection and achieving good backup support, are met at the beginning of the learning curve. In most cases, experienced operators can obtain guide support comparable to TFI.

There are many specially designed guides for right TRA, but the most commonly used ones are the workhorse femoral curves. Selection and manipulation of these shapes is somewhat different when compared to TFI.

Left Coronary Artery: When a JL4 is used in patients with vertical and normal-sized aorta, the resultant backup support is 1.6 times lower in comparison with the one provided by the same guide if used transfemorally, as demonstrated by Ikari *et al.* [12]. The point of contact with the contralateral aortic wall during right TRA moves further up above the left coronary ostium and this results in reduced backup force. That is why experienced operators use JL3.5, which provides better support (Figure 7-9). JL is an appropriate guide in the setting of noncomplex lesions or in left main (LM) stenosis, where good support is not a critical factor. When the target artery is the left circumflex (LCX), a 0.5 larger size is preferred for better coaxial alignment. If active support is deemed necessary, deep seating for the LAD can be achieved

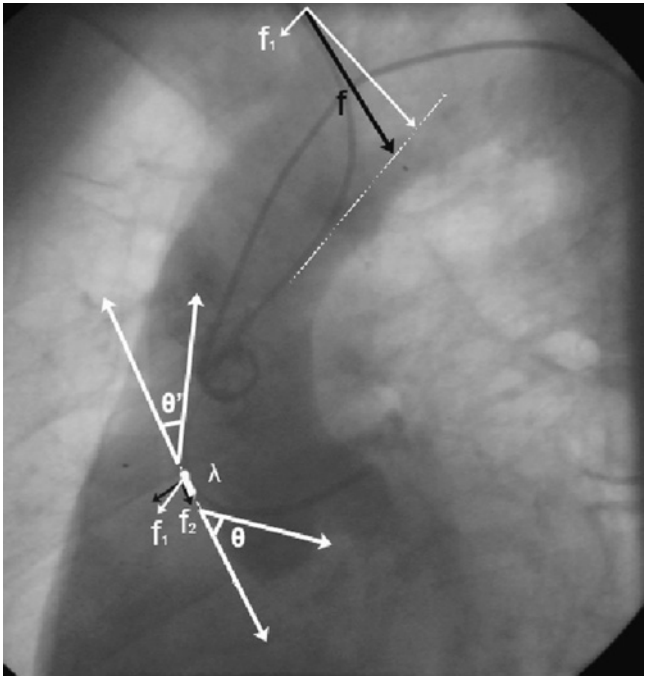

Figure 7-9 Mechanisms of back-up and pushing force according to positions and properties of the guide.

with 5Fr short-tip JL. This maneuver should be accomplished over the shaft of a balloon or stent with the assistance of deep inspiration and an apparent lack of LM or ostial left anterior descending artery (LAD) disease. Extra back-up guides provide greater support than JL, due to the larger contact area and the nearly right angle with the contralateral aortic wall. An inherent drawback of extra back-up guides is the tendency for deep intubation of the LAD or LCX in the presence of short LM (especially with larger sizes). In cases of complex LAD or LCX angioplasty and adequate length of LM, wide bifurcation angle or extreme proximal tortuosity, it often becomes the guide of first choice. AL 1.5 or 2.0 is suitable for complex lesions of LCX and provides greater passive support. Because of the downward pointing AL tip, the operator should pay attention to prevent dissection caused by deep intubation when pulling the balloon or catheter out of the ostium.

Cannulation of the LCA ostium and obtaining optimal backup support might be fairly difficult in patients with dilated and unfolded aorta. In cases with distal origin of the innominate artery, the guide approaches the LCA more from the left and this encumbers manipulation. Deep inspiration, leaving the wire in the guide during maneuvering or choosing guides with brachiocephalic curve may help in this setting.

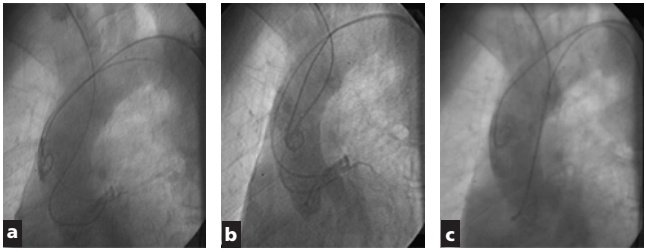

Figure 7-10 (a) An illustration showing the position of JL3 in TR and JL3,5 in TF approach in the setting of horizontal aorta. Parts (b) and (c) show the position of XB3,5 and JR4 in the same setting.

The mechanisms of back-up and pushing according to positions and properties of the guide are illustrated in Figure 7-9. Backup support depends on the forward force (f), static friction (λ), angle between the forward force vector and aortic wall (θ') and angle between the distal curve of the catheter and the opposite aortic wall (θ). Factors that determine f include catheter size, catheter material and number and magnitude of bends from the access site to the ascending aorta. Static friction depends on the contact area between the catheter and aortic wall. Backup support increases as θ is closer to 90° and $\theta' = 0$ degrees.

In cases of horizontal aorta or proximal tortuosities, the pushing force applied at the puncture site decreases significantly towards the tip of the catheter due to the increase in cumulative nonaxial forces. Guides, like Extra back-up (XB) or Amplatz left (AL), requiring pushing against the coronary sinus for LCA engagement, may be difficult to manipulate. Deep breath, putting the tip of the guide into the left coronary sinus and gentle forward push combined with counter-clockwise rotation are often helpful. (f = forward force at the origin of innominate artery, $f >> f_1 > f_2$; Figure 7-9).

Right Coronary Artery: The first choice guide for noncomplex or ostial RCA lesions is the Judkins right (JR) in sizes similar to the ones used for TFI. In cases of dilated aorta, there is lack of contact area with the contralateral aortic wall, which results in poor support. 5Fr JR and MP are suitable for deep seating or so called guide "Amplatzising". Indeed, best support can be achieved with the AL catheter but the operator should be extremely careful not to cause dissection with the traumatic AL tip. After overcoming the learning curve, the operator becomes quite confident with most femoral curves for interventions via right TRA (Figure 7.10).

TRANRADIAL COMPLEX INTERVENTIONS

A frequent argument against the radial approach has been techniques and device incompatibility for complex coronary interventions. Saito *et al.* have determined by ultrasound a mean radial diameter of

3.10 ± 0.60 mm and 2.80 ± 0.60 mm in Japanese males and females respectively. The cumulative relative frequency of radial artery diameter shows that 5Fr introducers can be used in 93% of patients, 6Fr in 85% of males and 72% of females, 7Fr in 71% and 40% and 8Fr in 45% and 24% respectively [11].

As the majority of coronary interventions today are performed via 6Fr guides, it is obvious that there are no substantial restrictions to the radial route. The same is true for many devices such as intravascular ultrasound (IVUS), a certain range of rotablation (ROTA) burrs, the cutting balloon and dedicated thrombectomy catheters. When in doubt, the operator can determine the radial diameter by angiography or echography. If the radial approach is incompatible with the intended technique or device, one obviously has to switch to the femoral approach.

Primary PCI: Data from the TEMPURA trial and a couple of registries (ICPS) comparing TFA and TRA in the setting of acute myocardial infarction (MI) have demonstrated that the radial approach is as effective as the femoral for primary angioplasty. Importantly, however, the TRA is practically devoid of access site complications, even in patients under heavy anticoagulation [13,14].

CASE REPORT Primary PCI through the Radial Approach: A 56-year-old lady presented with acute MI. Her ECG showed 3 mm ST-elevation in II, III, aVF, V3-V6. A diagnostic angiogram was performed through the right TRA with a AL3/5F and showed subtotal occlusion of proximal RCA with the LM originated from the proximal RCA (single coronary artery anomaly). PCI was performed successfully with a 6Fr JR 4 guide by direct stenting with Express 4.5 mm/20 mm at 16 atm (Figure 7-11).

Bifurcations: TRA is compatible with most bifurcation techniques and new generation balloons and stents with the exception of trifurcation PCI, certain cases with standard crush technique, kissing stents and some dedicated devices when radial arteries cannot accommodate larger than 6F guides [15].

Chronic Total Occlusion: Good guide support, which is essential for this subset of PCI, can be achieved with most femoral curves transradially. Other techniques specific for chronic total occlusion (CTO) PCI, such as the use of microcatheters, anchor balloon, double or triple wire, and deep intubation, can be easily applied via the radial approach [16]. If needed, contralateral injection can be performed by using the left radial artery or the ipsilateral femoral artery.

COMPLICATIONS

The complication profile of TRI is different from those related to the femoral route with no life-threatening complications and no need of vascular surgery or blood transfusions! TRI complication rate in 10847 patients: surgery 0.09%, blood transfusion 0.04% (TR workshop Massy, 2006). Most radial complications are preventable.

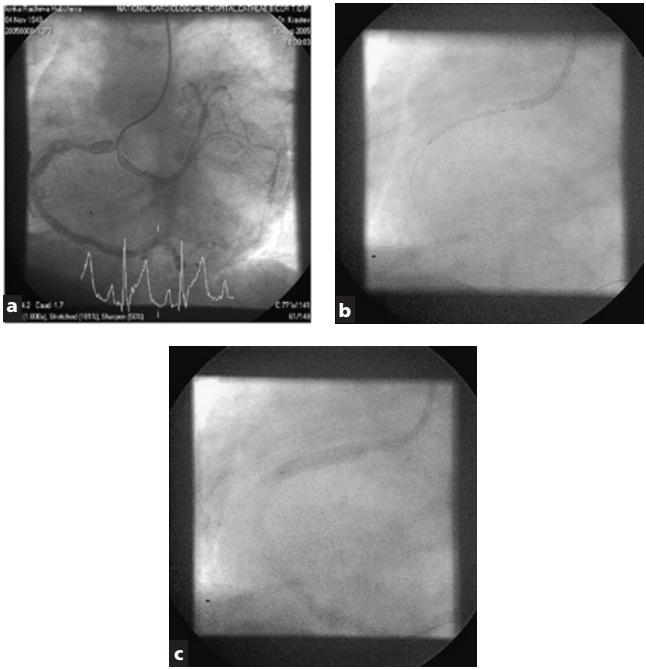

Figure 7-11 (a) RCA originated from the left system with lesion and thrombus in the proximal segment. (b) A stent is positioned across the proximal lesion. (c) Post balloon dilation with excellent results.

Radial Occlusion: Occurs in 3–5% of cases and is asymptomatic as a rule. 50% of radial occlusions spontaneously recanalize over time. Predictive factors for radial occlusion include long duration of catheterization, high sheath : artery ratio, heparin dosage, longer sheath and prolonged compression times [17].

Bleeding, Iatrogenic Radial Artery Perforation: One of the most common reasons for femoral cross-over is iatrogenic perforation or dissection of the radial artery, which can usually be treated conservatively with a proximal pressure bandage. If undetected, perforation may lead to severe forearm hematoma.

TECHNICAL TIPS

****How To Continue the Procedure when a Perforation has Occurred:** A long sheath is inserted across the perforation or a 4Fr MP long (130 or 150 cm) catheter and over it a 5 or 6Fr guide. This maneuver reduces friction and the 6F guide temporarily seals the dissection/perforation plane. This is especially helpful when there is accompanying spasm. The maneuver resembles the "mother and child" technique

used for increasing support during CTO PCI. It can be used in cases of radial perforation when one wants to finish the procedure transradially or there are no other available access sites. Perforation is usually caused by the guidewire and frequently there is spasm in the adjacent segments of the radial artery and it is usually impossible to traverse it with a 6F guide. If the wire is still in place, the operator can insert a 4F long MP /130 or 150 cm: Cordis and Terumo have these longer version catheters. Then the 6F guiding catheter, that has been premounted over the 4F MP before intoducing the "ensemble" into the 6F sheath, is navigated over the 4F MP catheter thus reducing the risk of further vessel trauma. If the guidewire had been withdrawn proximal to the perforation, the operator can use a nonhydrophilic PCI wire and insert over it the MP catheter and so on. Once the guide is in the left or coronary sinus near the ostium of the target vessel, the 4F MP can be withdrawn and PCI performed.

Distant Hematoma: Associated with bleeding remote from puncture site. Most distant hematomas are caused by perforation of a small side branch by the wire, especially with concomitant use of GP 2b3a blockers. It can be effectively managed with a cuff or elastic bandage applied to the forearm. It is reasonable to lower blood pressure or reverse heparin with protamine when feasible.

Rare Complications – Pseudo-aneurysm: Small as a rule, and can be usually treated conservatively by exerting local pressure. **Hematoma under tension** is a rare complication which may require surgical drain for a couple of days. **Volar compartment syndrome** is an extremely rare complication for which surgical fasciotomy may be indicated. **Entry site infection/allergy** presents as delayed skin reaction (2–3 weeks post catheterization) and is caused by sterile inflammation. It is related to transradial catheterization and the use of hydrophilic sheaths.

In a series of over 9000 patients the rate of access site complications, apart from radial occlusion that is as a rule asymptomatic, was very low. There were two cases with false aneurysm formation, which were treated successfully with prolonged compression and no major hematomas requiring vascular surgery. Most hematomas occurred as a result of improper wire and/or catheter manipulation, and they were more frequent at the beginning of the learning curve. Regarding coronary complications, there were three RCA and one LCA ostial dissection that were successfully managed percutaneously. Quite importantly, no dissection occurred with the Tiger catheter (three with AL and one with JL) (Personal data).

Prevention of Radial Complications: The rate of radial access site complications is negligible compared to femoral ones but still they do exist. Most of them can be prevented by observing the following take-home message.

TAKE HOME MESSAGE

Perfect TRI

1. The first attempt puncture is the best: it has the best chance of success
2. The first wire should be nonhydrophylic
3. Never force a wire or catheter against resistance
4. Have a low threshold for fluoroscopy. Look any time having trouble. Look before you push
5. Keep an eye for prompt diagnosis of hematoma
6. Treatment of the problem at earliest stage will bring the best result: Quick compression of forearm and arm in case of hematoma

NONCORONARY INTERVENTIONS

Patients After Coronary Artery Bypass Graft (CABG): Left internal mammary artery (LIMA) and right internal mammary artery (RIMA) angiography can be easily performed with the Tiger, JR or IM catheter from the ipsilateral radial artery. Most saphenous vein grafts (SVG) can be easily cannulated with the Tiger, JR or JL catheters via the right transradial approach. Frequently the insertion of a wire in the catheter may be helpful. The wire can be advanced for RCA grafts or pulled to the secondary curve for lower inserted left grafts (diagonals) and then pulled all the way back for high take-off LCX graft ostia. When the Judkins catheter fails, probably the best option is the AL catheter.

Carotid Angiography: Despite reports of difficult cannulation of the carotids, left and right selective carotid angiography have been performed with the JL or JR catheter from the right transradial approach with a high success rate – 84%. Lately successful cannulation of both carotids was achieved in a series of patients with the Tiger catheter via right radial approach (personal data).

Renal Angiography: Regarding the renal arteries, it is easier to cannulate most renals from the transradial approach than from the femoral approach as they originate typically in a caudal fashion from the aorta. The catheters of choice for renal angiography are the JR, MP or IM in their longer versions (125 cm). Transradial approach for renal artery stenting is feasible with «coronary technique». Furthermore it allows a better guide alignment with the renal artery. Longer balloon catheters (150 cm) and stents are needed in patients of larger stature [18]. The advantages of the radial approach are the same as for transradial coronary interventions: no access site complications, early ambulation, outpatient procedures, reduced cost.

Right Heart Catheterization: A small number of patients undergoing coronary angiography have indications for right heart catheterization. In keeping with the philosophy of immediate patient mobilization, right heart catheterization can be easily and safely performed from the veins of the elbow. In most cases the veins are 5Fr compatible. This allows implantation of a 5Fr temporary pacemaker if needed. When the access site is the cephalic vein, there are sometimes difficulties navigating the wire or catheter because of the unfavorable angle with the subclavian vein. If the wire or catheter repeatedly enters the coronary sinus, it can be withdrawn and preformed with a large curve that enables entry into the right ventricle and pulmonary artery.

CONCLUSIONS

Since the first publications by Campeau in 1989 and the first coronary angioplasty by Kiemeneij, the transradial approach has been transformed from an alternative to the femoral into a routine method in many centers [19,20]. The undoubted advantage of the radial approach when compared to the femoral is the extremely low risk of access site complications, patient comfort and preference [21]. Limitations are the longer learning curve and lower rate of procedural success compared to the femoral approach. After the inherent learning phase TRA failure rate is very low (about 1%). With enough experience and intimate knowledge of guide behaviour, complex interventions like primary angioplasty, bifurcation and CTO PCI can be safely and easily performed via the radial route.

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Chapter 8
High-Risk Patients

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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4. Closure of atrial septal defect and PCI at the same session

Technical tips

***How to predict and prevent pulmonary edema after closure of ASD

5. PCI and septal alcohol ablation for HOCM

6. PCI and CABG for patients with aortic stenosis

GENERAL OVERVIEW

High risk is defined as the probability and more importantly, the consequence, of abrupt closure of the dilated site, occlusion of large side or distal branches or widespread microvascular obstruction with spasm.

Abrupt closure of a large epicardial artery or of a side or distal branch is recognized by standard angiogram while occlusion at the microvascular level is seen as slow flow or no-reflow secondary to showers of atheromatous material from ruptured plaques. Their immediate clinical presentation and long term outcome is determined by the amount of myocardium in jeopardy and the degree of myocardial reserve. The treatment for abrupt closure at the epicardial artery is mainly by prompt stenting. However, the preventive measures of occlusion at the distal branches and at the microvascular level may require deployment of distal protection devices. The ultimate goals are to preserve the patency of the coronary arteries, the systolic function of both right and left ventricle and to prevent negative localized or global ventricular remodeling.

In general, as the patients undergoing percutaneous coronary interventions (PCI) are older and present to the hospital sicker, they are more vulnerable even with short episodes of ischemia or worse when there is irreversible acute closure due to extensive dissection or refractory thrombus. To facilitate short and long term success of PCI in complex lesion, the rationale that justifies the procedure and strategies to achieve clinical and procedural goals include: (1) identify clinical and angiographic features of complex PCI; (2) determine the appropriateness of complex PCI compared to alternative therapies such as open heart surgery (coronary artery bypass graft, CABG); and (3) formulate a strategy that maximizes immediate and long term success [1].

The clinical risk factors predicting in-hospital mortality and morbidity are listed in Table 8-1 [2]. The most important factor is severe left ventricular (LV) dysfunction. However, the risk of acute periprocedural closure depends on the high complexity of lesion morphology (left main disease, angulation $>45°$, lesions with thrombus, long lesion, bifurcation lesion, multiple vessel disease) and the suboptimal results of PCI.

As a result of acute closure of the dilated artery, the most common presentation prior to fatality is profound cardiogenic shock. The predisposing risk factors for shock are listed in Table 8-2. The factors associated with abrupt closure are mostly lesion-based. In contrast, the factors associated with shock and/or mortality are mostly clinically based, reflecting the poor ventricular function and greater extent of coronary artery disease.

Table 8-1 Clinical and Anatomical Predictors of Mortality and Morbidity Following Acute Closure

1	Left ventricular ejection fraction <40%
2	Creatinine >1.5
3	Diabetes mellitus
4	Triple vessel disease
5	Age >70
6	Acute coronary syndrome
7	Female gender

Table 8-2 High Risk of Shock if Acute Occlusion of Target Vessel

1	Left ventricular dysfunction (ejection fraction <30%)
2	Target vessel supplying more than 50% viable myocardium
3	Circulation to both papillary muscles compromised
4	High jeopardy score >3 [3]

Table 8-3 Strategies for Complex and High-Risk Lesion PCI

1	Over-prepare the possibility of a complication. Rehearse the scripted case strategy
2	Attention to the general factors: control diabetes, enough hydration to optimize renal function, lower use of contrast, etc.
3	Early and appropriate use of anti-platelet agents
4	Hemodynamic support (IABP) for hypotension or LV dysfunction
5	Meticulous access site management
6	Necessary and adequate sedation and anesthesia
7	Conservative equipment selection (larger guide, rotational atherectomy only if necessary)
8	Short procedure
9	Accept less than perfect results when benefits/risk unfavorable
10	Stop when appropriate before disaster, always favor safety over completion of procedure
11	Obsessive post-procedural care
12	Early follow-up angiography for selected group of patients (e.g LM disease)

When performing interventions in complex lesions, more complications must be anticipated. Should disaster occur, the management approach should already be identified, and all the mechanisms set in place can be put in motion as planned [4]. In the case of acute closure, a stable blood pressure is very important to secure survival. Lower blood pressure correlates with higher risk of death [5]. Thus, every attempt has to be made to keep a decent blood pressure while the patient undergoes cardio-pulmonary resuscitation (CPR) or emergency measures to reopen the artery with a perfusion balloon or stent are taken. Strategies for PCI of complex lesions in high risk patients are listed in Table 8-3 [5].

MODIFYING THE RISK FACTORS

Left Ventricular Dysfunction

LV dysfunction is the most important predictor of immediate and long-term survival in patients with coronary artery disease (CAD). Ejection fraction less than 30% and a target vessel supplying more than 50% of the remaining viable myocardium are considered high-risk factors for mortality and severe morbidity if there is acute occlusion of the target vessel [6]. The mortality of these patients ranges between 12% and 33%. Therefore, optimal medical management and fluid status, stabilization of decompensated congestive heart failure and unstable angina symptoms plus surgical consultation prior to PCI are needed. There is an absolute need to minimize the impact of peri-procedural ischemia by using short inflation time. PCI in a lesion of an artery providing collaterals should be efficient thus avoiding ischemia in distant areas. A cautious approach should be exercised in PCI of lesions that can cause slow flow or no reflow (lesion with thrombus, large bulky atheromatous lesion, degenerated vein graft etc.). In patients with significant baseline LV dysfunction, liberal use of right heart pressure monitoring, inotropic support, and intra-aortic balloon pump (IABP) are suggested.

Right Heart Pressure Evaluation and Monitoring: While undergoing complex coronary interventions, every patient, especially the patients with LV dysfunction and renal insufficiency, need adequate hydration to sustain a stable coronary and renal perfusion pressure. Monitoring the pulmonary capillary wedge pressure (PCWP) prior to intervention helps to optimize fluid management without provoking congestive heart failure and pulmonary edema, and is also a powerful adjunct for procedure planning. In some cases, with an elevated PCWP, the procedure may be deferred to allow time for clinical optimization by medical therapy. These patients may benefit from IABP support. Those with low cardiac output should be treated with afterload reduction or inotropic support during intervention.

Intra-Aortic Balloon Pump: The most common strategy to support LV dysfunction during complex coronary intervention is diastolic counterpulsation by IABP. Its mechanism is to increase the diastolic pressure and by that, the diastolic coronary perfusion. With inflation of a 40-cc balloon to coincide with the closure of the aortic valve, IABP increases the cardiac output roughly 30%. The indications for insertion of IABP prior to PCI are listed in Table 8-4. In patients with left ventricle ejection fraction (LVEF) $<25\%$ with stable BP >100 mmHg and PCWP <20 mmHg, then IABP standby is suggested [7]. After the patients successfully undergo the complex procedures, IABP can be removed. In some cases it is useful to continue IABP support on the day post procedure. This strategy can be followed if the ventricular function is severely depressed, filling pressure is elevated, or PCI results are suboptimal. When the PCWPs are adequate after the procedure, and the PCI results are good, balloon pump support may be discontinued prior to transfer out of the catheterization laboratory. Hemostasis can be

Table 8-4 Indications for Insertion of IABP Prior to PCI: Ejection Fraction <25% plus

1	Target vessel supplying the majority of viable myocardium
2	Jeopardy score >3
3	Abnormal resting hemodynamics (low BP <100 mmHg with high PCWP >24 mmHg)
4	Cardiogenic shock and multivessel disease

achieved with a vascular closure device making this approach very practical.

Options for Extremely High-Risk Patients: In general, there are many patients with so complex lesions on top of severe LV dysfunction, in whom the temptation of PCI should be resisted under normal circumstances (Table 8-5). However, as the left ventricular assist device (LVAD) is more available and refined, some patients could have PCI while under support of the LVAD and with successful PCI, could be weaned off the LVAD [8].

Acute Coronary Syndromes

In general, the patients with acute coronary syndromes (ACS) having continued, recurrent or refractory angina should be referred for coronary angiography for subsequent PCI if needed. PCIs in these patients were laden with complications related to the presence of intracoronary thrombus and the hypercoagulable state triggered by ruptured complex plaque. Effective antiplatelet drugs have had a dramatic effect in stabilizing these patients prior to intervention and diminishing peri-procedural events. The Evaluation of 7E3 For the Prevention of Ischemic Complication (EPIC) and the Evaluation in PTCA to Improve Long-term Outcome with abciximab GP 2b3a blockade (EPILOG) trials confirmed the clinical benefit of platelet inhibition in ACS [10,11]. However, GP 2b3a inhibitors are most beneficial when given prior to the procedure in patients with positive troponin. They did not improve the outcomes in low-risk patients (type A lesions), or in procedures with mechanical difficulty (chronic total obstruction), or heavy atheromatous burden with high possibility of distal debris embolization (degenerated saphenous vein graft (SVG)) [12].

Table 8-5 PCI Should be Refused [9]: Ejection Fraction <20% plus

1	PCI to the only patent vessel (especially SVG)
2	PCI required deep engagement of guide
3	Use of rotational atherectomy
4	Complex lesion morphology (e.g. in no-option patients, . . .)
5	Unstable hemodynamics with decompensated CHF, severe pulmonary hypertension
6	Not an ideal anatomy for stenting

Renal Insufficiency

Acute renal failure (ARF) after PCI is defined as an increase of creatinine level of >0.5 mg/dL or 25% from baseline. The incidence was 3.3%. Among patients with creatinine level <2 mg/dL, the risk of ARF was higher in diabetic than in non-diabetic patients whereas all patients with baseline creatinine level >2.0 mg/dL had significant risk of ARF. 20% of patients with ARF died during the index hospitalization compared with 1.5% without ARF. The risk factors for contrast-induced ARF are listed in Table 8-6 [13].

Hydration: With many protocols published but no one specific regimen identified, it is strongly recommended to parenterally administer a total of at least 1 L of isotonic saline beginning at least 3 hrs before and continuing at least 6–8 hrs after the procedure. Initial infusion rates of 100–150 mL/hr are recommended with adjustment post procedure as clinically indicated. Appropriate caution should be applied in the patient with known reduced left ventricular function or congestive heart failure [14].

Sodium Bicarbonate: The use of isotonic sodium bicarbonate has been demonstrated in one study to be marginally superior to isotonic sodium chloride (saline) in preventing contrast-induced nephropathy (CIN) in the high risk patient [15]. This protocol used an infusion of 3 mL/kg/hr for 1 hr before and 1 mL/kg/hr for 6 hrs after the procedure [14].

Medications: Pre-procedural management of patients at risk for CIN requires a review of the patient's medications and withholding, as clinically appropriate, potentially nephrotoxic drugs, including aminoglycoside antibiotics, anti-rejection medications, and nonsteroidal anti-inflammatory drugs (NSAID). Although optimizing volume status is essential, the decision to interrupt diuretic therapy must be individualized. Angiotensin converting enzyme inhibitor therapy may be continued but neither initiating nor changing dose should be considered until the patient is safely past the risk period for CIN [14].

N-Acetylcysteine: Despite multiple single studies, as well as several meta-analyses, the true benefit of N-acetylcysteine (NAC) is still unclear. However, NAC remains the most frequently prescribed medication in this setting, as a likely consequence of its low cost and lack

Table 8-6 Pre-procedural Clinical Risk Factors for CIN

Modifiable risk factors	Non-modifiable risk factors
Contrast volume	Diabetes
Hydration status	Chronic kidney disease
Concomitant nephrotoxic agents	Shock/hypotension
Recent contrast administrations	Advanced age (>75 years)
	Advanced congestive heart failure

of serous side effects. 600 mg of NAC should be administered orally q 12 hrs × 4 doses by mixing it with soda or orange juice [14].

Lower Amount of Contrast: Intuitively, the less contrast administered, the lower the risk for CIN. However, there are no studies that prospectively evaluate this hypothesis. Retrospective analyses have suggested that a total dose of <30 mL for diagnostic studies and <100 mL for interventional procedures lessen the risk for CIN [14]. The recommendations for prevention of CIN are listed in Table 8-7.

Stents should be used liberally to shorten procedural time and achieve stable acute results. Intravascular ultrasound (IVUS) can be used to monitor the procedure. A variety of adjuncts such as the guidewire with interval markers, digital road mapping can help in positioning stents or balloons. All of these efforts are to minimize the amount of contrast agent used during the procedure. There is of course no risk of nephrotoxicity in patients already on dialysis, though volume overload should not be overlooked. While patients with chronic renal failure could have high acute procedural success, their long-term outcome is still poor compared with patients without renal failure [16].

Table 8-7 Recommendations for Prevention of CIN

1 Identify risk
  a. Low risk – eGFR > 60 mL/1.73 m^2
    i. Optimize hydration status
  b. High risk – eGFR < 60 mL/1.73 m^2
    i. Schedule outpatient for early arrival or delay procedure time to allow time to accomplish the hydration
    ii. Consider the following recommendations (**2**–**5**)
2 Manage medications
  a. Withhold, if clinically appropriate, potentially nephrotoxic drugs including aminoglycoside antibiotics, anti-rejection medications and NSAIDs
  b. Administer *N*-acetylcysteine (equivocal data, see text)
    i. 600 mg administered orally q 12 hrs × 4 doses beginning prior to contrast
3 Manage intravascular volume (avoid dehydration)
  a. Administer a total of at least 1 L of isotonic (normal) saline beginning at least 3 hrs before and continuing at least 6–8 hrs after the procedure
    i. Initial infusion rate 100–150 mL/hr adjusted post procedure as clinically indicated
  b. Sodium bicarbonate (limited data, see text)
    i. 154 mEq/L @ 3 mL/kg/hr starting 1 hr before contrast
    ii. 154 mEq/L @ 1 mL/kg/hr for 6 hrs following contrast
4 Radiographic contrast media
  a. Minimize volume
  b. Low- or iso-osmolar contrast agents (ongoing data, see text)
5 Post-procedure: discharge/follow-up
  a. Obtain follow-up SCr 48 hrs post procedure
  b. Consider holding appropriate medications until renal function returns to normal, i.e. metformin, NSAID

TECHNICAL TIPS

****How to do PCI with 20cc of Contrast?** In a case report by Yamamoto *et al.*, the patient received two wires: one through the lesion and one in a branch nearby. Throughout the procedure, the positioning of the balloon and the stent was determined by referring to the bifurcation of the wires. Instead of regular angiography, selective angiography with 1.5 mL of contrast media was performed by injecting the contrast through the tip of a multifunctional transport catheter, which was inserted into the proximal portion of the artery along the marker wire. IVUS was repeatedly performed to confirm the result of each intervention. Because of these efforts to minimize the dose of contrast media, the session was completed with the use of 15 mL of contrast media [17].

TACTICAL MOVE

BEST Options in Minimizing Contrast During PCI

1 $ **FIRST Best option:** Graduated wire
2 $ ⌛ **SECOND Best option:** another additional wire in the side branch, close to the target lesion
3 $ ⌛ **THIRD Best option:** Perform selective angiography with 1.5 cc of contrast from the tip of a small catheter that was placed into the index artery
4 $$ ⌛⌛ **FOURTH option:** IVUS only, no angiogram

MULTIVESSEL DISEASE

Not every lesion needs revascularization because it is too distal, located in a small branch or in completed infarct areas. For patients with significant multivessel disease (MVD), there are safety concerns, rationale for indications, principles and strategies guiding the performance of their PCI. They are listed in Table 8-8.

Table 8-8 Strategies Guiding the Performance of PCI in MVD

1	**Pre-procedure evaluation**
	a. Solid indications
	b. Comprehensive evaluation of risks
	c. Practical assessment for chance of success
	d. Throrough discussion with patient and family about risks and benefits
2	**Strategy for SAFE procedure**
	e. Stratify the significance of contribution of each artery in which the lesion is located in the maintenance of blood pressure
	f. PCI FIRST the artery that is not ESSENTIAL in maintaining a decent blood pressure
3	**Procedural tactics for safety and success**
	g. Set up with good rationale the sequential order of lesions to be dilated
	h. Constant monitoring the progress and safety of the procedure
	i. Detecting early signs of hemodynamic unstability
	j. Dilating the first lesion: The challenging first minute
	k. Transforming high risk MVD PCI into single vessel PCI

STRATEGIC MAPPING

The master strategy of performing high risk PCI in patients with MVD is to identify from all the arteries, with or without lesions, which one is essential or non-essential in preserving adequate LV contractility and decent blood pressure, or in other words, sustaining life. Then performing PCI first on the lesion of the artery that is not vital in maintaining decent blood pressure would offer a safe and BEST strategy. The reason is that during PCI of an artery with severe lesion which DOES NOT contribute to the maintenance of a decent blood pressure, in the case of acute occlusion and that being the dilated artery, a decent blood pressure is still maintained. Looking at the other side of this (coin) strategy, there are lesions that are located in strategic locations to watch for because occlusion of these lesions could bring persistent hypotension, shock and mortality. Another approach is to stent any type A lesion, if the chance of success is high. Different tactics in evaluation of lesions and patients by clinical, non-invasive and invasive methods are discussed below.

CAVEAT

Identify the Lesions that Bring Catastrophe:
During PCI, if there is acute occlusion of the target lesion, the highest risk of catastrophe is from persistent shock leading to mortality. The cause of shock is due to refractory right and left heart failure, aggravated by the infarction of the papillary muscles triggering acute mitral regurgitation. Other mishaps include persistent complete heart block or right ventricular infarction. Surely these complications can happen and are non-lethal in patients with good LV function. However, many high-risk patients undergoing PCI have poor LV function. They are more vulnerable to any ischemic challenge and any mildly prolonged low diastolic blood pressure that decreases coronary perfusion. So during PCI of these lesions, the operators should be aware of these possible complications in order to prevent them, suspect their appearance at the earliest and reverse their course before the situation become critical. The list of these lesions in strategic locations is in Table 8-9.

CAVEAT

Lesions That are Possibly Safe to be Dilated First: There are many situations where the master strategy of dilating first the lesions that are non-essential for maintaining a decent blood pressure can be applied. In patients with arteries connected

by collaterals, it is best to perform PCI on the lesion of the recipient artery, unless the collaterals are minimal. The reason is that acute occlusion of the artery that supplies the collateral can cause remote ischemia and infarction in the recipient artery [18]. PCI of the stenosis on the supplying artery can be performed only if the first PCI was successful, with disappearance or reversal of collaterals. In patients with ACS, it is rationally sound to perform PCI first on the culprit lesion. In patients with acute myocardial infarction (AMI), it is now accepted to dilate the infarct-related artery, even without surgical stand-by. In patients with stable angina, performing PCI first on the lesion of the artery that is not vital in maintaining decent blood pressure is the best and safest strategy. In patients with subtotal ostial right coronary artery (RCA), if cannulation of the RCA by a diagnostic catheter causes ventricularization of the pressure then PCI of the RCA seems to be safe because the RCA may not contribute much to the maintenance of systemic blood pressure. In the patients with equally significant lesions in large arteries of equal size, without prior infarct, without any non-invasive imaging available, then there is no convincing rationale on a clinical basis for which lesion should be dilated first, except the type A lesion that is easier to be opened can be stented first (hit and run approach).

Table 8-9 Lesions in Strategic Locations with Possible Catastrophic Complications

1	**Proximal RCA**	Large amount of myocardium in jeopardy Closure of sinus node artery causing complete heart block Closure of PDA causing acute mitral regurgitation Persistent right ventricular infarction
2	**Proximal LAD**	Very large amount of myocardium in jeopardy Closure of diagonal causing acute mitral regurgitation
3	**Sinus node artery**	Complete heart block
4	**First Diagonal**	Acute mitral regurgitation due to ischemia or infarction of the postero-medial papillary muscle
5	**First large OM**	Acute mitral regurgitation due to ischemia or infarction of the anterolateral papillary muscle
6	**Right ventricular branch**	Persistent hypotension due to RV infarction
7	**PDA of RCA or PDA or postero-lateral branch of the LCX**	Acute mitral regurgitation due to ischemia or infarction of the postero-medial papillary muscle

CRITICAL THINKING

Why Are These Lesions Possibly Benign, While Others Are Unpredictable? In patients with an artery receiving collaterals, PCI of the recipient artery may causing no hemodynamic instability unless there is distal embolization cutting off the contralateral collateral flow. In patients with AMI, opening the acute occlusion may not cause any hemodynamic disturbance unless there is transient vaso-vagal symptom, no reflow or large distal embolization. The reason is that because the patient tolerated the acute occlusion and survived, so the transient occlusion of a balloon should not cause hypotension of catastrophic extent. It is the same rationale for ACS patients with elevated level of troponin, for patients with previous MI, chronic total occlusion (CTO) or subtotal ostial RCA. In patients with stable angina (no previous MI), unstable angina (without elevation of cardiac enzyme), these patients never experience any transient acute occlusion, then the clinical reaction to any acute occlusion has not been tested. These patients also do not have ischemic preconditioning so their reactions are unpredictable (Table 8-10).

Table 8-10 The Possible Benign Lesions to Be Dilated First

1	The lesion of artery receiving collaterals
2	The culprit lesion of non-Q AMI
3	The acute occlusion lesion in AMI
4	The lesion of old MI
5	The chronic total occlusion
6	The subtotal ostial RCA
7	The lesions of small arteries

TECHNICAL TIPS

****The Challenging First Minute:** When the balloon is inflated for the first time, during balloon occlusion observe carefully the ECG and the symptomatic reaction of the patient. Marked ST-segment elevation, severe pain, malignant ectopy, hypotension, marked decrease of wire tip movement (distal hypokinesis) all portend a major adverse clinical event in case of acute vessel closure [19]. This is why the first inflation should be short, of lower pressure and the speed of inflation slow and gradual. The second inflation may bring fewer symptomatic reactions because of collaterals recruitment and ischemic preconditioning. If the patient is symptomatic with the inflation, keep the inflation time short. The interval between the first and second inflation should be more than 2 minutes in order to achieve ischemic preconditioning [19].

How to Identify the Culprit Lesion by ECG and Angiographic Findings: In any situation, a comprehensive evaluation should be carried out. The interventional cardiologists come to the bedside, examine the patient, review the history, assess the problems, discus the risks and benefits. Usually the ECG would show some hints on the location of the lesion and the angiogram would pinpoint the culprit lesion that is subtotal and has haziness due to thrombus. The following case report illustrates this strategy.

CASE REPORT Assessment by History, ECG and Angiographic Findings: A 73-year-old nurse with recurrent typical angina at rest had a coronary angiogram that showed severe lesion in the proximal, mid-LAD (left anterior descending artery) and proximal RCA. Both arteries were large, not tortuous, nor too close to the ostium and had no side branch involvement. Which one should be dilated first? There is a need to identify the culprit lesion and rationalize the sequential order of dilation. The ECG showed mild ST depression in 2, 3, AVF so most likely the culprit lesion was in the RCA. The lesion in the RCA was subtotal with haziness suggestive of thrombus, a hallmark of unstable, fractured plaque. The lesion in the mid-LAD was severe and had sharp border, so it was most likely a stable lesion. The patient underwent successfully PCI of the RCA first by direct stenting, followed without complication by POBA and stenting of the two LAD lesions.

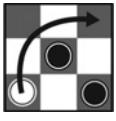
How to Assess the Lesion by History and ECG: Rationale for the Dilation Sequence: One of the ways to guess the significance of the severity of two lesions is to reconstruct the historical sequence of symptoms which show the clinical stability of a significant older lesion (while patient had stable angina after an AMI) and the clinical destabilization by the appearance of a new lesion (patient now has unstable angina). If the first lesion was caused by myocardial infarction, then the artery containing the first lesion was not vital in maintaining a stable clinical condition and indirectly not responsible for the preservation of blood pressure. PCI of the first lesion is most likely safe. The subsequent case report illustrates this strategy.

CASE REPORT Assessment of Patient with History of an Old MI: An 80-year-old woman with unstable angina had coronary angiogram that showed severe lesions in the proximal LAD and mid-RCA. Both were of type A. PCI should be technically easy and smooth for both lesions. Which one should be dilated first? In order to evaluate the contribution of each lesion to symptom and the maintenance of blood pressure, a resting ECG was ordered and a detailed history was taken. The patient had an AMI 7 years ago, which was confirmed by Q wave in V leads. She had stable angina since then: it means that the LAD lesion caused only stable angina, without much interference to the preservation of LV contraction or blood pressure. About

6 months ago, she began to have symptoms of unstable angina with chest pain with lower level of activities, chest pain at rest, more shortness of breath and more dizziness. Most likely the symptoms were caused by worsening of the lesion in the RCA that (without a lesion) did most likely contribute the most to a stable clinical condition in the last 7 years and indirectly to the preservation of blood pressure. By this rationale, PCI was performed on the LAD lesion without causing any hypotension. Once it was successful, PCI of the RCA was done without technical difficulty. However, the patient developed chest pain, hypotension, elevation of ST segment in leads 2, 3, AVF due to slow flow in the distal RCA. Coronary angiogram showed persistent patency of the newly stented segment with distal no-reflow. The patient recovered later with intra-coronary vasodilators. In this case, clearly the RCA was the one that maintained a decent blood pressure. The rationale for PCI first in the LAD lesion was justified. If PCI was performed in the RCA first, in the case of no-reflow without adequate blood supply from the left system, then severe low blood pressure with cardiogenic shock could have had happened with possible fatality.

Non-invasive Evaluation of Lesions: In general, besides a comprehensive history in order to identify the culprit lesion and its contribution in maintaining a decent blood pressure, non-invasive studies (ECG, exercise stress testing, stress echocardiography, nuclear stress testing) can be done in order to identify objectively the culprit lesion, its compromised area and its extent of reversible ischemia, wall motion abnormality or ST segment change. Nuclear scan can also identify easily other areas with adequate perfusion at rest and under stress. By that, PCI should be performed FIRST in the area of ischemia while adequate blood supply at rest to the other areas that maintain a decent blood pressure is assured by other patent arteries.

TACTICAL MOVE



BEST Technique in Identifying Essential and Non-Essential Lesions

1 **No added cost FIRST Best option:** Historical reconstruction of symptoms in order to identify the old lesion causing stable angina versus the new destabilizing lesion causing recent onset angina
2 **$ SECOND Best option:** Resting ECG identifying location of Q wave or ST-T depression
3 **$$** ⧖⧖ **THIRD Best option:** Nuclear scan for reversible ischemic change and extent of abnormal and normal areas
4 **No added cost FOURTH Best option:** Pressure tracing showing ventricularization during to cannulation of RCA ostial lesion by a diagnostic catheter

CAVEAT

Deceiving Nuclear Scan: The main mechanism of a nuclear scan is to show a 7% difference of isotope uptake between territories. If there is dramatic change in one area, subtle change in other areas may be missed. If there is diffuse disease, then there is not much difference between territories even there are significant lesions. It is well known that a nuclear scan can look normal in patients with three-vessel disease because there is homogenous, diffuse and widespread decrease of blood flow at stress and at rest.

TECHNICAL TIPS

****Important Meaning of Ventricularization of Pressure during Diagnostic Cannulation of the RCA:** The combination of a subtotal ostial lesion and ventricularization of pressure by a diagnostic catheter might suggest the irrelevant contribution of that artery in maintaining blood pressure. The case report below illustrates that notion and the safety of performing PCI in this kind of subtotal ostial lesion.

CASE REPORT Tactic During PCI of Patient with Subtotal Ostial RCA and LM Disease: A patient came to emergency room with recurrent angina at rest. A coronary angiogram showed subtotal ostial lesion in the RCA and 50% lesion in the mid- and distal LM. Could the patient safely undergo PCI of the ostial RCA? The cardiac surgeon refused to do CABG surgery because the LM lesion did not seem severe enough to require surgery. During the diagnostic angiogram, cannulation of the RCA with severe ostial lesion by a 4F Judkins right persistently caused decrease of systolic and diastolic blood pressure without any symptom. Because the patient was asymptomatic with occlusion of the RCA by a Judkins right catheter, it seems that PCI of the RCA would not cause any major hemodynamic and symptomatic disturbance. Convinced by this rationale, one of the authors successfully carried on a long and complex PCI of the ostial lesion without any decrease of blood pressure. Following the same strategy, recently another patient with severe ostial RCA and proximal LAD successfully underwent PCI of two vessels. Because the RCA seemed not to contribute much in maintaining blood pressure, the RCA lesion was dilated successfully without problem followed by uneventful PCI of the LAD.

How to Perform PCI in the Patients with ACS or AMI: In patients with non-Q AMI, opening the subtotal occlusion may not cause any hemodynamic disturbance, unless there is transient vaso-vagal symptom, no-reflow or large distal embolization. The reason is that the patient tolerated the acute transient occlusion and survived, so the transient occlusion of a balloon would not cause hypotension of catastrophic level. It is the same rationale for PCI in ACS patients with elevated level of troponin (non-Q MI). This rationale does not apply

to ACS patients who do not have cardiac enzyme elevation (unstable angina). This case report below illustrates the rationale and tactic during PCI of these extremely high risk patients.

CASE REPORT PCI of the Acutely Transient Total Occlusion due to Non-Q AMI: A patient came to the emergency room with chest pain, acute ST-segment elevation in V1–V4 and a blood pressure of 80–90 mmHg. When the patient arrived to the cardiac interventional laboratories, the pain subsided, the ECG changes were less prominent, however, the blood pressure was still in the 80–90 mmHg region. The coronary angiogram showed an old total occlusion of the RCA and LAD. The LCX was acutely occluded with minimal collaterals to the distal segment. The left ventricular end-diastolic pressure (LVEDP) was 30 mmHg. After CABG was declined by the surgeon due to extreme high risk, the patient successfully underwent PCI of the mid-LCX with IABP, and pacemaker on standby. The lesion was predilated with an undersized balloon, then a full size stent was successfully deployed. The tactic was that the balloon should be undersized so there was less chance for dissection and distal embolization. The first inflation was short (10 seconds), of lower pressure and the speed of inflation slow and gradual. The goal was to open a channel for stent position (Figure 8-1).

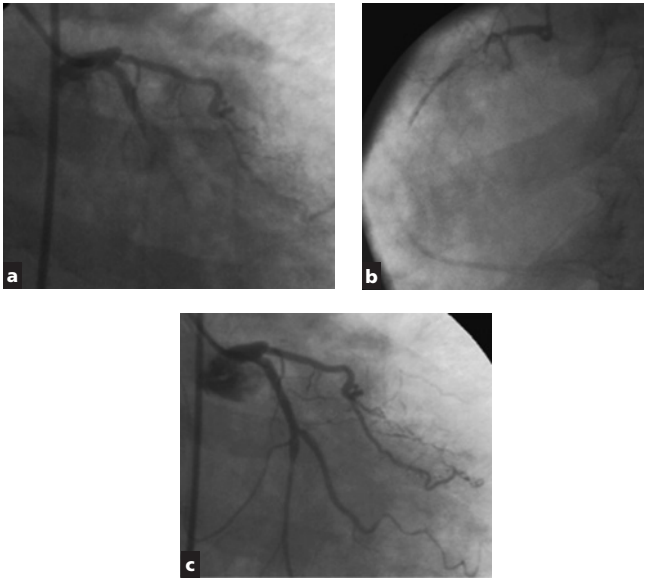


Figure 8-1 Strategy and tactics for PCI in patient with triple vessel disease and AMI. (a) The LAD was occluded at its ostium. It was seen as a stump. The LCX was acute occluded. Only a ramus intermedius was patent. (b) The RCA was severely and diffusely diseased. (c) The LCX was predilated with balloon and successfully stented.

EXOTIC COMPLEX INTERVENTIONS FOR THE WEEKEND URBAN WARRIORS

1 Hybrid MIDCAB and PCI: A patient with severe co-morbidity had severe lesion at the bifurcation of the LAD, large ramus and distal LM. The risk of mortality was too high for the patient to undergo conventional CABG, so the patient was scheduled for left internal mammary artery (LIMA) graft to LAD surgery, while the Ramus and LM lesion is to be dealt with by PCI. Under general anesthesia, a LIMA to LAD anastomosis was performed via a 2-inch left lateral intercostal incision (minimal invasive bypass surgery (MIDCAB). Patency of the LIMA to LAD graft was confirmed. Then the patient was prepared for PCI of the LAD on the table in the operating room. With the LAD territory protected by the newly placed graft, the ostia ramus and circumflex disease was approached with a provisional bifurcation stent strategy. The patient had been pretreated with aspirin. Unfractionated heparin was used to maintain the active clotting time (ACT) at >250 sec. The ramus branch was stented successfully. A follow-up angiogram revealed a widely patent stent with plaque shift to the LAD ostium which was protected by the LIMA graft. The appearance of the circumflex ostium was satisfactory and therefore felt not in need of intervention. The patient was extubated in the operating room [20].

2 PCI with a PVAD: The TandemHeart is a left ventricular assist device (PVAD) that can be implanted percutaneously. The device decompresses the left ventricle and maintains adequate perfusion of major organs. A transseptal puncture is performed initially, then a 21Fr cannula is advanced from a femoral vein to the left atrium. The inflow into the centrifugal pump, therefore, is oxygenated blood from the left atrium. The pump can deliver up to 3.5 L through a 15Fr or 17Fr femoral arterial cannula.

The disadvantages of the TandemHeart include procedural complexity, vascular complications and high cost. An initial transseptal puncture is required. Interventional cardiologists who have not participated in radiofrequency ablations or septal closures may have limited experience performing transseptal punctures. Many operators will prefer intracardiac ultrasound guidance to advance a large 21Fr catheter safely across the interatrial septum. The 21Fr venous cannula may cause a venous thrombus while implanted, or an iatrogenic atrial septal defect when removed. Similarly, the large arterial cannula may be associated with bleeding, traumatic arterial injury or limb ischemia. Finally, it should be recognized that there is a significant expense associated with this technology. The disposables for the TandemHeart now costs $15,000 per case. There is also an initial capital investment for the console, although that cost may be included in the disposables for institutions with high caseload.

Perhaps high-risk PCI patients should receive a provisional TandemHeart. A smaller 6Fr catheter could be placed in the left atrium and a 5Fr sheath inserted into the contralateral femoral artery. An iliac angiogram would be performed to insure that it was feasible to insert

a 15Fr arterial sheath. The TandemHeart itself would only be implanted in those who did not tolerate transient ischemia during the PCI [21].

3 Combined CABG and Carotid Stenting: In patient with symptomatic CAD and carotid artery disease, more than 30 days wait for surgery was considered a high-risk situation; similarly, a combined 30-days antiplatelet treatment after stenting with a subsequent staged cardiac procedure within the first months after carotid stenting was also considered a potentially dangerous strategy, because of the higher incidence of bleeding complications after cardiac surgery. Cardiac surgery was indicated when two or three major coronary vessels had >50% stenosis and PCI treatment was not feasible, and/or severe mitral and/or aortic valve diseases were present.

Consequently all patients were pretreated with aspirin and none of the other oral antiplatelet agents (clopidogrel, nor ticlopidine). Full dose of unfractioned heparin (UFH) was always used (usually 10,000 IU) when the introducer set was inserted in the femoral artery; an additional bolus was applied if the procedure lasted more than an hour. The carotid lesion was stented successfully. Immediately after stenting, all patients were transferred to the operating room for planned and subsequent uneventful CABG [22].

4 Closure of Atrial Septal Defect and PCI at the Same Session: In a case report from Tomai *et al.* a patient had PCI of lesions in the LAD and LCX then had closure of an atrial septal defect (ASD) with an Amplatzer. After the procedure, the patient was transferred to the ICU. One hour later, the patient developed severe shortness of breath, rales and pulmonary edema requiring intubation and mechanical ventilation. 12 hours later the patient slowly recovered with medical treatment. The problem is that closure of ASD can cause abrupt increase in LV preload and myocardial oxygen consumption, on a LV with possible transient dysfunction from slow flow. The lesson is not to perform PCI in the same time with closure of ASD. Wait until the patient hemodynamic recovers well after closure of the ASD [23].

TECHNICAL TIPS

***How to Predict and Prevent Pulmonary Edema after Closure of ASD:** After percutaneous ASD closure, the elderly patients with ASD are at risk of left heart failure. The reason is that they could have latent restrictive left ventricular dysfunction which could be responsible for an excessive rise in left atrial pressures leading to pulmonary edema. In order to detect and prevent that, temporary balloon occlusion of the defects is suggested. Prophylactic medication should enable the left ventricle to take over an increased diastolic volume immediately after ASD closure. Diuretics lower preload, thus shifting the pressure volume curve to the left; inotropic agents provide the ventricle with better contractility and can lead to enhanced diastolic suction. This concept was highly effective even in patients with an increase of left atrial pressures up to 32 mmHg, who

in our opinion were at a high risk of left ventricular failure after defect closure [24].

5 PCI and Septal Alcohol Ablation for HOCM: A patient underwent septal ablation for hypertrophic obstructive cardiomyopathy (HOCM). The baseline angiogram showed significant lesion in the LAD. After successful injection of alcohol in the septal artery, the gradient across the aortic valve improved. However the patient developed chest pain, hypotension with a systolic blood pressure of 50 mmHg, and significant ST-segment depression in the anterior precordial leads 12 hours after successful septal ablation. The patient underwent emergency and successful intervention of the LAD.

The reason is that the patients with CAD stay stable if the blood pressure is well maintained. In the case of acohol septal ablation, which is technically a provoked proximal septal myocardial infarction, patients require good blood pressure to maintain adequate flow across lesion, so PCI of the CAD lesion needs to be carried out first, before septal ablation [25].

6 PCI and CABG for Patients with Aortic Stenosis: Many elderly patients present with concomitant CAD and various levels of severity of aortic stenosis (AS). In patients with mild and moderate AS, they can undergo PCI as any other patients. The possible problem encountered during PCI is that the balloon inflation time should be short (10–15 seconds) because the patient could develop significant hypotension. Once the balloon deflates, the blood pressure would return to the prior level. The patients with AS have fixed cardiac output, this is why they already have difficulty of maintaining a decent blood pressure during any myocardial dysfunction from transient ischemia. These patients would have much more difficulty maintaining a decent BP during a short episode of acute occlusion of the index lesion. This is why extraordinary efforts should be made so that the balloon inflation time is short and stenting should be prompt if there is acute occlusion. In general, PCI should be done before the patients undergoing percutaneous aortic valve replacement [26]. These patients also undergo PCI with DES before minimally invasive aortic valve replacement because the mortality of a combined CABG and aortic valve replacement (AVR) is extremely high in elderly patients, while the mortality of minimally invasive AVR without CABG is low. The patient has PCI on clopidogrel and heparin. Right after PCI, the patient goes directly from the cardiac catheterization laboratory to the operating room for a minimally invasive aortic valve replacement [26].

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Chapter 9
Left Main

Run Lin Gao

GENERAL OVERVIEW

Left main (LM) coronary artery disease is present in 7% to 15% of patients with coronary artery disease confirmed by coronary angiography. Among the patients enrolled into the CASS Registry with >50% stenosis of the LM, the mean survival was 13.3 years for the surgical group, but only 6.6 years for the medical treatment group [1]. The survival benefit was confined mainly to patients with >60% stenosis,

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ < 10000 $US extra;**$$** > 10000 $US extra
⧖ < 10 minutes extra; ⧖⧖ > 10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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particular in the presence of left ventricular dysfunction. At this present time in the US, unprotected LM disease is indicated for coronary artery bypass graft surgery (CABG). In other parts of the world, percutaneous coronary interventions (PCI) of unprotected LM stenosis were performed with a success rate of >90%.

The long-term prognosis for patient undergoing plain balloon angioplasty (POBA) in selected cases was very poor and the actual 3-year survival for elective cases was only 36% [2]. In the bare metal stent (BMS) era, PCI of protected LM disease can be performed with a perioperative mortality and 1-year survival comparable with outcomes of repeated CABG while avoiding the potential mortality associated with a repeat operation [3]. Recently, unprotected LM stenting with BMS was increasingly performed in selected patients at many major aggressive centers in Asia and Europe [3–12]. The procedure was safe and feasible, the long-term prognosis was acceptable in highly selected patients, however, in-stent restenosis remained a major limitation to long-term efficacy and may be associated with increased long-term mortality [4,10]. In 2003 the observational studies of drug-eluting stents (DES) implantation for LM disease have reported a low binary restenosis rate and a much more favorable clinical outcomes [13,14]. In 2005 three major non-randomized studies on unprotected LM stenting using DES compared with historically matched BMS control revealed that DES were markedly superior to BMS in reducing restenosis and major adverse cardiac events (MACE) [15–17]. At Fu Wai Hospital (Beijing, China) elective PCI with DES for unprotected LM disease were performed in 220 patients from April 2003 to February 2006. Compared with the 224 patients treated with BMS before March 2003 in a Chinese registry of unprotected LM stenting [11], the Fu Wai DES group had more triple vessel disease, more LM bifurcation lesions and poor left ventricular ejection fraction (LVEF), however, the cumulative cardiac death (0.5% vs 4.9%, $p = 0.004$), target vessel revascularization (TVR) (5.9% vs 11.6%, $p = 0.034$) and MACE (9.5% vs 16.5%, $p = 0.029$) rates were significantly lower in the DES than in BMS control group during the a follow-up period of 15 months [18]. In this chapter, the technique of DE stenting for the treatment of LM disease will be discussed in detail.

INDICATIONS

CABG is still an absolute indication for unprotected LM disease (class I recommendation) [19]. Although some studies revealed the DES implantation for treatment of unprotected LM disease was feasible and safe [13–18], and a single center non-randomized study [20] showed there were no differences in the degree of protection against death, stroke, myocardial infarction (MI), and revascularization between PCI with DES and CABG for LM disease, the long-term efficacy of DES implantation compared with CABG is not confirmed by any randomized clinical trial. In the current guidelines stenting for unprotected LM disease in absence of other revascularization options is a IIb recommendation [21].

The indications for LM stenting are suggested as follows:

1. Patients with unprotected LM disease contraindicated to CABG, or when bypass surgery has very high perioperative risk (e.g. EuroScore > 10%).
2. Protected LM disease.
3. Low risk patients with unprotected LM disease, including patients with good LVEF (≥40%), isolated LM disease or LM disease combined with multivessel disease in whom complete revascularization can be obtained.

According to the studies in BMS era the prognosis of unprotected LM stenting was related to the risk of the patients for CABG. One study showed 1-year mortality was 20.2% in high risk group, while 3.4% in low risk patients [8]. Decreased LVEF is significantly correlated with cardiac death, while LVEF <40% is an independent predictor of cardiac death. Except for LVEF, mitral regurgitation grade 3 or 4, presentation with MI and shock, creatinine ≥2 mg/dL and severe lesion calcification are also independent correlates [8]. In the DES era, LVEF <40%, and incomplete revascularization are still the independent risk factors for MACE according to our experience [18]. The other unfavorable features for LM PCI are: (1) occlusion of a major coronary, (2) occlusion of a dominant RCA, (3) left dominant circulation, and (4) coexisting three-vessel disease [22].

Therefore, LM stenting, even with DES, should be reserved to highly selected patients, and only be performed in high volume centers by experienced operators.

OSTIAL AND MID-SHAFT LESION

Ostial and mid-shaft lesions of LM are good indications for LM stenting. The technique is relatively simple and the immediate and long-term outcomes are good. The lesion at the mid-shaft can be predilated and then stented as any discrete lesion in other vessels. If the stenosis is not severe, direct stenting is also technically feasible. If the shaft of LM is too short, the stent can be implanted into left anterior descending artery (LAD) across the ostium of left circumflex artery (LCX).

TECHNICAL TIPS

****Ostial LM Stenting:** For the stenting techniques of ostial lesion of LM, there are some key points to follow (Figure 9-1). (1) Select best projection to clearly show the ostium, usually A-P cranial and (or) slightly LAO cranial view are the best position. (2) A 6Fr or 7Fr Judkins left short tip guide with sidehole is often selected to engage the ostium. During stenting the guide must be completely removed from the ostium to avoid trapping the stent, but the guide must be close enough for opacification of the ostium of the LM. (3) The stent must be precisely positioned so that the ostium is adequately covered but only a short segment (around 1–2 mm) of stent protrudes into the aorta. The proximal end of the stent should be flared by balloon inflation to ensure complete proximal stent apposition.

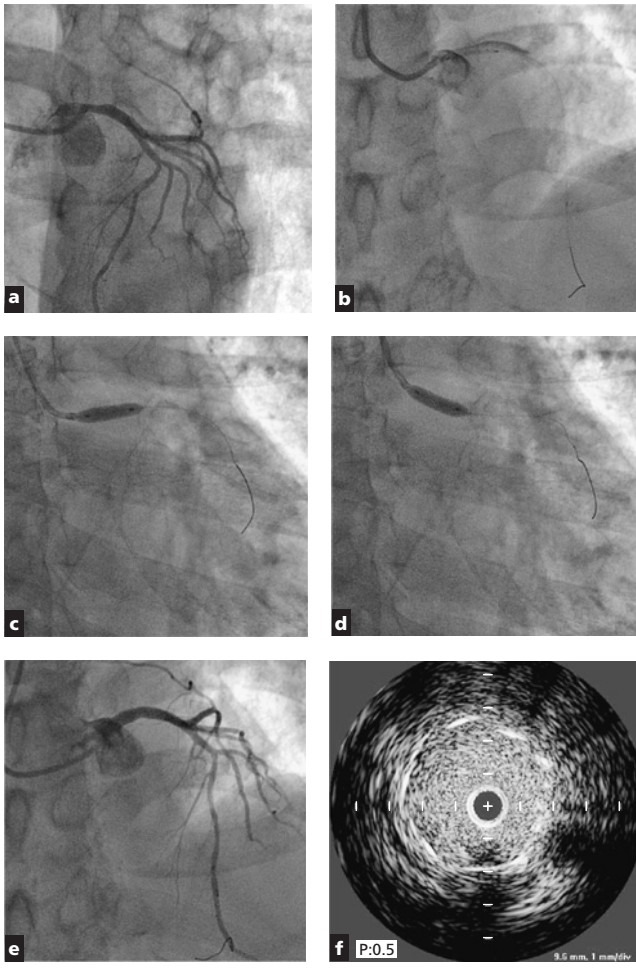

Figure 9-1 A case with ostial stenosis of LM. (a) A-P cranial view best shows the ostial stenosis of LM. (b) A 6F JL-4 short tip guide (Cordis J&J, USA) is engaged, BMW wire is placed in LM-LAD, a 4.0/12 mm TAXUS Express-2 Stent (Boston Scientific, USA) is positioned at the ostium and protruded into aorta 1 mm, the guide is then completely removed from the ostium. (c) The stent is deployed at 20 atm. (d) The proximal past of stent balloon is withdrawn to aorta and postdilatation is performed to flare the ostium of stent. (e) Angiogram shows excellent final result. (f) IVUS examination shows the stent is fully expanded and the diameter of the stent is 4.5 mm.

LM BIFURCATION LESIONS

The techniques for LM bifurcation stenting are still challenging due to technical difficulties and possible narrowing of the large side branches after stenting. Currently, the stenting techniques used for treatment

of LM bifurcation stenosis include stenting across the LCX ostium (crossover), T stenting, simultaneous kissing stenting (V-stenting), culotte stenting and stent crush technique. According to the report of Colombo *et al.* [23] in the first randomized trial using sirolimus-eluting stent (SES) for bifurcation stenting, the restenosis rate in the main vessel was significantly lower, and there were no benefit of side branch DES over balloon angioplasty in terms of side branch patency. A study by Hoye *et al.* showed the crush technique of bifurcation stenting with DES was associated with favorable outcomes for most lesions; but the efficacy appeared significantly reduced in LM bifurcation [24].

Based on these studies a crossover technique is commonly adopted in most cases with normal or diminutive (vessel diameter < 2.5 mm) LCX, if necessary provisional stenting of LCX is performed. In cases of severely disease in the ostial and proximal LCX with a vessel diameter $= 2.5$ mm, complex techniques such as T stenting, V-stenting, Culotte stenting and crush technique may be performed. The data from RESEARCH and T-SEARCH registries showed similar MACE rates between single vessel stenting and bifurcation stenting, and stenting technique was not a predictor of MACE or TVR in the current DES era [25]. According to our own experience, although the side branch (LCX) angiographic restenosis rate was significantly higher in the complex stenting than in the stent crossover group (25.0% vs 2.3%, $p = 0.003$), the overall TVR rates were not significantly different between the two groups (8.8% vs 4.1%, $p = 0.207$) [21]. These findings suggest that LM complex bifurcation stenting with DES using current techniques for patients with larger and diseased LCX may be feasible due to its large vessel size compared to that in bifurcations lesions in other vessels.

Stenting Across Ostium of LCX (Crossover Technique): The crossover technique is indicated in LM bifurcation stenosis with normal or diminutive (vessel diameter < 2.5 mm) LCX (Figure 9–2).

TECHNIQUE Stenting Across Ostium of LCX: A 6Fr or 7Fr extra-back-up (EBU) (Medtronic), XB-LCA (Cordis, Johnson and Johnson) or Judkins left guide is commonly used for engaging the ostium of LM. Two wires are advanced into the LAD and LCX, respectively. A stent is positioned from the ostium of LM to LAD to fully cover the lesion and then deployed at 12–14 atm. The wires are then exchanged, the LAD wire can be withdrawn and passed through the stent struts to LCX, and the "jailed" wire in LCX can be withdrawn and advanced to LAD. High pressure postdilatation may be applied if it is necessary. If the ostium of LCX is compromised, a kissing balloon technique can be performed. If the ostium of LCX is severely compromised, the provisional T stenting technique may be applied.

T Stenting Technique: T stenting can provide excellent results when the LCX originates at a right angle from the LM and LAD.

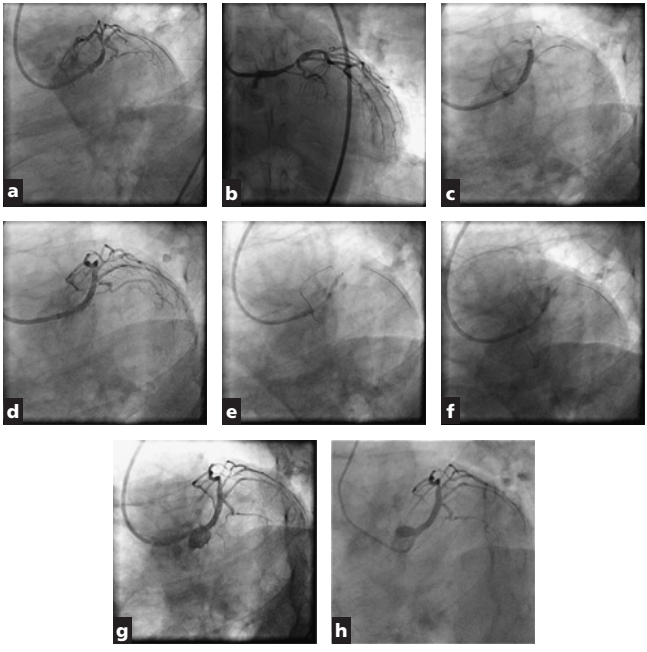

Figure 9-2 A case with LM bifurcation stenosis treated by crossover technique. Spider view (a) and RAO caudal view (b) show stenosis at LM distal bifurcation involved ostium of LAD, the ostio of ramus and LCX arteries have no significant stenosis. (c) A 7F EBU guiding catheter (Metronic, USA) is engaged, two BMW wires (Guident, USA) are placed in LAD and ramus artery, a 3.5/18 mm Cypher Select stent (Cordis, J&J, USA) is positioned from proximal LM to LAD across the ostium of LCX after predilatation with a 2.5/10 mm balloon, and is deployed at 12 atm. (d) Angiogram shows mild haziness of ostium of ramus artery with TIMI 3Flow. (e) The guidewires are exchanged, the LAD wire is withdrawn and crossed the stent struts into ramus artery and the jailed wire in ramus artery is withdrawn and placed in LAD. A 2.5/15 mm Cross-sail balloon (Guidant, USA) is advanced into ramus artery to expand the stent cell at 8 atm. (f) A 3.5/15 mm Power Sail balloon (Guidant) is placed in LM-LAD stent to perform postdilatation at 16 atm. Finally, kissing balloon postdilatation is performed at 8 atm. (g) Angiogram shows the lesion is fully covered by stent, no residual stenosis is seen, all branches are patent. (h) Angiographic follow-up at 7 months after stenting shows no restenosis.

TECHNIQUE T Stenting: A 6Fr or 7Fr large lumen guide with good support, such as the EBU, XB-LCA and Amplatz left (AL), etc., is commonly used. The wires are placed in both LAD and LCX. A stent is positioned in the side branch (LCX), and a balloon catheter is positioned in the LM-LAD. The stent in side branch (LCX) is deployed first with the proximal stent edge fully cover the origin of LCX, being sure that the proximal end of the stent does not protrude too much in the

main lumen while it does not also leave a large gap in the ostium. The wire and stent balloon are then removed, and the prepositioned balloon in LM-LAD is inflated in order to crush the possible protrusive edge in the main lumen. The LM balloon is then removed and a stent is placed over the wire and deployed in the LM-LAD across the origin of the side branch (LCX). Finally, a wire is recrossed through the LM-LAD stent into the sidebranch (LCX), and the origin of the LCX stent is postdilated to provide a large cell opening into the branch, then kissing balloon technique is performed to finish the procedure.

Provisional T Stenting Technique: Provisional T stenting is an alternative approach to T stenting.

TECHNIQUE Provisional T Stenting: The stent is placed first in the main lumen (LM-LAD) after predilatation. If after placement of the main lumen stent, the result in the side branch (LCX) is poor and appear to require stenting, a wire is passed through the main lumen stent into the side branch (LCX), a balloon is advanced to dilate the stent strut of the origin of LCX and then a stent is passed into the LCX, another balloon catheter is positioned in the main lumen. The LCX stent is then deployed with its proximal margin fully covered the origin of LCX. The prepositioned balloon in the main vessel is inflated, and finally, kissing balloon technique is performed to complete the procedure.

Crush Technique: The Crush technique is a new stenting strategy to treat true bifurcation lesions [26]. This technique is suitable for LM bifurcation patient with diseased ostial and proximal stenosis at both LAD and LCX, in whom the diameters of LM and LAD are comparable, the LCX is big vessel (diameter $\geq$ 2.5 mm) and the angle between LAD and LCX is less than 70º (Figure 9–3).

TECHNIQUE Crush Technique: A 7Fr or 8Fr large lumen guide providing strong support, such as the EBU, XB-LCA etc., is selected in order to pass two stents simultaneously. A 6Fr guide can be used in the step crush technique. Two wires are placed in LAD and LCX respectively. After balloon predilatation, two stents are advanced to the LM-LAD and LCX respectively. The proximal marker of the LCX stent must be situated in the LM at a distance of 3–5 mm proximal to the carina of the bifurcation and the LM-LAD stent must cover the bifurcation as well as the protruding segment of the LCX stent. The LCX stent is deployed first and the balloon and wire are then removed. The stent prepositioned in the main LM-LAD is deployed to completely cover and crush the protruding segment of the LCX stent against the vessel wall of the LM-LAD. A wire is then recrossed through the LM stent into LCX and a balloon is advanced in LCX to expand the LM stent struts at the origin of LCX. Kissing balloon inflation is then applied to finish the procedure.

The final kissing balloon postdilatation is very important to reduce in-hospital and long-term MACE [18, 27], whereas, the key to successful final kissing balloon postdilatation is to recross the wire into LCX

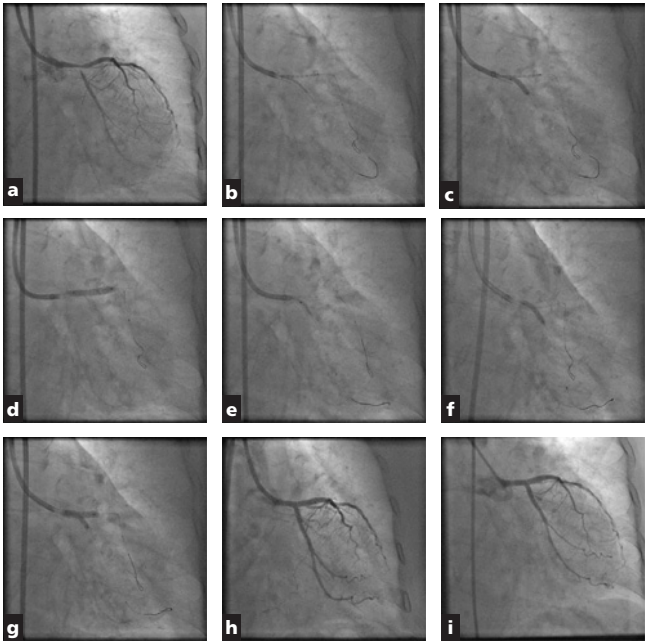

Figure 9-3 A case with distal LM bifurcation stenosis treated by crush technique. (a) RAO caudal view shows distal LM bifurcation stenosis involving the ostia of LAD and LCX, the stenosis at ostium of LCX is very severe. (b) An 8F EBU (Metronic) is engaged and two BMW wires (Guidant) are placed in LAD and LCX, after predilatation using 2.5/15 mm Cross Sail balloon (Guidant) a 2.75/13 mm Cypher Select stent is advanced into LCX and the proximal marker of the stent is situated in the LM at a distance of 4 mm proximal to the carina of the bifurcation; a 3.5/18 mm Cypher Select stent is placed in LM-LAD to cover the bifurcation as well as the protruding segment of the LCX stent. (c) The LCX stent is deplayed first at 14 atm. (d) The wire and stent balloon in LCX are removed and the LM-LAD stent is deployed to crush the protruding segment of LCX stent at 16 atm, the stent balloon is then removed. (e) A BMW wire is readvanced across the three layers of stent struts into LCX and a 1.5/15 mm Sprinter balloon (Metronic) is advanced to expand the stent struts. (f) A 2.5/15 mm Maverick balloon (Boston Scientific) is advanced to further expand the stent struts and the LCX stent at 16 atm. (g) A 3.5/15 mm Quentum Maverick balloon (Boston Scientific) is placed in LM-LAD to perform a postdilatation at 16 atm. Kissing balloon postdilatation is then performed at 8 atm. (h) Angiogram shows the distal LM bifurcation lesions are fully covered by the stents, there is no restenosis. (i) Angiographic follow-up at 6 months after stenting shows no restenosis.

through three layers of stent struts. Any wire can be used to recross, steerable soft wire such as the Balance-Middle-Weight (BMW) wire (Guidant), is most commonly used, but hydrophilic wire is also chosen by some operators. If a 1.5 mm balloon can not be advanced over the

wire into LCX, try to recross the wire through other struts which might help to pass the balloon.

The step crush technique can be performed via a 6Fr guide. A stent is placed into LCX and a balloon placed in LM-LAD. The stent and balloon are positioned as in standard crush technique. The LCX stent is deployed and then the wire and stent balloon are removed. The prepositioned balloon in LM-LAD is inflated to crush the protruding segment of LCX stent against vessel wall of the LM and then a stent is placed over the wire and deployed into LM-LAD. The wire is then recrossed into the LCX and final kissing balloon postdilatation is applied to finish the procedure.

Mini-Crush Technique: The procedure is similar to crush technique. The only difference is that the LCX stent protruded into LM is less than standard crush technique, the proximal marker of the LCX stent may be situated in the LM at a distance of 1 or 2 mm just fully covering the origin of LCX. The procedure is then continued as in the standard crush technique.

Simultaneous Kissing Stent (V-Stenting): Kissing stenting is indicated in patients with large LM lumen where the size of the LM will permit simultaneous high-pressure balloon inflation and when there is a relatively smaller lumen of LAD and LCX with ostial stenosis at both vessels (Figure 9–4).

Kissing stents are placed in both branches (LAD and LCX) of the bifurcation with overlapping of the proximal stent portions [28]. Wires are placed in both LAD and LCX and, with or without predilatation, stents are deployed in both LAD and LCX with proximal overlap. This may be done with either simultaneous stent deployment with equal pressure in both balloons or with sequential stent deployment followed by final simultaneous inflation of both stent balloons.

With this technique, a double-barreled lumen is created in the LM with a metallic carina not opposed to any vessel wall. According to our limited experiences the metal carina can be re-endothelialized in some cases, but the long-term outcomes are still to be studied.

Culotte Stenting: In culotte stenting, a stent is placed in one branch, usually from LM to LAD, with a second stent placed through a cell of the first stent into the branch vessel (LCX) with a overlapping of the proximal portions of both stent [29]. This technique is indicated in patients with similar anatomy of LM disease to crush technique.

Wires are initially placed in both LAD and LCX and predilatation can be performed if necessary, either sequentially or simultaneously. A stent is then placed and deployed in one vessel, usually LM-LAD, covering the segment proximal and distal to the bifurcation across the opposing branch (LCX). Another wire is then advanced across the deployed stent into LCX, and the jailed wire can be removed. A balloon is advanced over the wire and dilated to open the stent cell in preparation for stenting of the LCX. The first wire (to LAD) is then removed and the second stent advanced over the LCX wire and

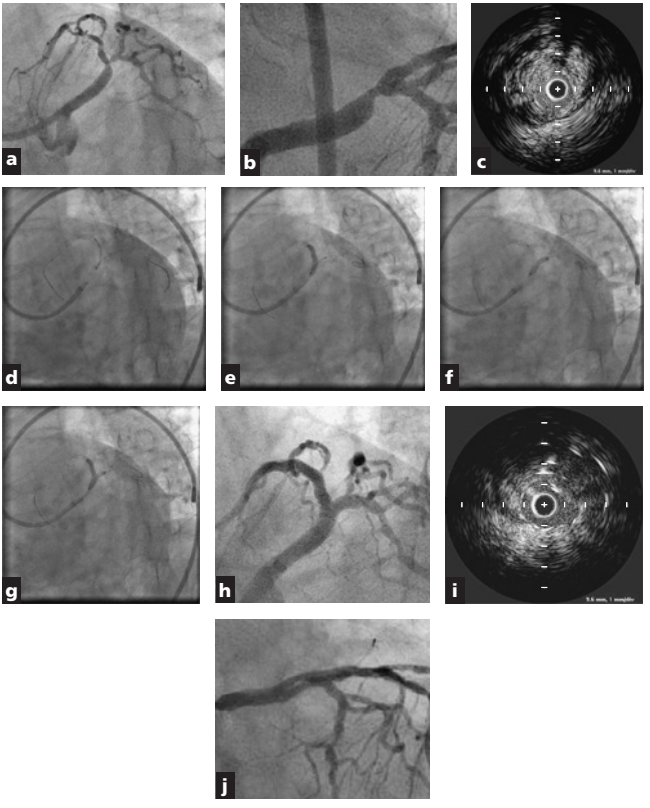

Figure 9-4 A cases with distal LM bifurcation stenosis treated by simultaneous kissing stenting. Spider view (a) and RAO caudal view (b) show distal LM bifurcation stenosis involved both LAD and LCX, the LM is big vessel, whereas, the diameters of LAD and LCX are relatively small compared with LM. (c) IVUS examination shows the plaque burdens at the distal LM, the diameter of LM is 5.0 mm, the proximal of LM is normal. (d) Two BMW wires are placed in LAD and LCX. Two TAXUS Express-2 stents (Boston Scientific) are positioned from LM to LAD and LCX with overlapping of the proximal portions to fully cover the LM stenosis. (e, f) The 2.75/24 mm stent in LAD is deployed at 20 atm and the 2.75/12 mm stent in LCX is deployed at 18 atm, sequentially. (g) Kissing balloon postdilatation is performed at 10 atm. (h) Angiogram shows excellent immediate results without residual stenosis. (i) IVUS examination after the procedure shows two stents are expended well and occupied the whole LM lumen. (j) In the 1-year follow-up angiogram, a linear translucent area is seen at distal LM which may illustrate the metal carina covered by the tissue with mild proliferated intima. The patient has no any symptoms and exercise test is negative.

deployed so as to cover the LCX lesion and widely overlap the proximal portion of the previously placed stent. A wire is then readvanced across the struts of both stents into the LAD and two balloons are advanced over both wires to perform kissing balloon postdilatation.

In culotte stenting of LM bifurcation lesion, usually the first stent is implanted into the LAD, however, if the LCX is big vessel and if there is marked angulation, the first stent can also be implanted into LCX to permit easier crossing into the opposing branch (LAD).

ADJUNCTIVE MANAGEMENT

Intravascular Ultrasound (IVUS): IVUS is an important adjunctive imaging modality for optimal intervention of LM stenosis, which can help the operator to choose the optimal size and length of the stents, to assess the conditions of stent opposition to vessel wall, and to decide if additional postdilatation should be performed. So, IVUS is strongly recommended to be used in the PCI of LM lesion.

Debulking: In the era of BMS, directional coronary atherectomy (DCA) was recommended for treatment of unprotected LM with significant lesions and large plaque burden at the distal bifurcation. DCA usually is performed prior to stenting if the lesion is suitable for debulking. Rotational atherectomy prior to stenting is also performed if the plaque has diffused superficial calcification.

In the era of DES, according to a study of BMS implantation with optimal use of DCA resulting in larger lumen gain than DES implantation alone, DES was associated with less angiographic restenosis and TLR compared with BMS [17]. There is no evidence to show debulking prior to stenting with DES can further improved the long-term outcomes. The only indication for debulking may be severe superficial calcification.

Intra-Aortic Balloon Pump (IABP): The patients with normal left ventricular function can tolerate well the global ischemia during balloon occlusion of the LM. So, IABP is not routinely used during unprotected LM stenting. However, IABP must be ready for emergent use, although it is not necessary to insert prophylactically the balloon. If the left ventricular function is poor, or if the patient has a history of MI involved inferior and posterior wall, or if the lesion is in very high risk for PCI, prophylactic IABP should be performed to prevent life-threatening hemodynamic collapse.

Adjunctive Medical Treatment: Dual antiplatelet therapy should be started before the procedure for elective LM stenting. Aspirin 300 mg and clopidegrel 300 mg of loading dose should be given at least one day before stenting. During the procedure heparin 7000–10000 u (100 u/kg) is given via the arterial sheath. After the procedure aspirin is prescribed at a dose of 300 mg daily for 3 months, followed by 100 mg daily indefinitely, and clopidegrel 75 mg daily is taken for at least 1 year. Use of glycoprotein 2b3a inhibitors is left to operator's discretion. In acute coronary syndrome and (or) intervention of lesions containing thrombus glycoprotein 2b3a inhibitors may be of greater benefit.

Follow-up: Follow-up is very important after LM stenting due to potential risks of sudden cardiac death induced by stent thrombosis or restenosis. All patients are advised to have outpatient clinic visit every 1 or 2 months in the first 6 months after stenting, every 2–3 months between 6–12 mounths, and every 3–6 months after 1 year, if without symptoms. If chest pain or any relevant symptoms developed after stenting, the patients should come to see their doctor immediately. The routine angiographic follow-up should be performed after 6 months or earlier if clinically indicated by symptoms or documentation of myocardial ischemia.

EXOTIC COMPLEX INTERVENTIONS FOR THE URBAN WEEKEND WARRIORS

1. Bifurcation Balloon for LM Acute Occlusion: Bifurcated balloon systems for angioplasty are currently under development and have had reasonable success rates reported in the literature. The Avion bifurcation RX2 balloon (Invatec, Srl, Roncadelle, Italy) is an example of such a recently developed, dedicated bifurcation system. De Man *et al*. [30] reported a 56-year-old male with cardiogenic shock and widespread ST-elevation. He had received ostial LAD and LCX stents at another institution 2 weeks prior to admission.

His blood pressure was 66/40 mmHg and his pulse rate was 50 beats/minute. His angiogram showed occlusion of both stents. Two angioplasty wires (Galeo Hydro, Biotronik, Berlin, Germany) were quickly placed in the distal LAD and LCX. A 3.0/2.5 × 20 mm Avion bifurcation RX2 balloon was successfully inflated at 10 atm at the LM carina for 20 seconds. This restored full TIMI 3Flow in both the LAD and LCX. The time from visualization of the LM occlusion to balloon inflation was no more then 5 minutes.

This bifurcated balloon system consists of a single hypotube that splits distally into two separate shafts, each of which has a balloon. As the two balloons are interconnected via a common shaft, preparation before introduction is similar to that of a single balloon. Suction of the system by hand to remove air is followed by attachment of the single hub to a single indeflator filled with 50:50 contrast–water mixture. Each balloon has a monorail configuration so that anchoring the wires together enables simultaneous passage of both balloons.

The system offers a number of advantages when compared with separate balloons. First, advancement and positioning of both balloons occurs simultaneously. This may save time when compared with two independent balloons that need to be advanced and positioned separately, and reduces the chance of one of the balloons being squeezed out of position by the other upon inflation. Second, passage of a single system is faster than two separate balloons, and the balloons are automatically in an overlap position. This allows the operator to concentrate more carefully on the patient and procedure rather than adjusting wire and balloon positions and contending with two indeflators. Third, the system dilates both branches of a

bifurcation simultaneously, therefore avoiding plaque or thrombus shift, also known as the "snow-plough" effect, into the ostium of the untreated vessel. The avoidance of plaque shift results in fewer balloon inflations and shortens the procedure. Finally, this bifurcation system passes easily through a 6Fr guide, thereby facilitating the use of other entry sites such as the radial artery. Bifurcation systems also have a number of limitations with regard to deliverability, deployment and sizing. First, it is of paramount importance to avoid entwinement of the wires. Using the kissing balloon technique, an individual balloon may pass a single or limited number of twists within the catheter or in the vessel before reaching the bifurcation, whereas the combined bifurcation system will tend to push any twists to the distal end and prevent delivery of the system. This problem is more frequent when the bifurcation lies distally in the coronary tree and the wires require extensive manipulation to pass into the appropriate branch. However, the carina of the LM is the most proximal bifurcation and does not require such extensive manipulation. Second, the simultaneous passage of two balloons, separately or as a single system, increases the stiffness and bulkiness of the system which may result in difficulty or failure when attempting to reach a distal lesion. The carina of the LM, therefore, is the ideal target for such a system. Third, the deployment of two balloons of correct size for the vessels may result in over-sizing in the section before the bifurcation. Such a problem is inherent in the use of two balloons and is unchanged with the bifurcation balloon system. Thus the disadvantages of the bifurcation balloon are, for the most part, minimized when the carina of the LM is the target [30].

2. LM PCI with Left Ventricular Assist Device (LVAD): During LM stenting in patients with severe left ventricular dysfunction, LVAD can provide more reliable hemodynamic support than IABP. Naidu *et al.* [31] reported an 80-year-old man with severe chronic obstructive pulmonary disease, chronic renal insufficiency and significant carotid artery disease presented with a 4-day history of episodic severe substernal chest pressure at rest. Echocardiography showed severe left ventricular dysfunction (ejection fraction 10%), anterior wall akinesis. moderate mitral regurgitation. Cardiac catheterization revealed a 95% distal LM stenosis that involved the ostia of both the LCX and LAD, which was also subtotally occluded, and a 50% stenosis in the mid right coronary artery. The LAD had TIMI grade 1 distal flow. Positron emission tomography revealed lateral wall ischemia and high anterolateral viability. The patient was felt to be at high risk for surgical revascularization, so LM PCI was performed with the TandemHeart percutaneous ventricular assist device (Cardiac Assist Technologies, Inc., Pittsburgh, Pennsylvania). A 21Fr in-flow cannula was advanced via the transseptal technique into the left atrium under intracardiac guidance, a 15Fr out-flow cannula was inserted into the right femoral artery and advanced to the right common iliac artery, and resultant left atrial-to-distal aorta bypass was achieved with a non-pulsatile flow rate of 3.0 liters per minute. The LAD and LCX were both wired, and stented utilizing two sirolimus-eluting

stents simultaneously deployed with a "kissing" technique. During balloon inflation, hemodynamic monitoring revealed a significant decrease in aortic pulse pressure due to diminished stroke volume. Despite the drop in pulse pressure, mean perfusion pressure was maintained and the patient remained hemodynamically stable without angina or arrhythmia. The final angiographic result shows percutaneous reconstruction of the distal LM and the proximal portions of the LAD and the LCX. The bypass cannulae were successfully removed immediately postprocedure, and hemostasis was achieved [31].

FUTURE PERSPECTIVES

Currently, unprotected LM disease can be successfully treated by stenting with DES in selected patients. However, all studies related to LM stenting are limited by the small number of patients. The randomized studies to compare the benefit and risk of the elective stenting with DES and CABG for unprotected LM disease are ongoing. We need to wait for the results to decide the value of stenting with DES for treatment of LM stenosis. Furthermore, the techniques for LM bifurcation stenosis are still challenging, optimal strategies need to be approached.

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Chapter 10
Chronic Total Occlusion

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

Chronic total occlusion (CTO) is defined as total occlusion with either known duration of more than 3 months or presence of bridging collaterals [1]. A chronically occluded artery with good collateral supply has the functional significance of a 90% stenosis [2].

In the past, the traditional factors predicting procedural success (still valid at this present time for beginners) are shown in Table 10-1. At this present time, with current level of expertise and new equipment, only target vessel tortuosity or bending at the occlusion site and severe calcification have significant impact on success or failure of percutaneous coronary intervention (PCI) for CTO.

However, not every CTO should be opened even they are technically feasible. PCI for CTO are not indicated if: (1) the myocardial area supplied by the CTO is small; (2) angiographic view of a vascular bed in a distal CTO is unclear and good flow is not expected after the recanalization of the CTO; (3) long-term prognosis is not expected for the presence of other malignant co-morbidity(ies).

Table 10-1 Factors Influencing the Success of CTO Interventions

Factors Currently Valid
1 Severe calcification (most important negative factor). If a balloon is inflated in the subintimal space with heavy calcification, the chance for perforation is higher)
2 Target vessel tortuosity or bending at occlusion
Traditional Factors Still Valid for Beginner Operators
1 Long occlusive duration or unknown duration
2 Absence of antegrade flow
3 Stump occlusion
4 Presence of bridging collaterals
5 Long occlusion length

Structural Difference Between a Recent or Old CTO: In the pathologic studies, the neovascular channels with diameter of 100–200 mcg are seen in CTO older than 1 year. These neovascular channels often are connected with the vasa vasorum in the adventitia while the channels in a younger CTO (filled by less than 1 year old thrombus) communicate with the distal lumen through the recanalization channels [3]. Knowledge of the lesion age is crucial for the operator in selecting the right type of wires. During interview with the patient, a history of myocardial infarction would help to identify the beginning of the CTO. A morning angina or angina at the start of low level activity, while being relieved with further exercise is very suggestive of a CTO. The reason for angina relief with further exercise is because the myocardium is recruiting (or opening) the collateral channels triggered by the ischemic stimulation (exercise). Review of previous diagnostic angiogram if available would help to time the CTO too.

TECHNICAL TIPS

****Scouting the Terrain and Looking for Sign Posts:** The need to scrutinize the diagnostic angiography frame by frame and from different angles can never be emphasized enough. Look for any dimple at the CTO lesion, find any potential recanalization channels or existing lumina inside the CTO. Because calcification is often an indicator of the vessel shape, it can sometimes be useful to delay the injection of contrast for a few heart beats, for a short plain cine to record a better image of the calcification. When the proximal and distal portions of the occlusion seem to slip out of alignment with each heart beat, it is a sign of tortuosity inside the CTO. It is crucial to have a clear image of the distal vasculature in order to guess the length of the CTO lesion. If the CTO is wrongly guessed to be long, an operator could set the distal re-entry point too far down and fail to realize that a long false lumen has been created.

It is important to identify the main collateral channels, and the vessel supplying and receiving them, so a supporting balloon catheter

should not obstruct the collateral antegrade flow if the balloon anchoring technique is needed. One pitfall is the presence of collateral from a conus branch to the LAD. This conus branch can be separate from the right coronary artery (RCA) ostium and be missed if only angiography of the main RCA is done [3].

ACCESS

Femoral artery access is preferred for CTO angioplasty by most operators, with utilization of 7–8Fr guides for passive support, though 6Fr guides may be considered for short occlusions or by operators skilled with active guide manipulation. Larger guides, however, provide the versatility to pass covered stent grafts more easily should a perforation occur, a complication that must be anticipated with PCI of CTOs. If there is a need for contralateral injections, a 4 or 5Fr catheter can be inserted into the contralateral femoral artery or either the radial artery [3].

GUIDES

The goal of intervention in CTO is to penetrate the total occlusion and place the wire in the distal vessel without causing intimal dissection. The choice of the guide is very important because without adequate support, it is impossible to push the wire or balloon across a CTO lesion. The principles of strong guide back-up include:

1 Large guide size (the larger the better)
2 Large opposite aortic wall contact area
3 Great (perpendicular) angle between the ascending aorta and the main distal segment of the guide (between the primary and secondary curve).

Another important factor for guide in CTO is to have a soft tip which is required when the guide is needed to be deep-seated in the proximal RCA or left circumflex artery (LCX) or left anterior descending artery (LAD) to increase backup. Usually, the left Amplatz or left extra-back-up design guide is selected for the LCX or LAD lesions and a left Amplatz shape or right extra-back-up design guide is selected for a RCA procedure. There is no role for the Judkins guide in PCI of complex CTO because they cannot be advanced deeply (and safely) into the coronary artery. The RCA guide could have side holes (if desired) to allow perfusion of the sinus node and conus branches during deep-seating. Aggressive manipulation of the guide or inadvertent deep intubation (which not infrequently occurs with the Amplatz guide) may dissect the ostial right coronary ostium (often requiring stenting), a complication that should be anticipated and recognized before wire removal [3].

TECHNICAL TIPS

***Misleading Angiographic View:** One of the reasons why the guide is not stable in cannulating the RCA is because the RCA exits the aorta on a steep angle on the sagital plane while on the anterior-posterior (AP) view, it looks normal (Figure 2-4a–b). This is the same

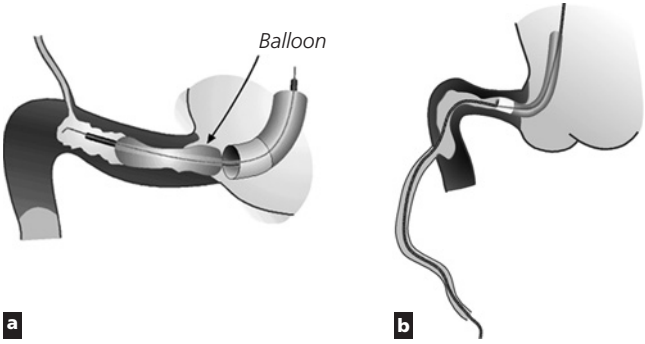


Figure 10-1 (a) When a stiff wire failed to penetrate a proximal fibrous cap, an OTW balloon is inflated in proximal to the occlusion as a support catheter for wire handling. Inflated balloon makes an extra back-up force for the wire tip to penetrate the proximal cap (b) Anchor wiring into a proximal branch also provides a better back-up force.

reason for unstable guide in the left main (LM). This is why if there is no lesion in the proximal RCA, a soft tip guide should be used and to be inserted deeply in the RCA for support [3].

****Guide Stabilization by Wire Anchoring:** Add a second stiffer wire, advance it to a side-branch proximal to the CTO lesion (Figure 10-1b). Back-up force is lower than balloon anchoring; however the wire provides a better coaxial alignment for the guide, especially when a strong support type wire is used [4].

****Guide Stabilization by Balloon Anchoring:** Advance a small balloon to a side branch located proximal to the CTO and then inflate the balloon. The inflated balloon plays a role in stabilizing the guide and provides the extra back-up force. However to prevent damage to the side-branch, careful attention must be taken during wire positioning and of balloon sizing. In this technique, a guide with a large lumen is required for accommodating the two balloons [4] (Figure 10-2).

*****Guide Deep-Seating with a Balloon:** In the attempt to cross a CTO with a (first) wire, a proximal calcified fibrous cap sometimes is hard to be penetrated. In such a situation, if there is some space in the lumen proximal to the CTO, an over-the-wire (OTW) balloon can positioned there and inflated. The OTW balloon provides a better back-up force for manipulation of the wire tip (Figure 10-1a). The guide can be deeply engaged by gently pulling back the inflated balloon system. The size of the OTW balloon must match the diameter of the segment proximal to the CTO (usually 1.25–1.5 mm diameter). Careful attention should be paid not to create a false channel in the proximal end of the CTO lesion [4].

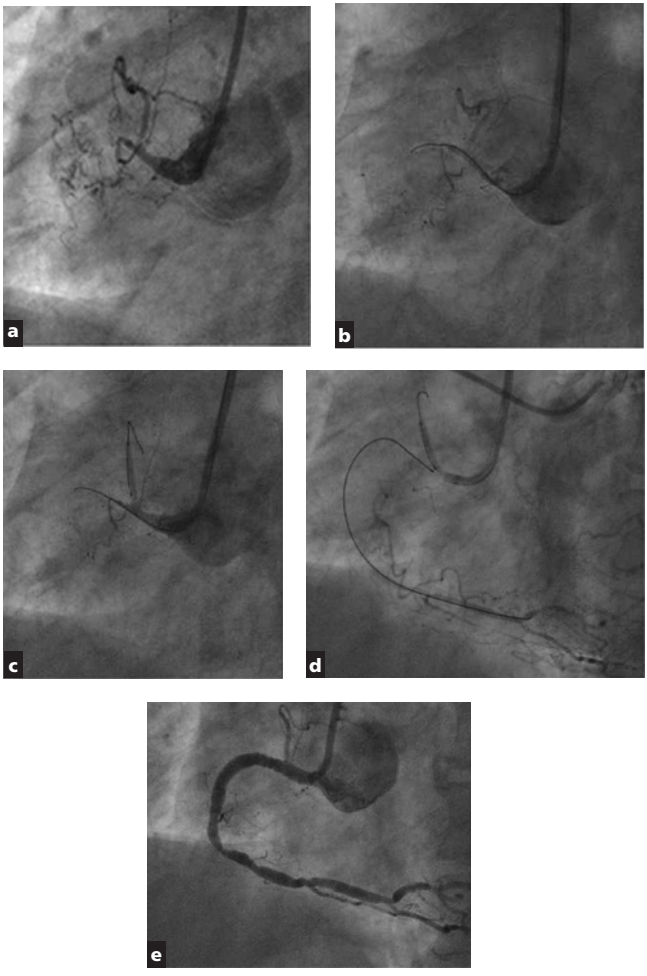


Figure 10-2 Case example with anchoring technique. The proximal RCA was completely blocked with bridging collaterals (a). A Judkins type catheter was used to prevent the damage of the RCA ostium. However, presence of tight plaque in the CTO causes unstable back-up support of guiding catheter during the wire handling. Hence, the wire could not be advanced intentionally (b). Then a 2.5 mm balloon was inserted and inflated with a low pressure in the conus branch to stabilize the guiding catheter (c). Under the use of this anchoring balloon, the wire control was improved, so that the occlusion was successfully negotiated (d). Final angiographic result after stenting (e).

****Encasing a Guide with a Long Sheath:** When working with an unstable guide, a long sheath can stiffen and support the guide, depending on how close it is advanced near the tip of the guide. The closer it is to the coronary ostium, the more back-up the guide provides.

At first, a sheath is advanced and its tip is positioned in the aortic arch and the interventional device is advanced. If it is insufficient to position the device, then the sheath is advanced further, close to the tip of the guide. As the sheath advances over the guide, it straightens the secondary and tertiary curves of the latter, causing the tip of the guide to move forward. Therefore, guides with relatively simple curves are probably safer and better suited for this technique. In order to avoid injury to the coronary ostium and dissection of the proximal segment of the coronary artery, instead of holding the guide fixed in place during advancement of the sheath, a gentle reverse traction on the guide is advised, so that its tip does not move forward. The operator should watch the guide tip continuously on fluoroscopy during this maneuver and should ensure coaxiality of the guide in two orthogonal views. Disengage the guide from the coronary ostium only after the sheath is retrieved away from it, probably to the descending aorta, with the guide fixed in place. It is possible that by just pulling the sheath away, the guide will disengage because of reconstitution of its curves [5].

****Stabilization of the Guide with Another Guide:** In a report by Saito *et al.*, a guide was inserted inside another guide to strengthen it. This is the case of a 5Fr Heartrail straight guide with 120 cm in length, whereas the 6Fr guide is 100 cm long. The 5Fr Heartrail catheter has a very soft 13 cm end portion. This soft end portion can easily negotiate the tortuous coronary artery with the minimal damage. The inner lumen of the 5Fr Heartrail guide is 0.059″ in diameter; it can accept normal balloons or stent delivery systems less than 4.0 mm in diameter. The inner lumen of the outer 6Fr guide needs to be more than 0.071″ in diameter to accommodate the 5Fr Heartrail guide. The Launcher (Medtronic), Heartrail, and Radiguide (Terumo) guide can have this large inner lumen diameter. First, the balloon or the stent is removed from the 6Fr guide. The Y-connector is also removed. Next, a 5Fr guide is inserted along the wire inside the 6Fr guide. At this point, the 5Fr guide should not protrude out of the tip of the 6Fr guide. Finally, the Y-connector is connected to the 5Fr guide and PCI could be restarted. Before the 5Fr guide is advanced into the target artery, a balloon catheter is advanced near the target lesion in the artery. Keeping a slight tension on the balloon catheter, the 5Fr guide is pushed out slowly in order to avoid the possible injury to the coronary artery by the tip of the 5Fr guide [6].

Why is a Guide Not Stable? During PCI of CTO, if the guide keeps backing out as the wires or devices are advanced, then the guide has inadequate back up. There is a need to stabilize the guide. The first corrective maneuver is to reposition the guide, put it in power position (active position) or deep seating it. If it works, then it is great.

If not, the guide should be changed for a larger one with better back-up support from the aorta (passive support), with better co-axial position (less trauma, no angle between the tip of the guide and the proximal segment of the ostial vessel segment). If the guide is ideal, then the proximal segment of the artery should be looked at. If there is proximal tortuosity, then the next step is to add a second stiffer wire. However, if the guide is not stable, adding another wire does not solve the problem. The third maneuver is by using a small balloon to anchor the guide. A long femoral sheath (70 cm), when advanced close to the tip may help to stifle the guide and prevent movement of the guide during advancement of the wire or any devices.

TACTICAL MOVE

Best Options to Stabilize a Guide

1 **No added cost** **FIRST Best Maneuver:** Put the guide in power position or deep seat the guide
2 **$** **SECOND Best Maneuver:** Add a second stiffer wire.
3 **$$** **THIRD Best Maneuver:** Use the balloon anchoring technique
4 **$** Change to a stronger guide

WIRES

STRATEGIC MAPPING

Entry Point: If there is an angiographically tapered occlusion, the tapering stump is the starting place to probe the occlusion. It frequently contains small recanalized channels (200 microns in diameter), which are potential routes for wire crossing.
However, if the stump is eccentrically oriented, there is higher risk of subintimal wire passage and vessel perforation. When there are extensive bridging collaterals (caput medusae), the chance of wire crossing is low. The reason is that these intracoronary collaterals consist of dilated vasa vasorum, which are very fragile and easily perforated.

Blunt (nontapered) occlusions can be difficult to cross, especially when a side branch arises at the site of the CTO. The concept that a plaque typically builds up in the opposite side of the origin of a side branch may help the operator to identify the true vessel course. However, the wire will often repeatedly deflect into the side branch, and it may be difficult to penetrate the proximal fibrous cap. As a last resort, dilatation of the artery with a 1.5 mm angioplasty balloon in the side branch may modify the anatomy and disrupt tissue planes to allow the wire to be redirected into the true lumen [3].

Piercing the Proximal Cap: Coaxial wire alignment along the course of the vessel and a shallow (15–20°) angled curve of the wire tip are recommended to pierce the proximal fibrous cap. A secondary bend may be placed in the wire 5–8 mm proximal to the primary bend to extend the reach of the wire if necessary without increasing the primary wire tip angle. Once the wire breaks through the proximal fibrous cap, it may be exchanged if desired for a softer wire with a slightly greater primary tip bend [3].

Traversing the Length of a CTO: Careful manipulation of the intermediate-strength wires, whose tip is bent by 45–90° at the distal 2–5 mm, can lead the wires through the loose fibrous tissues. However, if the intermediate-strength wires cannot penetrate the border between the loose and dense fibrous tissues, at this point, an OTW support system can be advanced and the wire is exchanged for a stiffer one with tapered-tip end (Cross-It 300 or 400 or Conquest). This stiff and tapered-tip wire has greater possibility for penetrating through the dense connective tissues into the distal true lumen than conventional wires [7].

Piercing the Distal Cap: Old occlusions (>3 years) typically taper at the end to form a convex structure, making penetration of the distal fibrous cap problematic. The optimal point for the penetration of convex distal fibrous cap is its center, although the newly created proximal channel often leads laterally. In curved vessels, the optimal point to attempt to perforate the distal fibrous cap is usually on the myocardial mural side, and the majority of these lesions require the parallel-wire technique to succeed.

Distal Lumen Penetration: Once a stiff wire (whether noncoated or hydrophilic) has crossed the occlusion, passed into the distal vessel and the lesion crossed with an OTW wire balloon dilatation catheter, the stiff wire should be immediately exchanged for a floppy-tipped noncoated wire placed distally to minimize the risk of distal wire perforation or dissection [8].

Conventional and Hydrophilic Wires: Hydrophilic stiff wires offer good maneuverability in tortuous vessel, however, they may not respond as well to the operator's attempt to make them to follow a precise, predetermined path through hard plaque. The hydrophilic wires are rarely used because they can go easily into the subintimal space without tactile feedback. The only exception is that the hydrophilic soft wire such as the Whisper wire in case of CTO lesions with straight micro-channel [8].

Conventional (nonhydrophilic-coated) wires are more controllable (and therefore less likely to dissect) and provide better tactile feel compared to hydrophilic wires. Noncoated coil wires tend to encounter more resistance inside the lumen than hydrophilic wires, but select

coil wires (especially the Miracle Brothers line; 0.014″ tip, available in strengths of 3, 4.5, 6, and, outside the United States, 12 g) and the Confianza (also known as the Conquest; 0.009″ tapered tip with 9 or 12 g force) have exceptional torque response even within a fibrocalcific CTO. The greater tactile feel of nonlubricious (non-hydrophilic) wires is especially important when attempting to penetrate the distal fibrous cap of a CTO and not create a false lumen. Notably, as the wire tip becomes stiffer, torque response increases, but less tip resistance is transmitted to the operator, making it easier to enter a false channel. Thus, lower-force wires are generally used initially (e.g., Miracle Brothers, 3 g), with progressive use of stiffer, more powerful wires if resistance to penetration is encountered [8]. The mechanical characteristics of the wires are listed in Table 10-2 [8].

Selection of Wire: According to the type of CTO lesion (recent or old), a wire can be selected as highlighted in Table 10-3 [7].

TECHNIQUE The Art of Wire Drilling: Once the wire crosses the proximal cap, it will be advanced slowly by the left hand while the right hand of the operator rotates it 180° back and forth. The wire with 1 mm of its tip curved to form an abrading tool is trying to grind through the lesion. If the wire buckles, it should be retracted, reoriented, then rotated rather than forced through the lesion. Constant forward pressure on the wire is more successful than aggressive tapping against the occlusion ("jack-hammering"), which does not transmit additional force [7]. Once the wire enters the distal lumen, its tip should show easy free movement and smooth retraction or advancement. If there is doubt about the intraluminal position of the wire, an angiogram of the contralateral artery may help to visualize the distal segment through collaterals. If there is no free rotation and no smooth advancement or retraction, the wire may lie subintimally or in a small collateral outside the lumen.

CAVEAT

False Lumen: Once a false lumen is created anywhere around the vicinity of a fibrous cap, the true lumen can be easily collapsed because the false lumen uses to extend (encircle) in a circumferential pattern around the arterial wall.

Curve at the Wire Tip: Large curve is good when from a false channel, it needs to locate the true channel. However, wire with large curve can make an existing false lumen larger so use the tip with the smallest curve as needed. The tapered curve is best to penetrate a CTO, however, it can be used only when the operator can be sure that it is truly coaxial before forcing it through the plaque [7].

Table 10-2 Characteristics of Wires for Crossing Chronic Total Coronary Occlusions [8]

Manufacturer	Wire	Shaft and tip diameter	Tip stiffness, g	Additional characteristics*	Recommended use(s)†
Guidant	High Torque Intermediate	0.014″	2–3		1
	High Torque Standard	0.014″	4	a	2, 3
	Cross-It 100	Shaft 0.014″ Tip 0.010″	2	b	1, 4, 10
	Cross-It 200	Shaft 0.014″	3	b	2, 3, 10, 11, 12, 13
	Cross-It 300	Tip 0.010″	4		
	Cross-It 400	Shaft 0.014″ Tip 0.010″	6	b	5, 8
	Whisper	0.014″	1	c, d	1, 4, 6, 7, 9, 10, 13
	Pilot 50	0.014″	2	c	1, 4, 6, 7, 9, 10, 13
	Pilot 150 and 200	0.014″	4 and 6	e	3, 10, 11, 12, 13
Boston Scientific	Choice PT and P2	0.014″	2	d, e, f	1, 4, 6, 7, 9, 10, 13
	PT Graphix and Graphix P2	0.014″	3–4	d, e, f	3, 10, 11, 12, 13
	Magnum 0.014	Shaft 0.014″ Tip 0.7 mm	2	g	1, 13

(*Continued*)

Table 10-2 (Continued)

Manufacturer	Wire	Shaft and tip diameter	Tip stiffness, g	Additional characteristics*	Recommended use(s)†
Asahi Intec	Miracle Brothers	0.014″	3, 4, 5, 6, and 12	h, i	1 (3 g), 2, 11 (4.5–6 g), and 2, 5, 8 (12 g); 14 (all)
	Confianza and Confianza Pro (Conquest and Conquest Pro)	Shaft 0.014″ Tip 0.009″	9 and 12	b, i, j, k	2, 5, 8, 10
Johnson and Johnson	Shinobi	0.014″	2	c, f, l	9, 10, 11, 13
	Shinobi Plus	0.014″	4	c, f, l	2, 3, 9, 10
Terumo	Crosswire EX (platinum) Guidewire GT (gold)	0.016″	2	e, m	1, 9, 10

*a, caveat: wire entrapment possible in long and hard occlusions; b, tapered tip; c, lubricious tip with nonlubricious shaft; d, difficult to shape tip; e, lubricious shaft and tip; f, poor tip memory; g, olive-shaped ball tip; h, excellent tactile feel; i, excellent torque control within occlusions and in long tortuous lesions; j, pro version has hydrophilic coating except at distal 1 mm of tip; k, pro version moves through long occlusions with little resistance; l, caveat: subintimal passage common; m, 45 and 70 degree angles.

†1, recent occlusions; 2, chronic occlusions >12 months; 3, chronic in-stent occlusions; 4, functional occlusions; 5, long and hard occlusions; 6, subtotal stenoses; 7, acute occlusions; 8, puncturing of fibrous cap; 9, tortuous anatomy; 10, intracoronary microchannels; 11, chronic occlusions <12 months; 12, occluded saphenous vein grafts; 13, recent in-stent occlusions; 14, best for parallel wiring due to excellent torque control.

Table 10-3 Rationale for Selection of Wires

Type of Lesion	Wire
<6 months	Intermediate wire (Miracle 3 g)
6 months to 1 year	Miracle 3 g (relatively hard, tapered lesion)
Recanalization channel	Choice or Whisper wire
Convex lesion, no dimple	Confianza or Cross-It XT 300–400

TECHNICAL TIPS

*****When Can Hydrophylic Wires be Used in CTO?** Soft-tipped hydrophilic wires such as the Whisper (the least traumatic hydrophilic wire) are preferred when a faint channel is visible, consistent with an intracoronary microchannel that may allow easy access to the distal lumen. Care must be taken in this circumstance, not to create a false lumen: in so doing, the operator converts a simple case into a big failure [8].

The Confianza (Conquest) Pro is a hybrid 0.014" wire that tapers to 0.009" and is hydrophilic-coated except at the tip, thus reducing the friction as the wire shaft passes down the vessel and through the body of the occlusion while theoretically retaining tactile response at the distal end. Because of its combined stiffness, hydrophilic coating, and tapered tip, this powerful wire (which is available in 9 and 12 g versions) should be reserved for experienced operators [8].

*****How do We Know the Wire is in the Sub-Intima?** When the CTO is tapered, then the wire is advanced smoothly. If the CTO ends abruptly, the wire could go down a wrong path without any resistance. The only way to know whether the wire is in the intima is to pull it back 1 to 2 mm. If the tip is in the intima, the operator would feel an unusual and unmistakable sensation of being stuck. A good rule of thumb: if any crunchy sensation is felt from the hard tissue at the wire tip, the operator can be certain that the tip is in the intima [3].

*****How to Avoid Entering the Sub-intima?** Avoid positioning the wire at the outer curve of the bend. If the wire goes subintimally on a straight portion then there are hard plaques inside the CTO which deflect the wire. If a false channel has been created then pull the wire back to the point proximal to the entry of the false channel and find a different new channel to go down [3].

*****Changing Wire in CTO:** If the first wire is removed then a new wire has to be inserted. It would take time and effort to do so. If the first wire is exchanged through a 1.5 balloon catheter, the latter can cause dissection. The best method is the parallel wire technique, discussed below.

****Where Are the Sign Posts?** After the proximal cap is penetrated, the wire must be passed through the body of the CTO to the

distal fibrous cap, which typically requires experience and reliance on knowledge of the natural course of the vessel, as well as collateral visualization of the distal vessel. Lesion calcification or occluded stents serve as guides to the vessel course. When negotiating an angulated segment in the vessel, the wire should be steered toward the inner curve to avoid extraluminal passage [3]. The presence of a tapering distal portion and chronic buildup of jagged hard plaque around the true lumen can make it extremely difficult for the wire to penetrate the CTO. Another important piece of information given by the collateral is where the collateral enters the arterial segment beyond the CTO: at its distal or mid segment. The suspicion is if there is any plaque or stenosis at the distal end, then the collaterals would enter the arterial beyond the CTO at its mid-segment [3].

TROUBLE-SHOOTING TRICKS

****Parallel Wire Technique:** In the technique called the parallel-wire maneuver or parallel wiring, when the first wire enters a false lumen, it should not be removed or advanced further to avoid enlargement of the subintimal space which easily causes collapse of the distal true lumen. The wire is considered completely in the false lumen when the resistance of the wire tip or wire movement is decreased. Then, a second wire is advanced through a 1.5 mm OTW balloon or micro-catheter in order to avoid twisting the two wires. When the second wire enters the CTO, it should be advanced parallel to the first wire while making sure it keeps the identical channel as the first. Once the second wire is in place, its tip should be led to the desired spot (Figure 10-3). The effectiveness of the parallel wire technique can be explained in two ways: (1) The first wire can occlude not only the entry into the false lumen, but also modify the arterial geometry and also become a landmark for the navigation of the second wire. (2) Thus, the second wire can more easily find the true lumen than the first and advance based upon the displacement in apparent direction of the first wire, using the first wire as a sign post of a passage to be avoided. In this situation, the tapered-tip wires are considered more adequate for the second wire than the conventional wires because they can create the channel different from the channel created by the first wire owing to their stiff and tapered tips. An example case is shown in Figure 4-2(a–h).

IVUS-Guided Wiring Technique (Bifurcation Technique): The IVUS-guided wiring technique is a useful strategy to detect the entry site of the CTO if the branch is large enough to advance an IVUS catheter (Figure 10-4). At first, the IVUS catheter is initially advanced into the proximal end of the CTO lesion and interrogates the surrounding area. Based on the initial IVUS images, the tip of the IVUS catheter is trying to pinpoint the central area of the main lumen at the beginning of the CTO lesion. This is the most suitable and desired position for wire entry. This location of the tip of the IVUS is identified exactly by coronary angiograms so the tip of a drilling wire can be positioned.

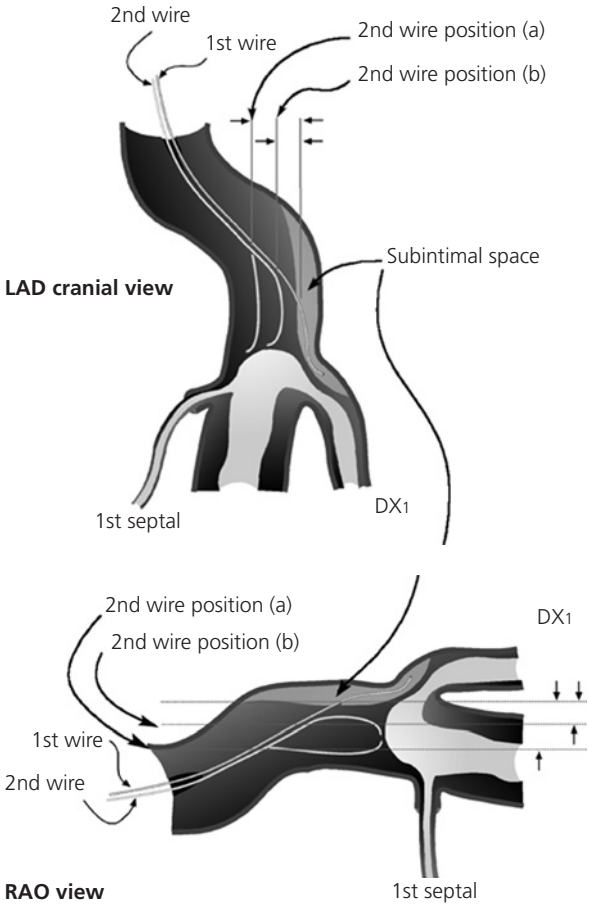


Figure 10-3 Concept of parallel wiring technique. This is a scheme of parallel wiring technique in a LAD-CTO case. As shown in the images, the first wire slips into the subintimal space in the pericardial side just before the distal true lumen. In this situation, subintimal space is easily enlarged if the wire is manipulated again, possibly causing the collapse of distal true channel. To prevent further expansion of subintimal space, the first wire must be left there as an indicator for the second. The second wire should be stiffer than the first wire. The second wire should be carefully advanced towards the distal true end under the marker of the first wire and must be positioned between (a) and (b). Finally, the distal fibrous cap should be penetrated from this position.

Then, the operator seeks a dimple at the entry with careful wire manipulation. This IVUS interrogation also helps to examine the composition of the plaque and its consistency (hard or soft etc.) at the entry site as well as to check the entry point of the first wire where and how it goes into the (unwanted) subintimal space [9] (Figure 10-5).

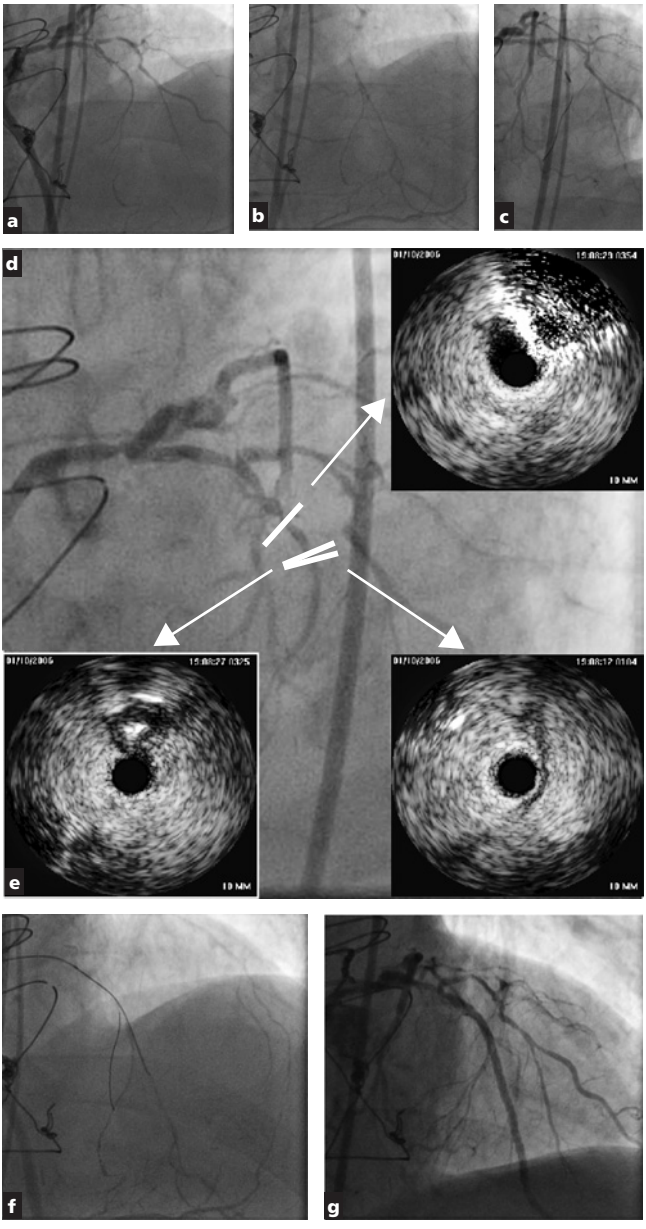


Figure 10-4 Case example with IVUS guidance. Although the LAD was completely blocked around mid-portion, it was hard to identify the entrance of a CTO despite the contra-lateral injection that was performed (a, b). Then an IVUS catheter was inserted to the septal branch (c). IVUS image was then easily identified the CTO entrance (d, e). This confirmation was also important to use a stiff wire to penetrate the tight proximal fibrous cap (f). Final angiographic result after stenting (g).

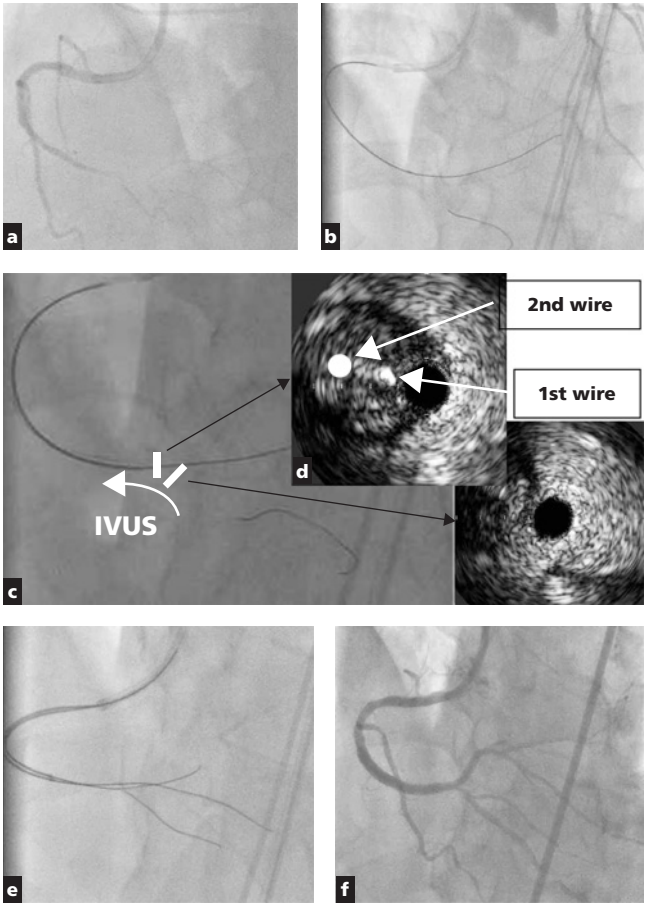


Figure 10-5 Case example with IVUS guidance. The first attempt to revascularize the distal RCA-CTO (a) failed. In the second attempt, the first wire (intermediate) easily went out of the true channel (b). An IVUS image from the proximal small branch (c) clearly showed that the entry point of the first wire to distal RCA was too close to the branch (d) so that it easily advanced into the subintimal space. The correct position of entry point for the second wire is in the center of the obstructed true channel which indicated the opposite direction to the branch. Therefore, the course of next wire was intentionally changed from the CTO entrance towards the opposite direction to the branch angiographically. Then this wire easily got into the distal small branch (e). Final angiographic result after stenting (f).

IVUS-Guided Wiring Technique (False Lumen Technique): IVUS can differentiate a true lumen from a false lumen by identifying the presence of side branches (which arise only from the true lumen) and intima and media (which surround the true lumen, but not the false lumen). Similarly, IVUS can confirm when the wire has reentered the true lumen from the false lumen [10]. With IVUS-guided wiring (false lumen) technique, the IVUS catheter is advanced through the first wire into the subintimal space. Enlargement of the subintimal space by wiring often causes collapse of the distal true lumen; therefore, this problem could not be observed or verified by contralateral angiography from the artery supplying collaterals. However, the IVUS image clearly shows the cross sectional information which is useful to guide the second wire into the true lumen. Stiff wires (Confianza or Miraclebros 12, Asahi Intecc, Japan) should be used as the second wire to penetrate the true channel. Figure 10-6 illustrates the IVUS-guided wiring maneuvers in the false lumen technique. This technique sometimes requires balloon dilatation in the subintimal space to deliver the IVUS catheter, however, it should never be performed when wire PERFORATION from the subintimal space is already detected. An 8Fr guide is required to perform the parallel wiring technique under IVUS guidance. After successful wire crossing, multiple stenting is mandatory to fully cover the enlarged subintimal space. By using IVUS, we can recanalize some CTOs that were initially unsuccessful under angiographic guidance [10] (Figure 10-7).

Retrograde Wiring Approach Including the CART™ Technique: In certain cases, antegrade wiring fails to cross the CTO lesions. However, retrograde wiring helps to penetrate the true lumen via a large collateral vessel connecting the distal vessel. Retrograde wiring was first performed via a bypass graft and initially reported in 1990 [11]. In this approach, a wire is advanced in a retrograde fashion through the best collateral channel of an appropriate opened artery to the distal end of the CTO.

The CART™ Technique: At first, a wire is advanced in an antegrade manner, trying to cross the CTO. When resistance is felt at the wire tip or the wire movement decreases, the wire is deemed entering the subintimal space. Leave the wire in this position. A second wire is advanced in a retrograde fashion through the collateral channel under the support of a balloon or a microcatheter which helps to prevent channel injury and facilitate manipulation of the wire. This second wire is positioned at the distal end of the CTO and tries to penetrate in a retrograde fashion from the distal true lumen into the subintimal space at the CTO site. After advancing a small balloon (1.5–2.0 mm) over the retrograde wire into the sub-intima, the balloon should be inflated in the sub-intima and also on the course from this sub-intimal space to the distal end of the CTO. To keep this sub-intimal space open, the deflated balloon should be left in place. As a consequence, the two dissections created by the antegrade wire and the retrograde balloon lie in the sub-intimal space at the CTO site.

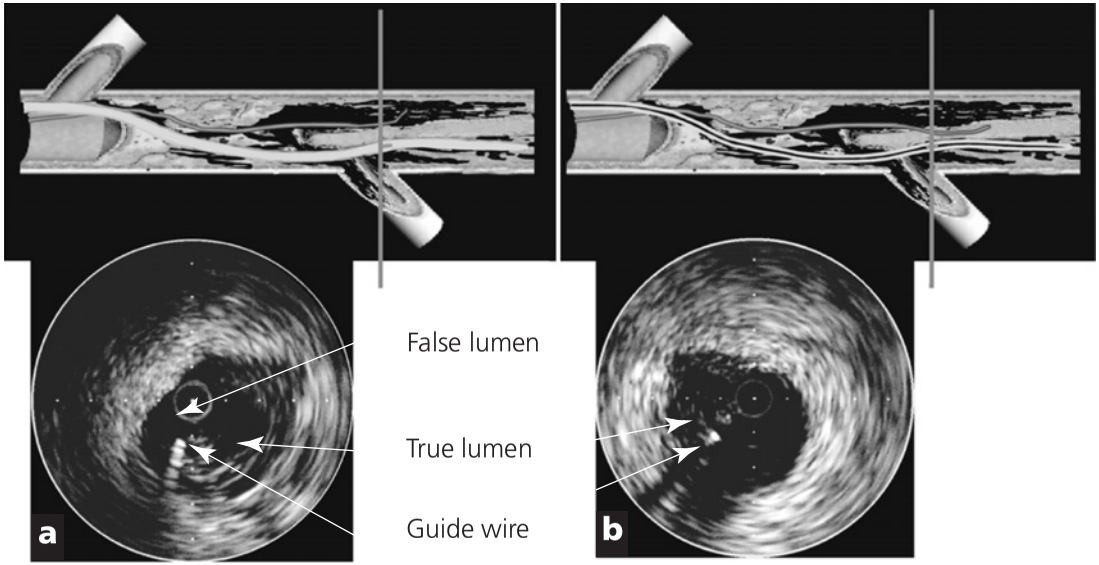


Figure 10-6 Concept of IVUS-guided wiring in false lumen technique. (a) The distal true channel is completely collapsed by the enlargement of subintimal space. The IVUS catheter is intentionally inserted into space to examine the cross-sectional information. The IVUS image clearly indicates the collapsed true channel and the wire in expanded subintimal lumen. (b) Under this IVUS guidance, it can be performed to lead the wire into the collapsed true channel.

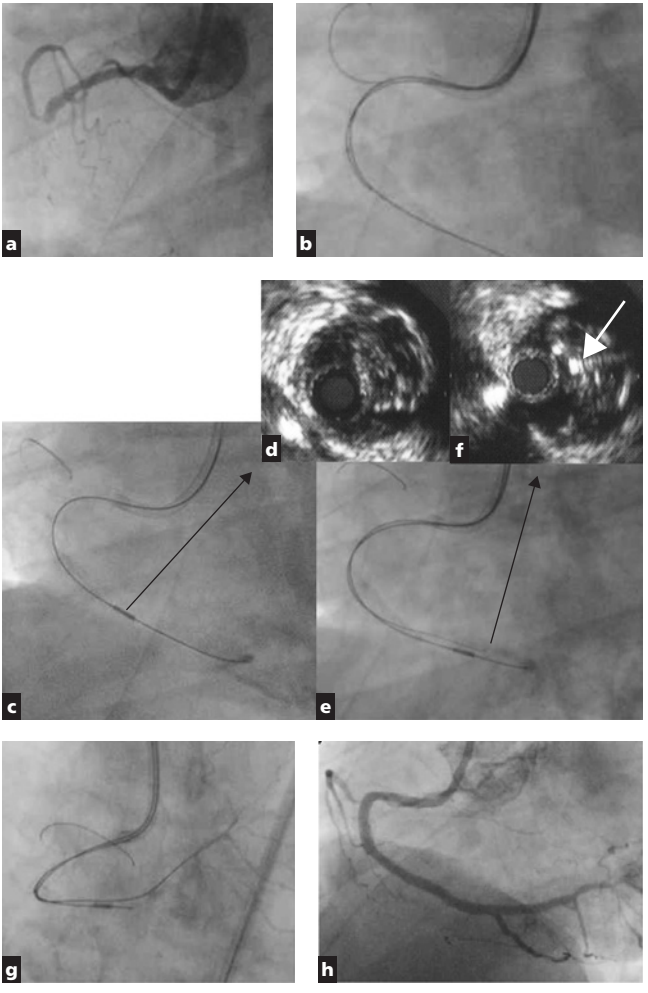


Figure 10-7 Case example with IVUS-guided wiring in false lumen technique. RCA had a very long occlusion including 3-years-old instent reocclusion (a). Parallel wiring technique using stiff wires could not provide successful wire crossing (b). An IVUS catheter was advanced through the wire in the false channel (c). The image clearly showed an expanded subintimal space and a collapsed true channel (d). Then a tapered stiff wire was delivered under the IVUS guidance penetrating the true channel from subintimal space and finally this procedure was succeeded (e, f). After the wire was carefully advanced to the distal true lumen (g). Final angiographic result after multiple stenting (h).

Thereafter, the antegrade wire is advanced further to the distal true lumen along the deflated retrograde balloon. Then ballooning and stent implantation can be performed over the antegrade wire [12]. The main advantage of this technique is the minimization of subintimal tracking only through the length of the CTO lesion. Hence, this technique is completely different from the STAR technique [13], and a better long-term outcome after the implantation of drug-eluting stent can be expected. Figure 10-8 shows a case successfully treated by retrograde approach combined with the CART™ technique.

TECHNICAL TIPS

*****Which Channel do We Use for Retrograde Wiring?** The retrograde wiring requires the use of collateral channels that can be one of the following routes: a bypass graft, epicardial collaterals, or an intraseptal channel (septal collateral). Despite this is a diseased graft, we can use it as a channel for retrograde approach. However, a completely occluded saphenous vein graft should not be used for this technique. In some rare cases, an arterial graft could be used. CTOs of RCA often receive collaterals from the LCX via epicardial collaterals. When an epicardial collateral channel is large and straight enough to accommodate a balloon or micro catheter through it, it can be used as a channel for retrograde approach (Figure 10-9). However, these epicardial channels sometimes have tricky configurations such as corkscrew aspect so that the wire crossing is difficult and poses higher risk of perforation. If the channel is the only collateral supplying the area beyond the CTO, this retrograde wiring procedure may provoke "ischemia at a distance" once the collateral flow is disrupted. Therefore careful wire handling and monitoring are mandatory. More recently, the retrograde approach using septal collateral channel has been introduced to treat CTOs of RCA or LAD [14]. In these CTOs, the anatomical course of the principal collateral is predominantly (80%) a septal connection (between septal perforators and the left anterior and posterior descending arteries) [15]. If perforation or rupture of these septal branches occurs, the risk of cardiac tamponade would be low. Compared to epicardial collaterals, the septal channel is also a shorter route to the recipient vessel and has a less tortuous, corkscrew-like configuration (Figure 10-10).

*****Retrograde Wiring:** The key to the success of this method is to study the diagnostic images carefully and to choose a less tortuous septal branch. It is crucial to give more importance to its course (tortuous or straight) rather than its size (including its distal portion). However, we have to avoid complications such as minor myocardial infarction (MI) due to collateral branch occlusion, ostial injury caused by an aggressively deep seated guide, and tamponade due to vessel perforation. Furthermore although it looks like a tiny channel, it can be dilatable by using a small balloon (1.25–1.5 mm) with very low pressure (up to 2 atm) to advance a catheter through it without major complication [14]. If the collateral vessel has lesion, then PCI of the collateral vessel should be done. If not, a different collateral route

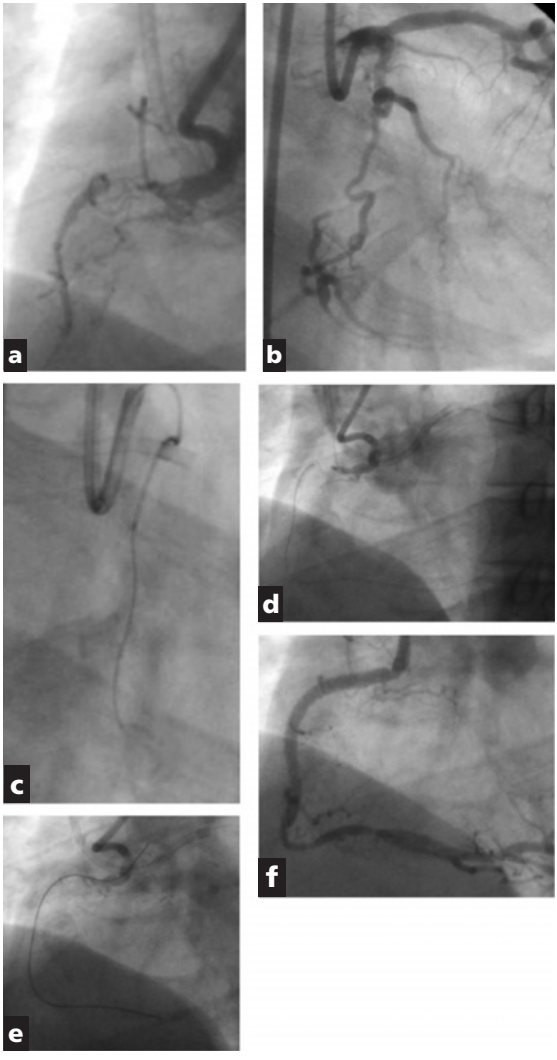


Figure 10-8 Case example with retrograde approach via epicardial collateral. The first attempt to open the proximal RCA-CTO failed due to the presence of severe bending in the proximal CTO (a). There was big enough collateral from the LCx to advance a catheter through it (b). So from the beginning of the second procedure, retrograde approach was performed. A hydrophilic soft wire was advanced through the collateral with a microcatheter (c), and it easily reached the distal true lumen of the CTO (d). Then the wire was changed to a stiff wire and carefully advanced into the occlusion, and finally it negotiated the blockage retrogradely (e). Final engiograhic result after stenting (f).

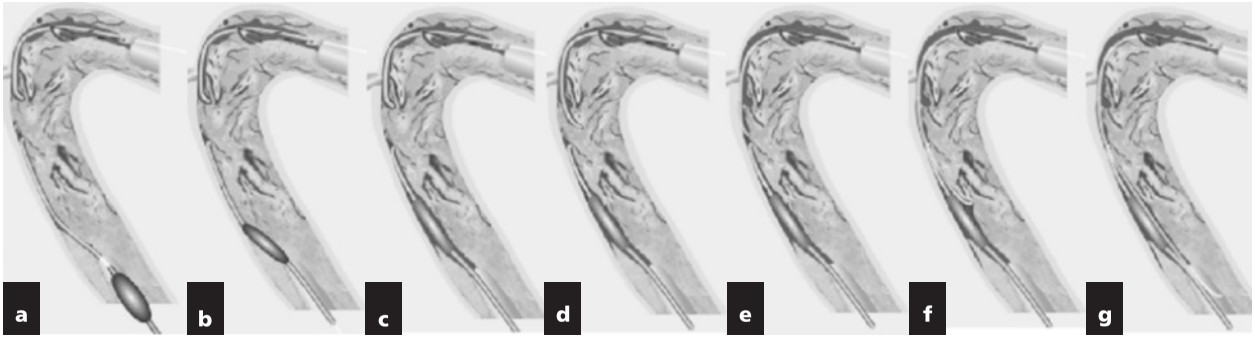


Figure 10-9 Concept of CART™ technique. Once the antegrade wire goes into the subintimal space, another balloon system is advanced retrogradely if possible (a). The retrograde wire is intentionally advanced into the subintimal space (b), and the balloon is inflated to expand the space (c). Then the antegrade wire is advanced (d). Basically the subintimal space is easily connected so that the antegrade wire easily goes into the retrograde direction created subintimal space (e, f) and negotiate the occlusion (g). The notable mechanism of the CART technique is the creation of the subintimal space limited with the CTO lesion.

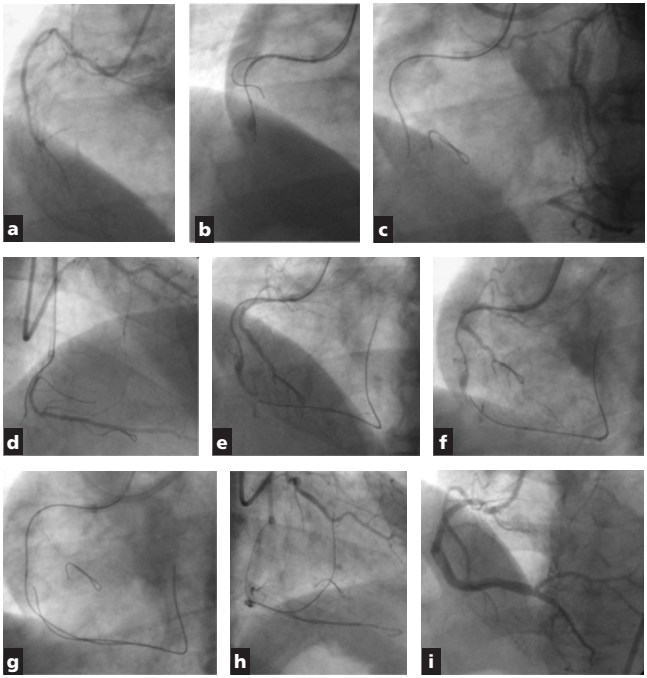


Figure 10-10 Case example with retrograde approach via septal collateral combined with the CART technique. The first attempt to negotiate the RCA-CTO failed. It was a very long occlusion reaching the distal bifurcation (a, c). At the second attempt, the parallel wiring technique under anchoring wire was also unsuccessful and easily made a subintimal space (b). Then retrograde approach was conducted. After the selective contrast injection in a proximal septal branch, a hydrophilic soft wire was carefully advanced through the septal branch and finally it reached the posterior descending artery (d). A 1.25 mm OTW balloon successfully negotiated the septal channel after dilatation of the channel by balloon to change the wire to a stiffer one. Then the wire was intentionally advanced into the subintimal space in a retrograde fashion (e). After balloon inflation in the distal part of CTO (f), the antegrade wire easily went into the same subintimal space (g) and was successfully advanced into the posterior descending artery (h). Final angiographic result after spot stenting in the proximal and distal part of CTO (i). In this case, the mid-portion of CTO did not require stenting because the IVUS examination confirmed large lumen area.

has to be selected or the retrograde approach should be abandoned. Although over-dilatation of septal branches may cause perforation to the cardiac ventricles, these connections rarely require additional treatments. A hydrophilic soft wire (Fielder, Asahi Intecc, Japan) should be used to negotiate the collateral channel with the support of a micro catheter (Transit, Cordis, MI) which minimizes wire kinking. On reaching the distal end of the CTO, the retrograde wire is often exchanged

for a stiffer wire to be advanced into the CTO lesions. Once the distal cap is pierced, sometimes the retrograde wire advances easily through the entire CTO lesion to the proximal true lumen of the CTO. However, there are some risks of creating a subintimal space extending to the proximal true lumen of the CTO which may cause a disastrous event when the CTO is located in the proximal part of the left coronary artery system (e.g. dissection of the left main). Although the antegrade wire can be advanced into CTO forward to reach the retrogradely advanced wire (kissing wire technique) [16], it is not easy to connect each to the other in the true channel.

*****Wire Manipulation:** The key to the success of this method is to VERY GENTLY manipulate the retrograde soft (first) wire, the transit catheter and the stiffer (second wire, exchanged through the transit catheter in order to cross the CTO from its distal end) in order to avoid damage to the collaterals.

CAVEAT

Prevention and Management of Septal Artery Rupture: When pushing the transit catheter, if the septal wire shows excessive kinking, then the wire has to be withdrawn because there is high chance of septal perforation. Once there is small perforation from the septal to the right or left ventricle, then observations can be made. However, if there is contrast spilling into the epicardial artery, then distal occlusion of the perforation is needed. The easiest way is to embolize the distal perforation with subcutaneous fat tissue.

PCI in Distal CTO Lesion and Moderate Ostial Lesion: In general, it depends on individual circumstances. Sometimes, it may be better if the proximal lesion is stented first so the wire handling can be facilitated, complications in the proximal segment be prevented and complications in the distal segment can be managed more easily or prevented.

CROSSING CTO WITH A BALLOON

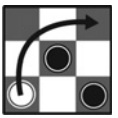
If the distal vessel is still not opacified, an OTW balloon may be advanced again across the CTO, the wire removed, and a small amount of contrast carefully injected distally via the wire lumen. Such distal contrast injections should be reserved as a last ditch effort, as they will either demonstrate intraluminal position of the catheter or worsen a subintimal dissection, typically ending the case. After the occlusion is crossed and dilated with the OTW catheter, the true dimensions of the CTO may be appreciated and subsequent angioplasty and stenting performed with appropriate-sized devices. It

should be noted that chronic low flow spasm in the distal vessel is common after CTO recanalization, so often large and repeated doses of intracoronary nitroglycerin or other vasodilators are needed for the true reference vessel diameter not to be underestimated.

When a Balloon Cannot Cross a CTO: Crossing a CTO with a balloon catheter may be difficult and, in up to 5% of cases, impossible, typically due to the presence of extensive fibrocalcific plaque, especially when the guide support is suboptimal. Methods that may be considered for such difficult situations include: (1) deep guide intubation; (2) introduction of a second wire into a branch proximal to the occlusion to increase the support of the guide; (3) introduction of a wire in the true lumen adjacent to the first wire as a buddy wire or to increase the dimension of the channel (after which the second wire is removed); (4) use of larger and more supportive guides; (5) inflation of an angioplasty balloon either in the main vessel or in a side branch to stabilize the guide; and (6) use of debulking devices. Though most studies have not demonstrated a role for debulking of CTOs to reduce restenosis, excimer laser or high-speed rotational atherectomy or recently with the Tornus catheter may allow balloon passage or expansion of otherwise nondilatable CTOs.

The tactical decisions of crossing a CTO lesion with its preferred sequence of different techniques: from simple to complex, from user-friendly to labor-intensive and from low-cost to expensive, are listed below.

TACTICAL MOVE



BEST Options for Balloon to Cross a CTO

1. **No added Cost** **FIRST Best Maneuver:** Deep-seat the guide
2. **$** ⧖ **SECOND Best Maneuver:** Advance a second wire along the first wire to straighten the artery or to widen the lumen for balloon passage
3. **$** ⧖ **THIRD Best Maneuver:** Advance a second wire to a side branch to anchor the guide
4. **$** ⧖⧖ **FOURTH Best Maneuver:** Change to a stronger guide
5. **$$** ⧖ **FIFTH Best Maneuver:** Inflate an OTW balloon in the proximal segment to anchor and deep seat the guide
6. **$$** ⧖⧖ ♦♦ **Debulking Atherectomy**

The TORNUS Catheter: The Tornus (Asahi Intecc, Aichi, Japan) catheter has been developed as a novel penetration OTW catheter for severe coronary artery disease. The Tornus catheter consists of three parts: the main shaft with surface coating, the polymer sleeve, and the hub connector. The polymer sleeve prevents leakage of blood. The main shaft is a coreless stainless coil that is right-handed lay (clockwise). Eight stainless wires are stranded in the coil. The outer

diameter is 0.70 mm (2.1Fr). The inner diameter is 0.46 mm and is suitable for the 0.014″ guidewire. Since the coil is stranded with eight stainless wires and a length of 150 mm from the tip is tapered, the Tornus catheter has desired flexibility and torquability. It can cross through the severe stenosis easily with counterclockwise rotation along the guidewire because the shaft is stranded clockwise. When the tip of the shaft is stuck, excessive rotational force may cause shaft breaking at the distal part. In order to avoid this breakage, particularly in the coronary artery, the shaft at the proximal end in the sleeve is tapered to make it weakest dynamically. Even if the Tornus catheter is rotated with excessive force when the tip is stuck, this weakest area at the proximal end will be destroyed first. The part of the sleeve that covers the weakest part of the shaft is thickened. The profile of the tip is 0.62 mm (1.9Fr) in diameter, and it is made of stainless-platinum alloy, which provides the tip strength and radiopacity. If the tip of the catheter does not advance through the lesion by manipulation, it is sometimes required to release the catheter from rotational force in order to avoid the breakage of the shaft [17].

Contralateral Injection: The information from the contralateral injection is very important to monitor the wire movement. However, if there is interference between an interventional guide and a diagnostic catheter then one should come from the radial approach [3].

TECHINICAL TIPS

***How to Use Less Contrast:** To reduce the amount of dye, contralateral injections may be performed through an end-hole catheter inserted distally into the artery to perform superselective angiography. This technique can restrict the amount of contrast to less than 1 cc per injection. Superselective injection with a transit catheter deep near the collateral branch will help to cut down the amount of contrast used [3].

CAVEAT

When to Stop: Variables that favor terminating an unsuccessful but as-yet uncomplicated procedure include reaching the upper limits of radiation exposure (e.g. 60 min of fluoroscopy time) or dye consumption (typically 600 cc in a nondiabetic patient with normal renal function; much less in patients prone to contrast nephropathy); the creation of a large false lumen, especially if adventitial staining is present; shearing off of collaterals resulting in loss of visualization of the distal vessel; and excessive patient or operator fatigue. A second try after a failed CTO attempt (usually performed 6–8 weeks later to allow vessel healing) may be successful in >50% of patients, especially when the mode of failure is understood and a feasible alternative strategic approach has been formulated.

CAVEAT

The Final Angiogram: Once the procedure is considered finished, a good angiogram needs to be done in order to check all arteries and branches for perforations. Pericardial effusion could develop many hours after successful PCI. Intramyocardial hematoma could develop and propagate further compromising antegrade coronary flow or obstructing right or left ventricular flow (See Chapter 17, Complications).

ANTITHROMBOTIC AND ANTICOAGULANT

Procedural anticoagulation considerations are similar for PCI of nonoccluded stenoses, except that direct antithrombins (DTI) and glycoprotein 2b3a inhibitors (GPI) are usually avoided because of the problem with anticoagulant reversal when there is perforation. GPI may be administered before angioplasty once the wire has successfully crossed the lesion and confirmed to be intraluminal. Similarly, the initial heparin bolus may be reduced to achieve an activated clotting time of approximately 200 sec until the wire has successfully crossed the occlusion, after which additional heparin should be administered before dilatation to achieve an activated clotting time of 250–350 seconds (if a GPI is not used). However, perfect anticoagulation is needed to prevent thrombus, activated clotting time (ACT) should be checked every 30 minutes [8].

CTO INTERVENTIONS ASSISTED BY MSCT

To date, there are no multicenter randomized studies conducted to demonstrate the significance of the multi-slice computed topography (MSCT) on the procedural outcomes of CTO interventions. There are some anecdotal reports describing the usefulness of MSCT on such complex interventions.

The Advantages of the MSCT: MSCT is an imaging modality which could outline the shadow of an angiographically-invisible and totally occluded artery. The possible information provided by MSCT includes:

1 Visualization of an occluded artery and collateral vessels
2 Length and diameter of the vessel in the CTO lesion
3 Morphology of a CTO lesion.

A volume rendering (VR) image is useful to characterize an overall picture of the coronary arteries around the heart. The VR function is used to identify the location, tortuosity, calcification, and bifurcation of a CTO lesion. A slab maximum intensity projection (MIP) image can be one of the most useful adjunctive diagnostic images for CTO-PCIs. The

location of plaque, the level of calcification, and the length of lesion on the slab MIP image are closely related to the information provided by a coronary angiography. The lesion morphology and wire direction are also detected by this function. In addition, a multi-planar reconstruction image is qualitatively identified a route of vessels and a location of calcification and remodeling. It is helpful to predict the degree of calcification.

In a typical example that favors the use of MSCT on a CTO-PCI, the collateral vessel could not be clearly seen on baseline angiography. However, MSCT identifies a short and soft occlusion without severe calcification which was only located in the distal artery. An intermediate wire was easily advanced into the distal artery within a few minutes after reviewing the MSCT images. As of any technique, preprocedural MSCT is not mandatory for every CTO cases. However, it can be useful in identifying the actual occlusion length and in visualizing the collateral vessels. So MSCT should be done when the occlusion length and/or the vessel course is unclear by conventional angiography. Further studies are necessary to demonstrate the effectiveness of MSCT on CTO-PCIs.

TAKE HOME MESSAGE

During PCI of a CTO lesion, after careful examination of diagnostic angiography, we usually use the antegrade approach using an intermediate wire (Miraclebros 3, Asahi Intecc, Japan) with an 8Fr guide through the femoral approach. When a better back-up force is required, the wire or balloon anchoring technique is considered. Contra-lateral injection is mandatory except in case of bridging antegrade collateral. Check ACT every 30 minutes. When the first wire fails to cross the lesion, it should be exchanged for a stiffer one (Miraclebros 6, 12, or Confianza family, Asahi Intecc, Japan). When a wire creates a false lumen, the parallel wire technique is used. If that technique fails to work, the IVUS-guided wiring technique or retrograde approach is implemented. However, in re-attempted cases, the retrograde approach is performed right away from the beginning. If the IVUS guided wiring or retrograde approach is not feasible or not working as expected by the operators, the procedure should be stopped before (rather than after) complete collapse of the distal true channel by an enlarged subintimal space. When wire perforation from subintimal space is observed, the procedure should be stopped and appropriate treatment should be given to control the extravascular bleeding.

The percentage of special techniques used in our institution is listed in Table 10-4.

Successful recanalization was achieved in 209 cases (88.6%) with no major adverse event (death, emergent bypass surgery, Q wave myocardial infarction). The average procedural time was 114 ± 48 minutes.

Table 10-4 Technique used for PCI of CTO

Parallel wiring technique (40.3%)
Anchoring technique (16.9%)
IVUS guided wiring in bifurcation technique (18.2%)
IVUS guided wiring in false lumen technique (13.6%)
Retrograde approach (16.9%)

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Chapter 11
Ostial Lesions

Szabo Szabo, Thach N. Nguyen

*Basic; **Advanced; ***Rare, exotic, or investigational.
$, 100.00 $US extra; **$$**. 100.00 $US extra
⌛, 10 minutes extra; ⌛⌛. 10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

Ostial lesions are defined as lesions within 3 mm of the ostium of the vessel at the aorto–ostial or branch–ostial junction. These lesions have unique pathological and morphological/angiographic features resulting in a challenging subset with inferior outcomes when compared to non-ostial lesions. Ostial lesions tend to have higher calcium and fibrous tissue content and increased elastic recoil tendency [1,2]. There is also increased intimal hyperplasia after stenting. As in other complex lesion subsets, the goal of the operator providing contemporary treatment is to deliver an optimally deployed drug eluting stent (DES). Although the DES appears to successfully tackle this unfavorable milieu and provide a high rate of procedural success and reduced rate of restenosis, the available data are limited. Small observational series by Lakovou *et al.* suggests that sirolimus-eluting stent (SES) implantation results in a target lesion revascularization (TLR) rate in single digits [3].

However, even in the DES era, ostial location appears to be correlated with poorer outcome. The location of these lesions pose inherent challenges to the operators due to: limited angiographic views, highly variable ostial anatomy, unstable guide support, accentuated cardiac motion and usually significant myocardium at jeopardy. Despite these disadvantages, precise stent placement has to be achieved to avoid geographic miss or the compromise of branches involved. It is imperative to exclude the frequently present catheter-induced ostial vasospasm before embarking on intervention.

CAVEAT

Strategic Concerns for Percutaneous Coronary Interventions (PCI) of Ostial LAD and LCX:
Interventions of ostial left anterior descending (LAD) or left circumflex (LCX) lesions pose many concerns which are listed below. (1) The balloon or stent can impinge on the origin of the non-dilated artery and obstruct flow. (2) If there is acute occlusion, there could be significant jeopardy to cardiac function or survival. (3) Significant difference of vessel size of the left main compared to the LAD or LCX. (4) Dissection at the proximal LAD and LCX could extend in a retrograde fashion into the left main (LM) segment. (5) Stent restenosis at the proximal end could result in restenosis of the LM. (6) Antegrade and retrograde embolization into the other artery (from LAD to LCX or vice versa, from LAD to LM and systemic embolization (etc).

Guide Selection: Preferred guide catheters are those that provide stable alignment with the axis of the vessel at the ostium without having a tendency to dive into the vessel. The interventional device, especially the stent, should be maintained in a stable position while

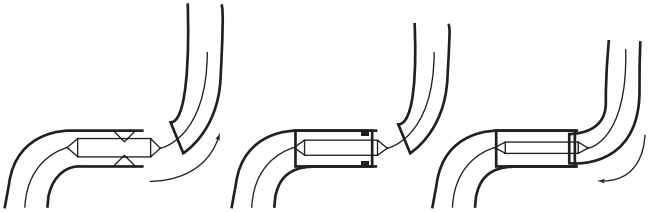
the operator moves the guide in or away from the ostium. Usually the Judkins type guides (some with short tip) best achieve this and in general the Amplatz type guides are avoided. For some right coronary artery (RCA) ostia with high take-off and RCA-vein grafts a multipurpose guide sits best.

TECHNICAL TIPS

***Guide Selection and Positioning:** Because of the proximity of the lesion to the ostium, the guide should not be fully engaged or deep-seated. The usual Judkins guide may provide sufficient back-up with coaxial alignment without aggressive intubation. A guide with side holes would help, however, waste of contrast media through side holes would mask the exact location and the severity of the ostial lesion. As long as coaxial alignment is maintained, the interventional device can be advanced, positioned and checked with the tip of the guide positioned just outside the ostium.

****Disengagement of Guides:** Once the balloon, undeployed stent or atherectomy blade, is properly positioned, the guide is then gently withdrawn 1 or 2 cm into the aorta, so the balloon or the stent is not inflated or deployed inside the guide. Gentle forward pressure on the device catheter or low-pressure balloon inflation (1–2 atm) may help to maintain proper balloon position while the guide is retracted. Ideally a "waist" is seen in the center of the exposed balloon during pre-dilatation confirming appropriate positioning (Figure 11-1). Frequent test injections should be done to verify that the tip of the guide does not inadvertently engage beyond the ostium nor the devices move outside its intended area.

****Re-Engagement of Guides:** After deployment of a stent, follow up angiogram, post dilatation or further devices might need to be delivered. Even with appropriate guide alignment it could be difficult to enter an ostially deployed stent. Therefore it is helpful to re-engage the stent first, before the final angiogram, by using the stent delivery balloon to "rail in" the guide tip (see Figure 11-1). Once the stent is deployed the deflated balloon is left in place or can even be advanced slightly. Then while applying traction on the balloon catheter, the guide tip can be advanced into the ostium of the stent to achieve perfect alignment without damaging the stent. When the balloon is to be removed, traction on the guide should be applied first, so pulling the balloon out will not move the guide forward into the ostium. Once the coronary angiogram is done, the guide can be backed out by slight forward push on the wire. This maneuver should be done while monitoring the tip of the wire, as throughout the entire procedure to avoid distal perforation of the coronary artery. It is important to handle the deployed stent with care while performing post-dilatation or intravascular ultrasound (IVUS) to avoid damage, dislodgement or even extraction of the stent due to entanglement of the struts with wires, balloon or IVUS tip.



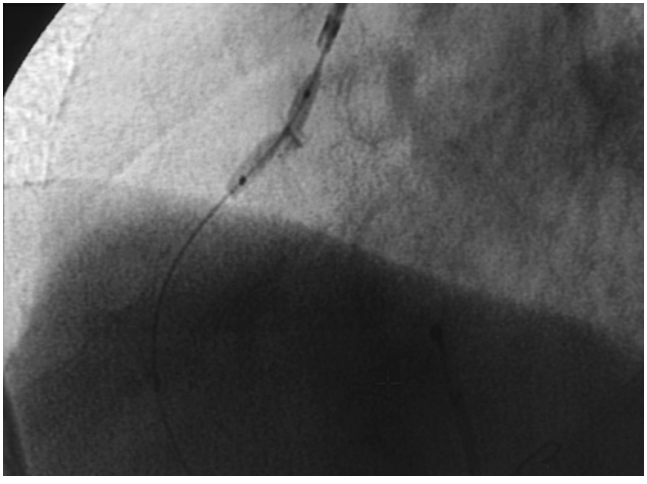


Figure 11-1 The balloon is positioned across the lesion. Next the guide is withdrawn into the aorta to expose the balloon. After inflation and stent deployment, the deflated balloon can be used to stabilize and "rail-in" the guide.

*****Two-Guide Technique:** Correct positioning of a stent at an ostial lesion can be difficult due to poor visualization once the guide is backed out of the artery to allow deployment. The simultaneous use of a diagnostic catheter allows optimal visualization of stent position, while maintaining a stable guide position well away from the stent. In a case report, by Lambros *et al.* which illustrates the technique of using a second diagnostic catheter to enhance visualization of the lesion during stent positioning, a patient underwent successful intervention for an aorto–ostial lesion in the saphenous vein graft (SVG) to LAD. An 8Fr Judkins right (JR) guide was stable and provided good support. However, with the guide in a position that would allow adequate visualization of the lesion, the tip of the guide sat on the lesion, precluding perfect stent deployment. When the guide was withdrawn slightly, visualization was poor and the proximal balloon remained within the guide, risking movement during deployment. Then a 6Fr diagnostic catheter was introduced from the opposite site and engaged, providing excellent vision for stent positioning [4].

Wire Selection: Usually intermediate strength wires provide adequate support as significant part of the wire is anchored far in the distal vessel. Avoid hydrophilic wires and monitor the wire tip during the entire procedure to prevent distal perforation as maintaining stable wire position tends to be difficult during device delivery and due to guide movement.

LESION PREPARATION

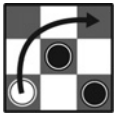
Although significant data suggests the feasibility of direct stenting in non-ostial lesions, the operator should have a very low threshold to predilate an ostial lesion. Since precise positioning is paramount, without predilatation the operator will have to "struggle" with the resistance of the lesion with further limitation of angiographic landmarks and risking watermelon-seeding. It is vital to recognize a poorly yielding or "non-dilatable" lesion in this heavily calcified subset before stent deployment.

TECHNICAL TIPS

****Watermelon-Seeding Effect of Aorto-Ostial Lesions:** In patients with ostial lesion, while inflating the balloon, sometimes the balloon migrates proximally or distally. So the balloon should be inflated more slowly: 1 atm at a time and the balloon catheter is gently retracted to keep the balloon from migrating distally. Even so there are cases of persistent watermelon-seeding when a buddy-wire or cutting balloon will help to solve the problem, especially in in-stent restenosis.

Undilatable Lesion: Because of increased rigidity and fibrous tissue in the ostial lesion, complete expansion of non-compliant and high pressure balloon is needed prior to stenting. If the balloon cannot be fully expanded with a pressure higher than 18 atm, then cutting balloon angioplasty or rotablation should be considered. Although routine debulking and/or cutting-balloon preparation is not recommended especially in the DES era, these devices have an important role in selected patients to assure optimal stent deployment. As in all difficult lesion subsets IVUS is very helpful to characterize the lesion and facilitate optimal deployment. When facing an undilatable lesion, which is the best option?

TACTICAL MOVE



BEST Options for Ostial Lesion Predilation

1. **No added cost FIRST Best maneuver:** Inflate the predilation balloon to maximum
2. **$ ⌛ SECOND Best maneuver:** Add a buddy wire and perform focused-force angioplasty [5]
3. **$$ ⌛⌛ THIRD Best maneuver:** Cutting balloon angioplasty
4. **$$ ⌛⌛💧 FOURTH Best maneuver:** Rotational atherectomy

STENT POSITIONING

It is frequently difficult to identify the proximal end of an ostial lesion with certainty. The first reason is that the guide cannot be engaged

deeply into the ostium or the lesion itself. The guide has to be seated outside the aorta so during injection, part of the contrast would enter the ostium and part would swirl under and along the curve of the coronary sinus, thus masking the exact location and the severity of the ostial lesion. The second reason is because there is a device across the lesion during positioning a balloon, stent or interventional device, so the minimal flow at the ostium may not be able to delineate the lesion and the location of the ostium. Different landmarks such a fleck of calcium in the aortic wall may also help to identify the entry point of the ostium. The presence of a second wire curving the coronary sinus may help to prevent deep engagement of the guide (Figure 11-2). Once a stent is deployed, it is also difficult to evaluate possible geographic miss of a short ring-like ostial segment that is left uncovered by the stent. A small guide can pass through it without causing any ventricularization of pressure and a coronary angiogram may not detect any abnormality because the ostial segment is covered with contrast from back flow. The matter can become worse if the ostial segment is filmed in a non-orthogonal projection. If viewed from an angled projection, the lesion cannot be seen because the adjacent contrast-filled vessel segments are projected over the short napkin ring size uncovered segment and mask it.

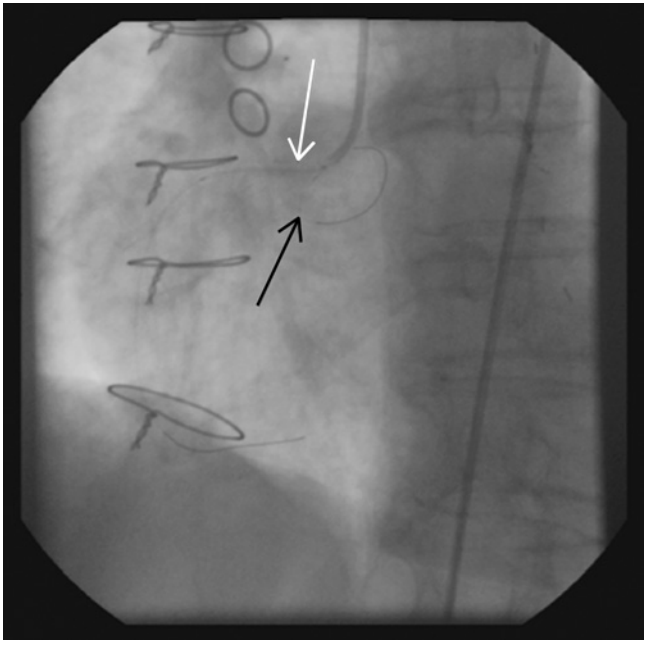


Figure 11-2 A second guide wire in the aorta helps to outline the sinus of Valsalva (black arrow). It can also be used to anchor the stent at the ostium (white arrow).

TECHNICAL TIPS

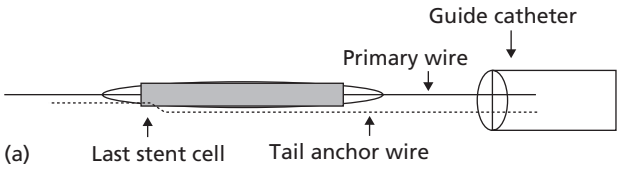
****Double Wire to Position Ostial Stent:** Difficulty in positioning the guide during stenting of especially an ostial RCA lesion is sometimes made easier by use of a second steerable, soft-tipped wire placed in the aorta just below the coronary ostium. This second wire stabilizes the guide outside the coronary artery and prevents the guide from moving deeply into the guide. It also defines the junction of the coronary artery and aorta, an important landmark for stent placement (Figure 11-2).

*****Double Wire in Ostial In-Stent Restenosis (ISR) Lesion:** Engaging a chronic ostial stent, as in a case of restenosis, could be challenging. One report describes using a wire threaded through the protruding stent strut in order to stabilize the guide position. Then a second wire is engaged and crosses the lesion [6]. This should be performed with caution as stent avulsion/migration has occurred during these manipulations [7]. It is also plausible that a DES could be at risk of migration for several months after implantation due to the lack of neointimalization or late malapposition.

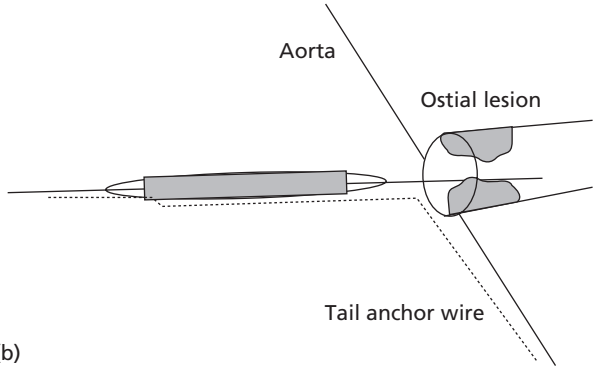
*****Stent Positioning with an Anchor Wire:** A second wire is placed into the aorta with the soft distal tip outlining the ostium. The second wire is back-loaded through the last proximal strut of the stent. As the stent is advanced across the lesion, this second wire anchors the last proximal strut outside the aorto-ostial junction Figures 11-2 and 11-3). In the case of ostial side branch, this technique is used to perfectly position the proximal end of a stent at the ostium of the LAD [8,9]. In case of LCX stenting, the anchor wire will be positioned in the LAD.

Stent Deployment: Once the stent is positioned across the lesion, the guide is slightly withdrawn while maintaining some forward push to expose the balloon stent fully from the guide (Figure 11-1). Inflating the balloon partially in the guide can lead to balloon rupture. It is often helpful to use some landmarks, like a speck of calcium in the aorta, during these manipulations. Contrast injections at this point might help by outlining the sinus of Valsalva. The patient can be asked to hold their breath. If significant motion is present, low-pressure inflation (1–3 atm) will provide some stabilization and allow final correction of the stent position, if needed [10]. The operator should be careful during these manipulations as well as when moving the stent catheter

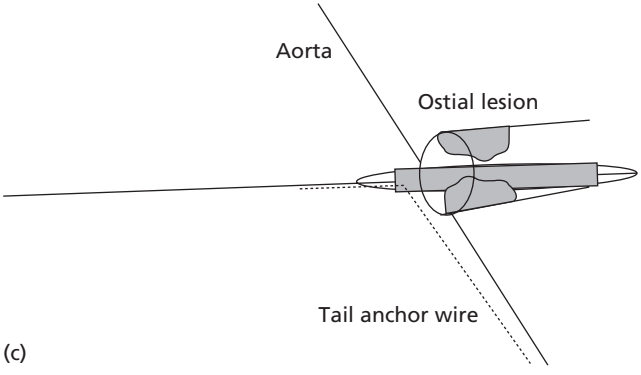
Figure 11-3 Using an anchor wire. (a) Outside the guide, the anchor wire is inserted under the last stent cell. (b) The stent is advanced into the guide over both the primary wire and the anchor wire, which resides outside the ostium in the aorta. (c) The stent is advanced into the ostial lesion. (d) The stent advancement is stopped by the anchor wire, and then, deployed at low atmospheres. The anchor wire is then removed, and the stent is deployed at high atmospheres. Modified from Kern MJ *et al.* [9].


Guide catheter
Primary wire
Last stent cell
Tail anchor wire

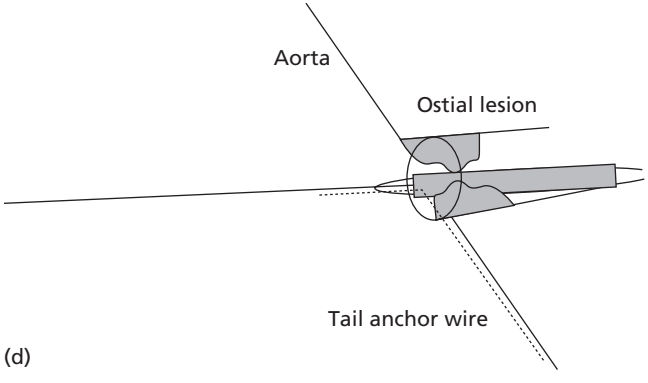
(a)


Aorta
Ostial lesion
Tail anchor wire

(b)


Aorta
Ostial lesion
Tail anchor wire

(c)


Aorta
Ostial lesion
Tail anchor wire

(d)

back and forth in these calcified lesions, especially during withdrawal back into the guide as stripping of the stent off the balloon can occur at this specific moment and location. It is recommended to position the stent 1–2 mm protruding into the aorta to prevent recoil of the lesion at the stent edge. The operator should avoid using very short (<12 mm) stents to provide adequate anchoring of the stent and lesion coverage distally. Appropriately sized (1:1 ratio) stents should be deployed at appropriately high pressures (⩾12 atm) to ensure optimal apposition. Usually a second higher pressure inflation is performed with the stent balloon slightly retracted allowing full apposition at the ostium but avoiding distal edge injury (Figure 11-1). Routine post-dilatation with larger balloons and "flaring" of the ostium of the stent, although often performed, is probably not necessary and should be balanced against the risk of potential retrograde dissection of the aorta.

OSTIAL SIDE BRANCH STENTING

It is not feasible and probably not necessary to stent every ostial lesion in various clinical and anatomical subsets, especially in the case of smaller side branches. Cutting balloon angioplasty in these situations appears to be a safe and effective option [11].

TECHNICAL TIPS

***Can We Have Perfect Position of a Stent for Sidebranch Ostial Lesions?** A stent can be positioned and deployed perfectly only if the angle between the side branch and the main branch is close to 90 degrees. If the angle is more or less than 90º (not perpendicular) then the proximal end of the stent can protrude into the main branch lumen or stay too far inside the side branch thus leaving a part of the ostium uncovered. In these situations, also depending on the anatomy of the parent vessel, one has to consider the various bifurcation techniques.

****Stent Pull-Back Technique:** In branch-ostial lesions, inflate a small balloon at low pressure in the parent vessel then pull back the stent to the ostium of the side branch while being sure that the balloon in the main brach is slightly compressed. This technique would result in less ostial miss and need for additional stents [12].

*****Cutting Balloon Angioplasty of a Jailed Side Branch Ostial Lesion:** When dilating an ostial lesion with plain balloon angioplasty (POBA) fails, then a cutting balloon could be used with extreme caution. In a case reported by Hongo *et al.*, repeated failures with POBA happened when dilating an ostial lesion through the side struts. Then a cutting balloon was inserted, with the proximal segment well inside the main vessel lumen, and the balloon was inflated with excellent results [13].

EXOTIC COMPLEX INTERVENTIONS FOR THE URBAN WEEKEND WARRIOR

1. PCI of Ostial ISR Lesion Through Side-Strut: In a case report by Burstein *et al.*, despite attempts with multiple guides and wires, the protruding stent in the ostial RCA ISR could not be engaged in a coaxial fashion, suggesting deformation of the intra-aortic segment of stent struts [14]. After failing to achieve coaxial guide alignment, a 6Fr Amplatz right guide was placed on top of the protruding stent segment and a Whisper® wire (Guidant, Indianapolis IN) was advanced to the distal RCA through the struts of the aorto-ostial stent. A 1.5 × 14 mm Maestro balloon (Abbott Laboratories, Abbott Park, Illinois) was inflated and widened the side-struts. This was followed by consecutive balloon dilatations using 2.0 × 14 mm, 2.5 × 14 mm, 3.0 × 14 mm and 3.5 × 14 mm Maestro balloons inflated up to 18 atm. At the end, a 3.0 × 16 mm Taxus™ stent (Boston Scientific, Natick, Massachusetts) was successfully advanced through the widened side-struts and deployed at 16 atm with 1–2 mm of the proximal stent segment protruding into the aorta for complete coverage of right coronary artery ostium. The fully-expanded Taxus stent displaced the intra-aortic segment of the previously placed stent inferiorly and created a new entry site into the artery, with excellent angiographic outcome.

In this technique, stents with open-cell design are more suitable, as the stent architecture permits unimpeded widening of the stent cells for up to 4 mm without damaging the stent struts. Stents with a closed-cell design permit only limited expansion of the stent cell, and the passage of larger balloons and stents may cause fracture of stent struts.

2. Rotablation for Stent-Jailed Side Branch Stenoses: In a report by Sperling *et al.* 30 consecutive unselected patients who underwent rotational atherectomy for jailed side branch stenosis underwent 38 procedures, including rotational atherectomy to treat 32 distinct ostial lesions in stent-jailed side branches. Rotational atherectomy was performed in the side branch and frequently in the parent vessel using a "stepped burr" approach over a rotational atherectomy guidewire (RotaWire™ Floppy or RotaWire™ Extra Support, Boston Scientific Corp., Natick, Massachusetts). Following debulking with rotational atherectomy, adjunctive balloon angioplasty was performed on all lesions. Frequent revascularization of the parent vessel was also performed with the use of the simultaneous balloon inflations in the parent and side branch vessels (i.e., "kissing balloon" technique) in the majority of cases. Rotational atherectomy was not performed in the setting of acutely stent-jailed side branch stenosis. Despite the high procedural success rate, repeat revascularization was common in the study. The TVR rate of 44.8% undoubtedly reflects the unfavorable impact of variables such small vessel size, ostial location and high incidence of previous revascularization of the treated side branches [15].

CAVEAT

Rotoblation Through Stent Struts: An important technical consideration when using rotational atherectomy to treat side branches covered by stents is the importance of ensuring that the side branch has been well dilated through the side-struts. This may facilitate passage of the burr and may reduce the potential of burr entrapment. Another concern is the embolization of metallic particles during rotablation. However, there were no cases of periprocedural myocardial infarction following angiographically successful revascularization of side branches with rotational atherectomy in our series, suggesting that rotational atherectomy through the sides of stents can be performed safely [15].

CAVEAT

Extraction of Stent by Cutting Balloon at Ostial Lesion: The cutting balloon is designed with blades mounted along its length. During inflation, the blades are protruded outwards and exposed. Then during deflation, there is a mechanism of gradual rewrapping the balloon with multiple wings over the blades. During this process of rewrapping, there is possibility of the creation of a recess in the form of an acute angle formed by the balloon and the blades. This recess can get stuck into the stent struts and prevent withdrawal of the cutting balloon. If the cutting balloon is pulled hard enough it could pull with it the stent or part of the stent. When the cutting balloon is withdrawn, the pulling force applied on the balloon catheter will not be parallel to the vessel axis. An anchoring point will then be formed at the stiff proximal edge of the blade and the soft proximal balloon catheter. This anchoring point can be easily caught on the proximal stent struts, especially at the RCA orifice, as an almost 90º curve is formed by the proximal RCA and the aortic wall. In order to prevent this problem, after the deflation, the cutting balloon should be advanced first, then withdrawn gently. Other possibilities [3] include: stent strut fracture by the microblades, or an under-expanded stent with inadequate strut apposition is another possible catching point [7].

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Chapter 12
Acute ST Segment Elevation Myocardial Infarction

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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Exotic Complex Interventions for the Weekend Urban Warriors

1. PCI in patients with lM compressed by an aortic disssection
2. PCI of a reconstructed prosthetic LM

   Critical thinking: Contrast leakage into the ventricle? Is it wire-induced perforation or impending rupture?
3. Primary PCI in STEMI after resuscitated cardiac arrest

GENERAL OVERVIEW

Acute ST-segment elevation myocardial infraction (STEMI) is usually caused by thrombotic occlusion of a major epicardial coronary artery in the absence of adequate collateral flow from other coronary territories. Prompt, complete and sustained recanalization of the infarct-related artery (IRA) with restoration of normal myocardial perfusion reduces the infarct size, preserves left ventricular function and reduces mortality. In comparison to thrombolysis, primary percutaneous coronary interventions (PCI) significantly reduce mortality, reinfarction and stroke rates [1]. This is true not only for patients admitted to PCI centers but also for patients transferred from non-PCI available hospitals or directly from the field by ambulance emergency teams [2,3]. However, it is very important to emphasize that every effort should be made to minimize the time delay from the first medical contact to the first balloon inflation regardless of patient location [4].

Indications: Any patient with chest pain coming to the hospital within 12 hours from onset and with an electrocardiogram (ECG) suggestive of ST-elevation acute myocardial infarction (AMI), can be offered emergency PCI. A catheterization laboratory team should be available to proceed with primary PCI within 45 minutes of activation [5]. It is reasonable to withhold thrombolytic therapy and transfer the patient for primary intervention if the D2B (door-to-balloon) time can be achieved within 2 hours [6]. The indications of primary PCI are listed in Table 12-1.

Urgent Coronary Angiography: If possible, the IRA should be determined from a 12-lead ECG. First, different views of the presumed non-IRA should be taken with a diagnostic catheter in order to estimate the extent of coronary artery disease supplying the non-infarcted

Table 12-1 Indications for Primary PCI [5]

1	Symptomatic AMI in less than 12 hours
2	AMI 12–24 hours with continued chest pain
3	Cardiogenic shock <24 hours
4	Thrombolytic failure within 12 hours of chest pain, especially if anterior MI
5	Suspected re-occlusion after thrombolytic therapy
6	Non-diagnostic ECG (left bundle branch block, Paced rhythm, ischemic ST-T changes), with positive enzymes, refractory angina, or hemodynamic instability/heart failure

myocardium and assess the collateral flow to the distal segments of the IRA. Angiography of the presumed IRA may be performed with a guide so PCI can be started promptly. The culprit lesion should be clearly identified and characterized in terms of diameter, stenosis, angiographic evidence of thrombus and epicardial thrombolysis in acute myocardial infarction (TIMI) flow [5] (Figure 12-1). During the diagnostic angiogram all the major vessels and their large branches should be accounted for in order not to miss the IRA (Figure 12-2). There is no need for routine left ventriculogram. If there is a need to assess the left ventricular (LV) dysfunction, a pigtail catheter can be inserted into the LV and LV end diastolic pressure can be measured.

Identification of High Risk Patients: Based on clinical evaluation, hemodynamic measurements (heart rate and rhythm, arterial pressure and LV end-diastolic pressure if necessary) and coronary anatomy, high risk patients should be immediately identified.

In the emergency room, if the patient presents with a heart rate of less than 100 bpm and a blood pressure more than 100 mmHg, their in-hospital mortality is very low [7]. The high risk patients are listed in Table 12-2.

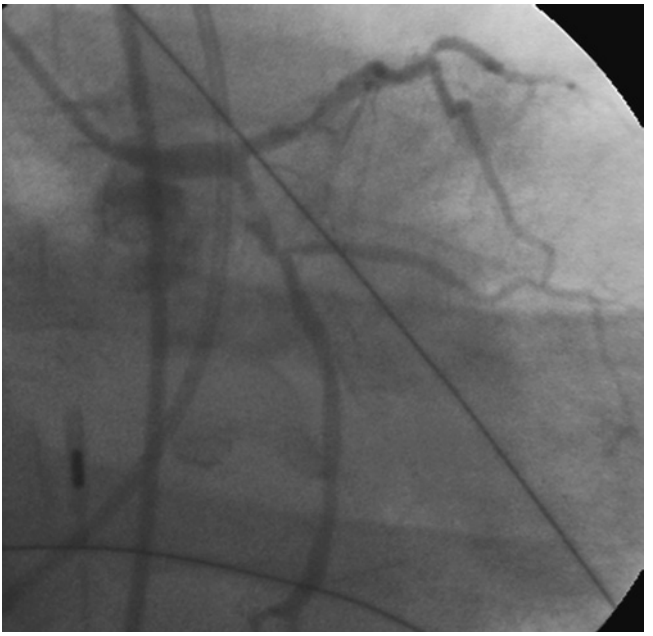

Figure 12-1 The TIMI flow. TIMI-0 flow: no antegrade flow (Figure 12-2a). TIMI-1 flow: some penetration of the contrast across the lesion. TIMI-2 flow: in this figure, the left circumflex artery is completely visualized but the flow is slower than the flow in the normal branch. TIMI-3: there is a strong and brisk flow (Figure 12-2b).

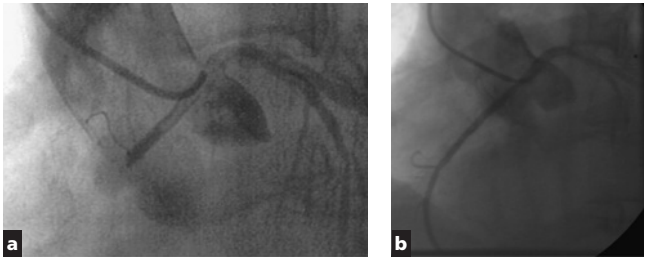

Figure 12-2 The missing RCA as IRA. The patient arrived with typical angina and ST segment elevation in leads 2, 3 and AVF. The LAD showed one moderate lesion in its mid-segment. The RCA was seen nowhere even with the aortogram. (a) The RCA was found to be in its usual location when it is originated from the left sinus: anterior and superior to the LM ostium. (b) The patient successfully underwent PCI of the artery. Courtesy of Starke Memorial Hospital, Knox IN.

Table 12-2 The High Risk Patients

1	>70 years of age
2	Ejection fraction <45%
3	Multivessel disease
4	Suboptimal PCI
5	Persistent arrhythmia
6	BP < 100 mmHg and HR > 100

Emergency electrocardiogram (ECG) rather than left ventriculogram should be performed in the cardiac catheterization laboratories (CCL) if postinfarction mechanical defect is suspected. A low threshold should be applied for placement of an intra-aortic balloon pump (IABP) in hemodynamically unstable patients. If pulmonary edema does not respond quickly to pharmacological treatment, endotracheal intubation and mechanical ventilation is mandatory. In such cases, the interventions of physician-intensivist are advised because it allows the interventional cardiologist to focus on the PCI procedure itself while medical and supportive care is effectively provided.

Primary PCI: After defining the coronary anatomy and clinical evaluation of the patient, primary PCI should be attempted if the IRA has a significant stenosis or thrombus with inadequate epicardial flow (TIMI < 3). The exclusions for primary PCI are listed in Table 12-3. Immediate surgical consultation regarding coronary artery bypass graft (CABG) is indicated in patients with concomitant multivessel disease or unprotected left main (LM) stenosis in excess of 50%.

Antiplatelet and Antithrombotic Strategies: Once the patient is seen, acetylsalicylic acid (clopidogrel) should be given.

Table 12-3 Angiographic Exclusions Precluding Performance of Primary PCI

1	Unprotected LM >60%
2	IRA with stenosis less than 70% and with TIMI-3 flow
3	IRA supplies small amount of myocardium: risk > benefits
4	Inability to clearly identify the IRA
5	Asymptomatic patient with multivessel disease with TIMI-3 flow, and CABG is indicated

Unfractioned heparin should be administered according to activated clotting time (ACT). With concomitant use of glycoprotein 2b3la inhibitors (GPI), an ACT of 200–250 seconds should be achieved. If the GPIs are not used, ACT between 300 and 350 seconds should be achieved. In patients with heparin-induced thrombocytopenia, recombinant hirudin, bivalirudin, or argatroban should be used during PCI. Bivalirudin, a direct thrombin inhibitor, is a short-acting anticoagulant with predictable pharmacokinetics and less demonstrable major bleeding complications than unfractionated heparin. Furthermore, its anticoagulant effect is predictable and has a short half-life in patients with normal renal function. One disadvantage of bivalirudin, however, would be the inability to reverse its anticoagulation process if this becomes necessary. This disadvantage, however, is at least partially offset by the very short half-life of the drug [8].

STRATEGIC MAPPING

The choice of adequate guide with coaxial alignment providing good support is crucial. If the IRA opens after passing the wire and if the distal parts are clearly visualized, direct stenting of the culprit lesion may be performed. This technique is not recommended in tortuous vessels, bifurcations and very complex lesions. It is important to notice that potential disadvantage of direct stenting is stent undersizing because the distal segment of the IRA may be inadequately filled by contrast due to significant residual stenosis or chronic spasm from low flow. Therefore balloon predilatation using undersized balloon (i.e. 2.5 mm) is usually suggested. If the patient is not hypotensive, intracoronary injection of nitroglycerin (100–200 micrograms) is useful to better appreciate the actual size and diameter of the IRA. This allows for better selection of the stent diameter and length. The goal should be a stent : artery ratio of 1:1. Oversizing of the stent may be associated with edge dissection, distal microvascular embolization and alpha adrenergic "storm" resulting in vasospasm in the distal microvasculature. Careful attention must be applied to ensure complete coverage of the culprit plaque and any residual intimal dissection. If the culprit

is a bifurcation lesion involving significant side branch (≤2 mm), its protection with additional wire before stenting (the trapped wire technique) is advised. In case of plaque shift with significant ostial stenosis or occlusion after stenting the main vessel, wires should be exchanged and final "kissing balloon" inflation performed. Systematic side branch stenting using different techniques such as "T, V, Y, culotte and crush" should be avoided because it does not reduce the rate of restenosis and may increase the risk of subacute stent thrombosis. At the end of the procedure, two orthogonal views of the stented segment should be obtained to confirm optimal angiographic result. Significant stenoses of non-IRA vessels should not be treated by the index PCI unless there is evidence of persistent ischemia, hemodynamic or electrical unstability despite adequate reperfusion of the IRA.

Primary PCI Without On-site Surgical Back-Up: PCI can be performed in hospital without on-site surgical back-up if the criteria detailed in Table 12-4 are fulfilled [9].

Primary Angioplasty Without Stenting: It is important to remember that not every lesion has to be stented. This is the case if optimal angiographic result (≤30% residual stenosis without evidence of dissection/residual thrombosis and TIMI 3 flow) is documented following only plain balloon angioplasty (POBA). In such cases, it is reasonable to wait for 5 to 10 minutes to confirm persistent angiographic result. If significant early elastic recoil, dissection or residual thrombosis is discovered, the culprit lesion should be stented. Only POBA has to be accepted in patients with excessive proximal tortuosity or/and calcification which prevents the passage of the stent. POBA with acceptable angiographic result is useful also in patients with IRA of very small diameter and in patients with contraindications for long term antiplatelet medications.

Table 12-4 Requisite Conditions for Primary PCI with no Surgery in Site

1	Experienced operators who perform regularly elective and primary PCI at tertiary centers
2	Nursing and technical staffs are experienced in handling acutely ill patients
3	Catheterization laboratories must be well equipped with resuscitative equipment, IABP etc.
4	Staff available 24/7/365
5	Must have protocols for emergent transfer to surgical centers (high grade LM, unstable 3-vessel disease)
6	Protocols should address in whom to delay PTCA (TIMI-3 flow with >70% residual etc.) [9]

Evaluation of the PCI Results: The goal of primary PCI is to achieve successful dilatation of the culprit lesion, normal epicardial blood flow and adequate microvascular reperfusion of infarcted myocardium [10]. Besides simple categorical estimate of TIMI epicardial flow, more accurate flow evaluation by using the corrected TIMI frame count (CTFC) (Figure 12-3). CTFC is defined as the number of angiographic frames needed for dye to traverse a coronary artery [11]. CTFC is particularly useful because it can account for rate of coronary filling and difference in epicardial vessel size and length, and also reduces inter-observer variability. The CTFC is an independent predictor of in-hospital mortality from STEMI and can further stratify patients with TIMI-3 flow into low- and high-risk groups. Restoration of flow in the IRA may not be a reliable predictor of restoration of tissue perfusion supplied by the IRA. Therefore the TIMI myocardial perfusion grading (TMPG) system was designed and validated to further risk stratify patients in whom successful epicardial reperfusion was achieved [12] (Figure 12-4). A very simple bedside indicator of microvascular reperfusion is also early ST-elevation resolution [13,14].

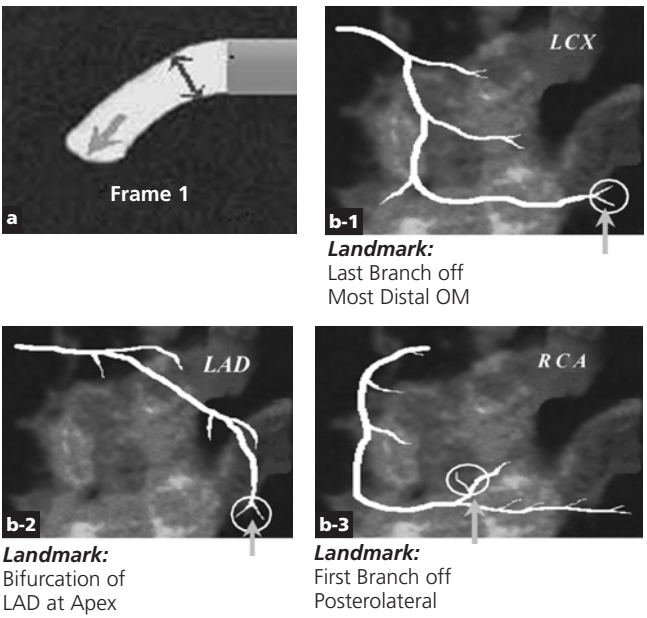

Figure 12-3 The Corrected TIMI frame count. (a) The first frame is defined as the frame in which injected contrast touches the two borders, but does not fully opacify it. The end frame is defined as the first frame in which contrast appears in the distal bed of the reference vessel. The distal landmark of the LCX (b-1), of the LAD (b-2), and of RCA (b-3).

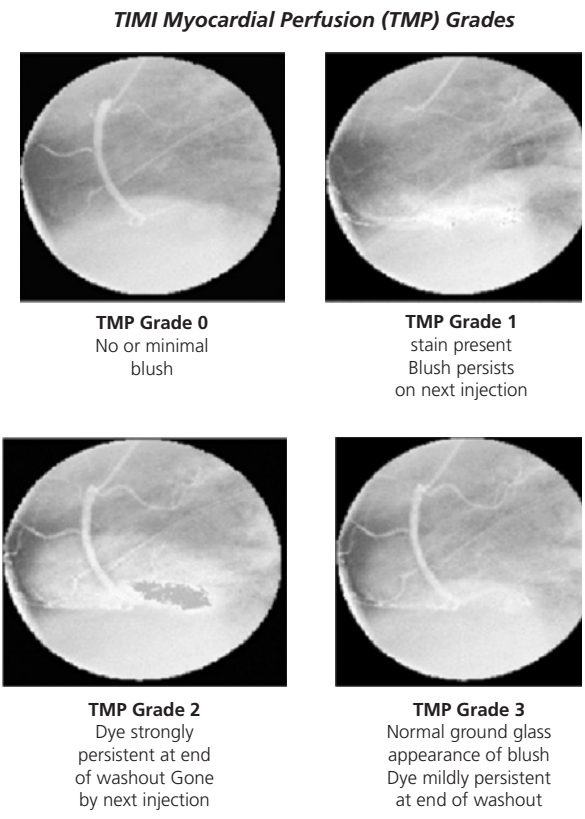

Figure 12-4 The Myocardial Perfusion Grading System. (a) TMP Grade 0: there is no dye entering the myocardium and there is minimal or no blush apparent in the distribution of the culprit artery. (b) TMP Grade 1 means appearance of blush in the distribution of the culprit artery, which persists on the next injection approximately 30 seconds later. (c) TMP Grade 2: there is a ground glass appearance ("blush") or opacification of the myocardium that is strongly persistent after three cardiac cycles of the washout phase and either does not or only minimally diminishes in intensity during washout. (d) TMP Grade 3: there is a ground glass appearance ("blush") or opacification of the myocardium in the distribution of the culprit artery, that clears normally and either gone or only mildly/moderately persistent at the end of the washout phase similar to an uninvolved artery.

Complete resolution (<30% residual elevation) is associated with better prognosis (Figure 12-5). On the other hand, absence of significant resolution identifies patients with more complicated in-hospital course and compromised long-term survival [15,16].

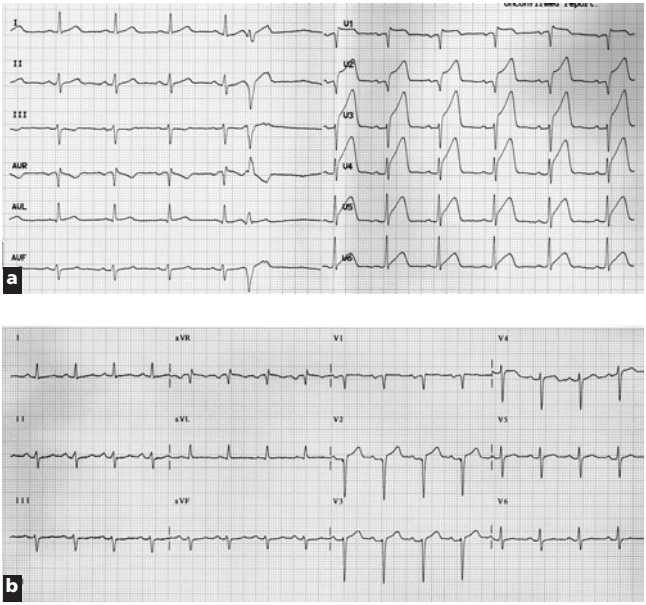

Figure 12-5 Complete ST-segment elevation resolution after primary PCI. (a) 12-lead ECG before primary PCI in a patient with evolving anterior STEMI due to proximal LAD occlusion. (b) 12-lead ECG in the same patient within 10 minutes after successful primary PCI.

CAVEAT

Did I Miss the IRA? During the diagnostic angiogram of a patient with STEMI, all the major vessels and their large branches should be accounted for. In a case report of a patient coming in with typical inferior wall myocardial infarction (MI) and ST elevation in 2, 3, F, an emergency angiogram showed moderate lesion in the left anterior descending artery (LAD) while the right coronary artery (RCA) was nowhere to be seen even with the aortogram. Because of such typical symptoms and ECG change, an extraordinary effort was made to locate the RCA which was found in its frequently seen anomalous location when the RCA is originated from the left sinus: anterior and superior to the LM ostium. The patient successfully underwent PCI of the anomalous RCA. In another encounter by one of the authors, a patient came in with severe chest pain. His angiogram showed patent LAD, left circumflex artery (LCX) and RCA. So where was the IRA? Because there was only minor ST elevation in leads I and AVL, so it was suspected that the diagonal should be the IRA.

A wire was advanced and probed the area suspected to be the possible origin of a large diagonal. The wire successfully entered and PCI of a large diagonal branch was performed successfully. In a case report by Sakurai *et al.* the coronary angiogram showed only minor lesion in a patient with typical ST segment elevation in all the V leads. So the guide was exchanged for a larger one, and LM dissection was demonstrated clearly. It was missed by a small deeply inserted guide bypassing the LM ostial dissection [17].

Avoiding Vasovagal Reaction: Rapid restoration of coronary flow, particularly to the IRA supplying inferior territory of the left ventricle, may lead to profound hypotension and bradyarrhythmias which are usually transient and benign events. It is recommended to aggressively hydrate patients with an inferior STEMI prior to PCI and avoid administration of nitrates and beta blockers. If bradycardia and hypotension develops, intravenous atropin bolus (0.5–1.0 mg iv) and rapid infusion of colloid solution is indicated. Venous sheath placed in the femoral vein prior to intervention may be useful for rapid flow of the fluid and insertion of a temporary pacemaker if necessary. It is also useful to ask conscious patients to perform "cough cardiopulmonary resuscitation" to overcome the short period of profound hypotension and bradyarrhythmia. If clinical suspicion for development of significant bradyarrhythmia is high, the operator may place a temporary wire in the right atrium, ready to advance into the right ventricle for pacing if necessary. If the patient developed ventricular fibrillation or tachycardia in the emergency room or in the field, prior to arrival in the CCL, cautions measures such as having all the patches for defibrillation readily taped on the chest and back of the patient, because the patient could develop VT or VF upon reperfusion with a wire or balloon.

Primary PCI of Saphenous Vein Graft Presenting as IRA: STEMI after previous CABG, usually related to saphenous vein graft (SVG) rather than mammary artery occlusion, affects smaller territories and presents with milder symptoms [18]. Because of the large size of the SVG and high thrombotic burden, there is a significant risk of distal embolization and "slow" or "no reflow" phenomenon [19,20]. Therefore, PCI of the native coronary artery supplied by the graft should be first attempted if the likelihood of success is realistic. If not, the operator should proceed with PCI of SVG. If anatomy and site of the culprit lesion allows, distal protection device should be used to reduce distal embolization, improve angiographic success and improve the clinical outcome (Figure 12-6). If large thrombotic burden is identified, distal protection should be combined also with thrombus aspiration.

Primary PCI of an Unprotected LM as IRA: The majority of patients with sudden thrombotic occlusion of unprotected LM die before reaching the catheterization laboratory. Only patients with

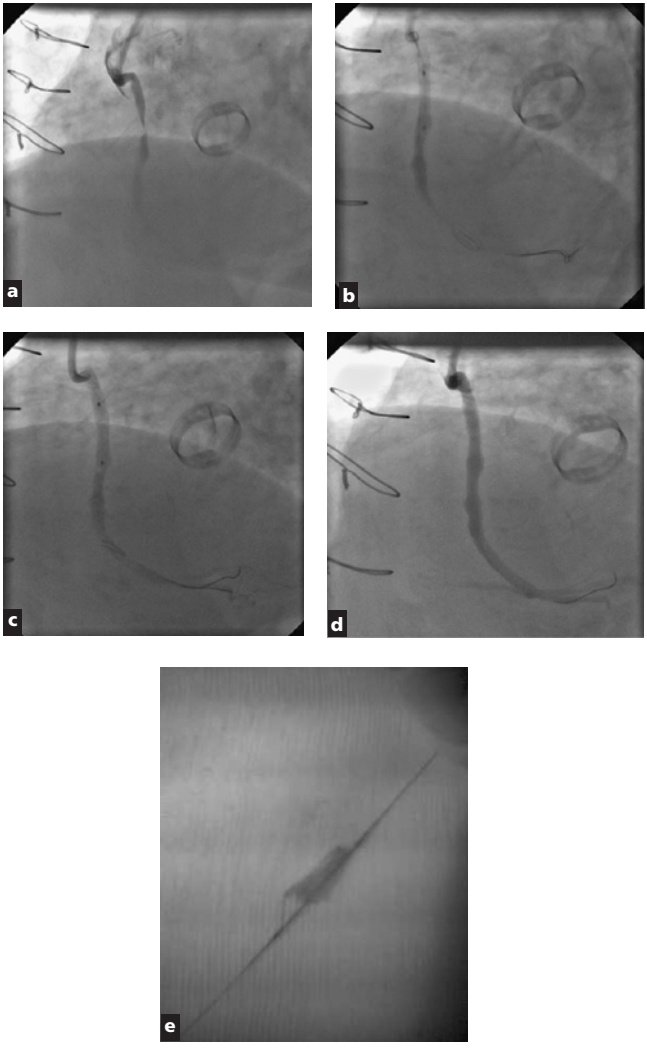

Figure 12-6 Acute occlusion of saphenous vein graft to distal RCA with evolving inferior STEMI. (a) Thrombotic occlusion of the graft with TIMI 1 flow. (b) Balloon predilatation using undersized balloon with distal protection (Filterwire) in place. (c) Stenting of the culprit lesion under protection of Filterwire. (d) Good angiographic result after removal of Filterwire. (e) Captured thrombotic debris in the basket of the Filterwire.

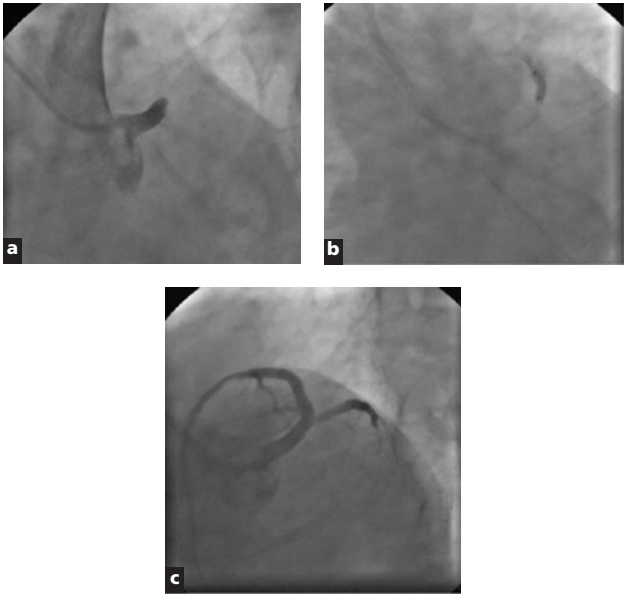

Figure 12-7 STEMI due to acute LM occlusion in a patient with small anomalous LCX arising from proximal dominant RCA. (a) Stump of the LM. (b) Following predilatation, stenting with wire protection of large D1 was performed. (c) Final angiogram revealed widely patent LAD with some thrombus-plaque shift in D1 and TIMI 3 flow in both branches. Because of cardiogenic shock, IAPB was inserted immediately after successful primary PCI.

intermittent LM occlusion, very dominant and patent RCA and anomalous origin of LCX, have a realistic chance of survival (Figure 12-7). Therefore, published experience with primary PCI in patients with unprotected LM-STEMI is rather limited. Since patients with acute LM occlusion will almost always present in profound cardiogenic shock, concomitant use of IAPB, vasopressors, inotropes and mechanical ventilation should be started before PCI. It is very important to distinguish between unprotected LM as IRA and significant stenosis of unprotected LM with the other coronary artery as IRA. In this case, only the IRA should be treated during the index procedure if the patient is stable and ischemia-free after primary PCI. Revascularization of unprotected LM should be postponed until the patient recovers from the subacute phase of STEMI. In the setting of ongoing ischemia due to significant LM stenosis despite successful primary PCI of the IRA and adequate hemodynamic support with IAPB, immediate PCI of the unprotected LM or CABG should be seriously addressed.

Rescue PCI After Failed Thrombolysis: Because of delayed access to PCI or because PCI is not available, many patients with STEMI

are treated with thrombolytic therapy (TT). In cases of thrombolytic failure, immediate angiography and mechanical recanalization of the IRA remains the treatment of choice [21]. Since the clinical signs and ECG data of reperfusion are not precise, the guidelines of the ACC/AHA task force suggest performing urgent angiography in any patients receiving TT with ongoing chest pain or hemodynamic instability, or in asymptomatic patients who are within less than 12 hours of symptom onset with persistent ST elevation after 90 minutes of thrombolytic therapy. It is important to notice that patients who require rescue PCI due to failed thrombolysis remained at increased risk for reocclusion, because they possibly had higher resistance to pharmacologic reperfusion, large thrombus burden or platelet-rich thrombi, factors unfavorable to the performance of mechanical intervention. Rescue PCI should be performed on high risk lesion (>75%) with TIMI of 2 or less.

Where is the IRA in Right Ventricular Infarction? The clinical manifestations of right ventricular infarction (RVI) include signs of acute right ventricular failure such as hypotension, jugular vein distension, right-sided fourth heart sound and sometimes Kussmaul sign. On the other hand, patients with inferior myocardial infarction with right ventricular involvement have an increased incidence of atrioventricular block, bradycardia, ventricular arrhythmia and shock, all of which result in higher mortality rates. Although it is very uncommon, some patients may suffer an isolated RVI. This may occur when a non-dominant or co-dominant right coronary artery proximal to an acute marginal branch, or an acute marginal branch, is occluded. An isolated RVI may also occur secondarily to an acute occlusion of the RV branch following angioplasty. The coexistence of RVI with severe left ventricular hypertrophy probably favored the appearance of hemodynamic manifestations in two ways. First, left ventricular diastolic dysfunction may produce an increase in pulmonary wedge capillary pressure and thus facilitate the occurrence of right ventricular failure. Second, the decreased left ventricular pre-load due to RVI may be potentially more serious in the presence of left ventricular diastolic dysfunction. On the other hand, right ventricular myocardial abnormalities that may be present in hypertrophic cardiomyopathy or even in hypertensive cardiomyopathy, could have contributed to the incidence of right ventricular failure [22].

TECHNICAL TIPS

****Crossing The Lesion:** Usually the IRA is occluded with a soft and fresh thrombus that can be crossed easily with a steerable floppy wire. In case of tortuosity and need for better wire pushability, a small balloon placed near the distal tip of the wire may help. Stiffer and hydrophilic wires are usually not required. The lesion can then be "dottered" by moving the uninflated balloon back and forth. This will allow flow of stagnant blood proximal to the occlusion to seep slowly into the distal vasculature. This maneuver prevents the abrupt

opening of the occluded artery, which theoretically may minimize the chance of flooding of the distal vasculature with stagnant (and possibly acidic) blood. This may decrease the probability of developing reperfusion arrhythmias, especially if the IRA is the RCA. After completion of this maneuver, the operator may inject a small amount of contrast to verify the position of the wire in the true lumen (and not in a sidebranch). If the wire position is still ambiguous, then a small over-the-wire (OTW) balloon can be advanced, the wire removed and contrast can be injected through the central lumen. This maneuver also helps in the assessment of the artery size for selection of subsequent balloon or stent.

LESIONS WITH THROMBI

In situations of small or moderate thrombotic burden, conventional PCI should be performed. Thrombotic burden is often large in patients with prolonged symptom duration or if the IRA is a large-diameter, or ectatic vessel such as an RCA or a saphenous vein graft (Figure 12-8). In such cases, it is common sense to remove the thrombus prior to stenting in order to reduce the likelihood of distal embolization in the IRA branches or microcirculation resulting in "slow" or "no reflow" phenomenon [23].

TECHNICAL TIPS

***Avoiding Antegrade Embolization:** Embolization to one or more peripheral IRA branches distal to the culprit lesion can occur due to strong contrast injection or as a consequence of thrombus dissolution by balloon inflation or stenting. This is why manipulation of hardware in the IRA has to be gentle and minimal to avoid dislodgement of thrombus. There is some evidence that systematic use of distal protection devices such as Guard Wire [24] and Filter wire [25] may reduce distal embolization [26]. These benefits have as yet not been translated into the better clinical outcome [25]. If embolization in a significant distal branch occurs, another guidewire should be placed in the embolized branch. The lesion can then be carefully "dottered" by moving back and forth a deflated balloon. If anterograde flow is established and significant lesion visualized, additional PCI may be performed if the branch supplies important part of myocardium (Figure 12-9).

***Avoiding Retrograde Embolization:** During PCI or thrombectomy at the proximal segment of the LAD and LCX, fragments of large thrombi may be squeezed back and occlude the ostium of the adjacent vessel. Fragments of the thrombus can even adhere to balloons or aspiration catheter, which when withdrawn or retracted into the guide, may lead to embolization and occlusion of the IRA and non-IRA branches. Occlusion of a large non-infarct vessel may have dramatic consequences including immediate cardiac arrest and profound cardiogenic shock. Embolization may be minimized by first opening the

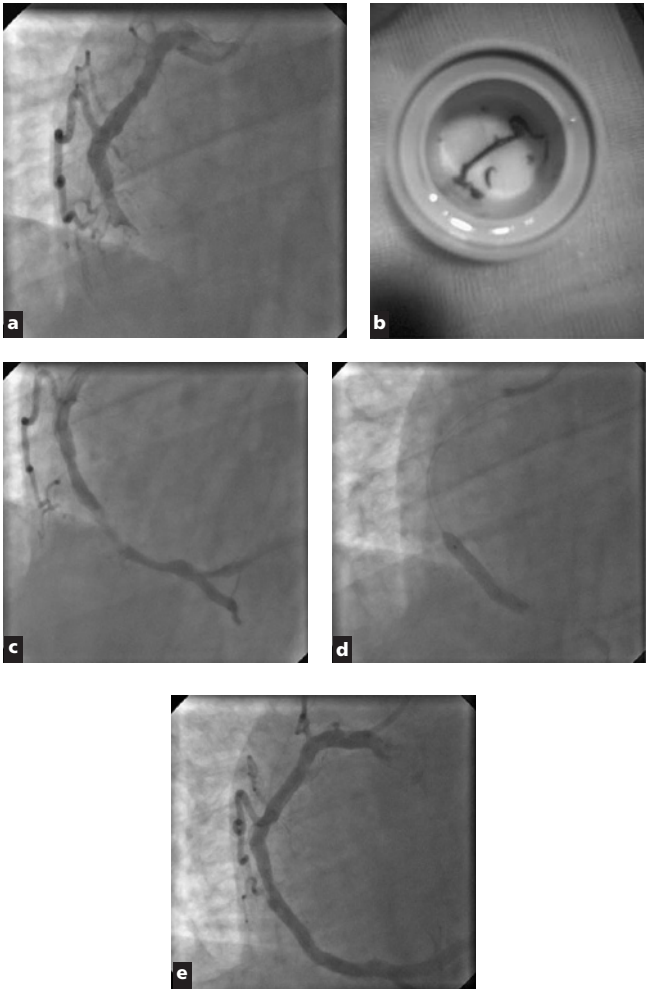

Figure 12-8 Catheter aspiration of thrombus in a patient with evolving STEMI in the territory of dominant RCA. (a) Thrombotic occlusion of the mid RCA. (b) Aspirated thrombus obtained after the first passage of Diver catheter. Subsequent passages did not result in significant aspiration. (c) Angiographic finding after aspiration. (d) Direct stenting of the culprit lesion. (e) Very good final angiographic result with TIMI 3 epicardial flow, TMP3 and significant early ST-segment resolution.

Y-connector prior to injection of contrast agent and allow back flow to remove any free thrombotic material. If ostial LAD or LCX culprit lesion is treated, it is reasonable to protect the adjacent vessel with additional wire to allow for immediate intervention if required (Figure 12-10).

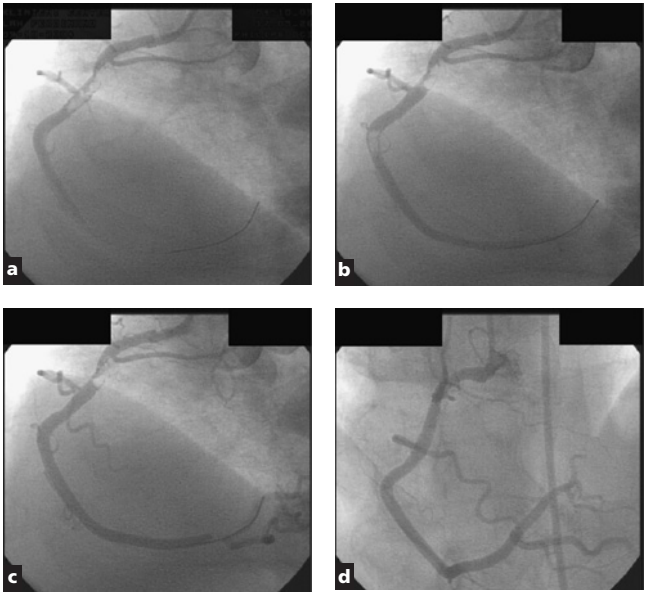

Figure 12-9 Distal embolization of large thrombus in proximal dominant RCA in a patient with STEMI and cardiogenic shock. Following smooth passage of the wire, angiography of the RCA was performed. (a) At the beginning of coronary injection, large proximal thrombus was identified. (b,c) During the contrast injection, a large part of the thrombus detached and traveled through the mid portion of RCA and stopped at the crux. (d) Following direct stenting of the culprit lesion and catheter aspiration of the distal thrombus, good angiographic result was obtained. For temporary hemodynamic support, IAPB was inserted.

***"Slow" or "No Reflow" After Stenting:** After the IRA is opened with a wire or after balloon predilatation, epicardial flow should increase. Then, after stenting, one-third of patients had substantially diminished epicardial TIMI flow, while the other two-thirds had increased flow or no change [27]. Diminished epicardial blood flow despite widely patent IRA, known as "slow" reflow (TIMI 2) or "no reflow" (TIMI 0 or 1) phenomenon, is due to compromised distal microvascular perfusion. The mechanism of "slow-no reflow" is probably heterogeneous and include thrombus-plaque microvascular embolization with subsequent platelet activation, release of potent vasoconstrictors and microvascular spasm [28]. Once this problem is encountered, the flow will improve after infusion of adenosine, nicorandil, calcium channel blockers, or nitroprusside.

Persistent Thrombotic Burden: In cases of persistent thrombus after repeated balloon angioplasty, the best treatment would be to attempt thrombectomy using either the Angiojet™ or the X-Sizer™

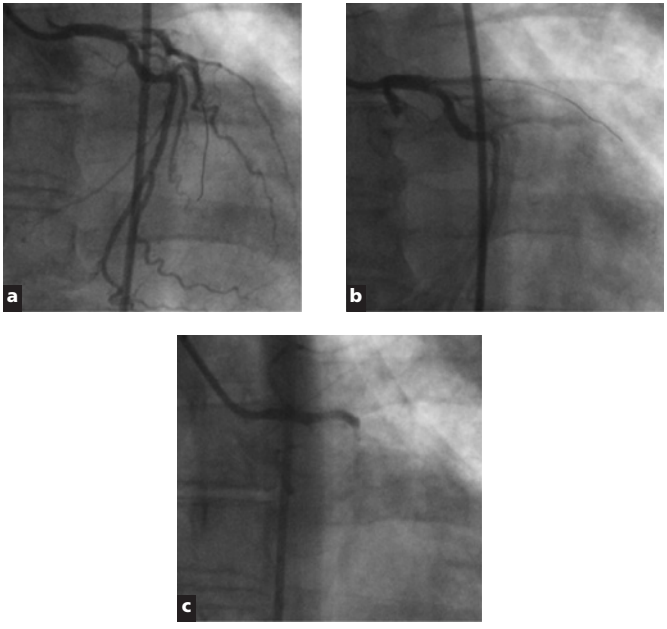

Figure 12-10 Retrograde thrombus embolization: (a) During PCI of the proximal segment of the LAD, a well organized large thrombus was seen to be benign with balloon angioplasty. (b) The thrombus was then squeezed back during stenting. (c) This resulted in acute occlusion of the left circumflex causing cardiac arrest.

device if the thombus is large. Smaller distal thrombi can be aspirated by a Pronto or Export catheter. Any residual thrombus could be lysed by intracoronary r-tPA (5 mg boluses down the left coronary artery via the guide every five minutes (up to 50 mg) will usually suffice). If the vessel then looks clear of thrombus and distal coronary flow looks good, IV heparin over the next 24 hours should be continued (ACT 200 seconds). If residual thrombus appears to exist or distal coronary flow appears suboptimal, then abciximab should be given (intracoronary bolus followed by a 12-hour IV infusion). However, in this situation, it is essential to carefully monitor the hematological and clotting indices and observe for any signs of bleeding if serious hemorrhagic complications are to be avoided.

If the therapeutic doses of antiplatelet, antithrombin and/or thrombolytic medications are not effective in reducing thrombus size and restoring antegrade flow in a coronary vessel or vein graft, there are almost always one or more mechanical lesions together with some degree of distal coronary vasospasm present and these needs to be addressed. In patients without an unusual clotting abnormality, using "super therapeutic" doses of multiple medications are more likely to

be associated with a significant bleeding complication, which itself, could be life-threatening [29].

PCI OF STEMI PATIENTS WITH CURRENT BLEEDING

In general, the principle is that PCI can be performed if the bleeding can be stopped by mechanical means (compressing or ligating the artery) and the patient can tolerate 4 hours of anticoagulant without excessive further bleeding during PCI. The favorite anticoagulant is unfractioned heparin (UFH) because of its short half life and it can be reversed by protamine.

CASE REPORT Patients with Bleeding due to Leg Fracture: A patient had AMI when driving a car. The patient lost control of the car and hit an oncoming vehicle. The patient suffered a fracture in the leg with profuse bleeding. In the emergency room the orthopedic surgeons put splints and stabilized the extremities, then the patient underwent a diagnostic coronary angiogram and bilateral femoral angiogram to check extravasion of contrast due to injury in the arterial system. If there is no arterial bleed in the extremities, then the patient could undergo PCI of the IRA under coverage with UFH and clopidogrel. After angioplasty and stenting, the patient can undergo noncardiac surgery. At this present time, intracranial, lower intestinal, or bleeds from the esophageal varices are the only contra-indications for PCI.

CASE REPORT AMI in Patient Recent Surgery: Less than 4 hours after removal of the right kidney because of cancer, a patient developed ST elevation in leads 2,3 AVF. So the patient was brought to the CCL, had balloon angioplasty of the RCA with standard dose of heparin to achieve an ACT of 250–300. No stent was used and no heparin given after the procedure. Because the short term of heparin use, not much bleeding in the surgical area was noted and no there was no impact on the surgical results. If the patient has a clean and limited surgery, then the patient could have drug eluting stent (DES) stenting because surgery causes no problem with long term antiplatelet therapy with clopidogrel [30].

AMI in Patients With Concurrent Stroke: If the patient has ischemic stroke, then with the agreement from the neurologist on the case, the patient could be given short term anticoagulant (UFH or direct antithrombins (DTI)) and long term oral antiplatelet drugs. Then the patient could undergo PCI and stenting. The two concerns are: (1) the risk of hemorrhagic conversion of the ischemic stroke with anticoagulant therapy; and (2) risk of cerebral emboli from the protruding plaque in the aortic arch if they were the cause of emboli stroke in the first place. The patient needs to have strong indication for PCI and the family and patient need to understand the benefits and the risks. If the benefits outweigh the risks, then the patient should have PCI.

STEMI in Patient with Atrial Fibrillation (AF) on Coumadin, INR > 2: Because coumadin does not have any effect on platelet, so patient with therapeutic level of INR still have STEMI. During PCI, the patient can be given oral loading and maintenance dose of antiplatet drug (such as aspirin (ASA) or clopidogrel) as usual. If the patient has therapeutic INR (2–3), no UFH is needed. If the INR is less than 2, then patient can be given UFH (as in the treatment of pulmonary embolism).

CASE REPORT Primary PCI in Patient with Recent Neurological Surgery: In a case report by Almeda *et al.* a patient had sudden onset of severe headache and lost consciousness. A stat CT scan of the head revealed a subarachnoid hemorrhage with evidence of hydrocephalus. Urgent cerebral angiography showed a basilar tip aneurysm of which he underwent successful coiling and a placement of a right frontal external ventricular drain. Approximately 6 hours after the procedure, while in the neurosurgery intensive care unit, the patient was found to have new 2 mm ST-segment elevation by 12-lead ECG in the inferior leads. Due to the recent placement of a right frontal external ventricular drain, and the coiling of the basilar tip aneurysm for the subarachnoid hemorrhage, the optimal anticoagulation strategy was discussed at length with the neurosurgeon. Use of a glycoprotein 2b3a inhibitor was deemed to be relatively contraindicated due to the risk for intracerebral bleeding, and the consensus was to utilize bivalirudin (60.7 mg bolus followed by 1.75 mg/kg/hour maintenance infusion) as antithrombotic. The patient was pre-treated with a loading dose of aspirin. The risks of any anticoagulation, including heparin or bivalirudin, or antiplatelet agents such as aspirin or clopidogrel, are clearly not quantifiable from available data in the literature. In this setting, placement of a coronary stent should be avoided if possible. The stent obligates the patient to both aspirin and clopidogrel in the acute setting. An adequate balloon angioplasty result would have accomplished the acute goal of restoring flow to salvage the myocardium. The higher rate of restenosis associated with balloon angioplasty in comparison to a stent would have been less of an acute concern than the increased risk of bleeding associated with the addition of clopidogrel. Furthermore, a Hepacoat stent could have been considered to treat a suboptimal balloon angioplasty result since there are some data to suggest that the subacute stent thrombosis rate is acceptable with this stent in the absence of clopidogrel [31].

EXOTIC COMPLEX INTERVENTIONS FOR THE WEEKEND URBAN WARRIORS

1. PCI in Patients with LM Compressed by an Aortic Disssection: In a case report by Cardozo *et al.,* a patient came with ST segment elevation. A coronary angiography was performed.

The initial injection revealed a very tight stenosis of the LMCA with persistence of contrast medium at the LAD and CX levels, despite a normal pressure curve (no ventricularization). This suggested that the main stem was undergoing extrinsic compression. The guide was then withdrawn through the ascending aorta, where a more powerful injection demonstrated an acute aortic dissection (AAD) with a false lumen compressing the LMCA. Subsequently, the left coronary ostium was engaged again and a more vigorous injection showed a typical image of coronary artery compression, i.e., complete resolution of the tight stenosis resulting in a wide open LMCA with intermittent diastolic collapse of the lumen. With a diagnosis of AMI secondary to LMCA compression by AAD, direct left main stenting was performed as a bridge procedure for possible subsequent aortic surgery. After stenting, a supravalvular aortography was performed and showed a Stanford type A aortic dissection. The lumen size varies according to diastolic and systolic flow, producing an image of "swinging lumen." In cases where this phenomenon reaches maximum amplitude, diastolic interruption of coronary flow will produce a persistent contrast-filling image despite perfectly normal pressure curves at the aortic level [32].

2. PCI of a Reconstructed Prosthetic LM: In a case report by Hussain et al., a patient with known Marfan's syndrome and an extensive past history of complicated aortic operations presented with chest pain and ST elevation in the V leads. In the past, because of aortic aneurysm, the patient had a Bentall procedure which essentially involves the complete replacement and exclusion of the ascending aorta with a composite Dacron tube graft and implantation of an aortic valve prosthesis. The coronary arteries are then reimplanted onto the sides of aortic tube graft. Because of aneursymal change in the aortic graft, the patient underwent a second procedure including a re-do replacement of the ascending aorta with a 28 mm tube graft, repair of the anterior and left posterolateral sinus of Valsalva and reimplantation of his right coronary artery using the classic Bentall technique and reimplantation of his left main coronary ostia utilizing a 10 mm Dacron graft (Cabrol graft) involving connection of the native coronary artery ostium to the aortic tube graft (Bentall) in a side-to-side manner by utilizing an interposed 8–10 mm diameter Dacron tube graft of variable length. During PCI for AMI, an aortic root angiography demonstrated the usual position takeoff for the right coronary artery and a high superolateral takeoff for the Cabrol graft. A standard Judkins right (JR 4) diagnostic catheter managed to cannulate the right coronary, which was angiographically unremarkable. Injections of the Cabrol graft confirmed a 90% severe hazy stenosis at the ostium of the left main at the anastomosis with the Cabrol graft. A JR 4 guide was used to engage the Cabrol graft. A 0.014" 300 cm supportive wire was passed into the distal LAD. The left main was stented successfully [33].

CRITICAL THINKING

Contrast Leakage into the Ventricle? Is It Wire-Induced Perforation or Impending Rupture?

About 10 minutes after the procedure, severe chest pain recurred and blood pressure fell to 70/40 mmHg. Physical findings were unremarkable and no heart murmur was audible. The ECG showed that the ST segment was elevated again in leads II, III, and aVF. The patient was brought back to the catheterization laboratory. This time, coronary angiography revealed contrast leak from small branches of the posterior descending artery.

Judging from the angiogram, together with the sudden hemodynamic collapse, guidewire-induced coronary perforation and cardiac tamponade were suspected. Echocardiography disclosed neither pericardial effusion nor abnormal color Doppler signal. However, right heart catheterization showed equilibration of atrial and ventricular diastolic pressures at 18–20 mmHg. A 1.5 mm balloon was advanced to the posterior descending artery and inflated at 1 atm for 15 min to seal the possible perforation. After inflation, the angiogram showed persistent contrast leak. The leakage sites even increased compared to the previous angiogram. No improvement was seen after further inflation for 30 min. Blood pressure fell to 50/30 mmHg and the patient was intubated for hypoxia. Then, echocardiography was repeated. Again, pericardial effusion was not identified, but color Doppler suggested shunt flow at the posterior septum. Blood gas sampling revealed a step-up in oxygen saturation from the right atrium (RA; 41.2%) to the right ventricle (RV; 74.8%; Qp/Qs = 2.8), and the diagnosis of septal rupture was confirmed. The following findings are the keys to differentiating ventricular septal rupture from coronary perforation: First, contrast soon disappeared following injection. If coronary perforation is caused by a guidewire, some contrast usually remains visible at the perforation site. The septal rupture dissected branches of the posterior descending artery during its progression, and the contrast directly drained into the ventricular cavity. Second, the contrast leak occurred from many small branches, and the number of leakage sites gradually increased. It would be unlikely for a guidewire to penetrate into all of these small branches [34].

3. Primary PCI in STEMI after Resuscitated Cardiac Arrest:

After successful cardiopulmonary resuscitation of patients with cardiac arrest, 12-lead ECG may show evidence of STEMI. Such patients represent 5–10% of STEMI population [35,36]. According to published experience, urgent coronary angiography and primary PCI are feasible, effective and safe with hospital survival of 77% [35,37–39] (Table 12-5).

Table 12-5 Experience of Primary PCI After Resuscitated Cardiac Arrest

Author	Successful primary PCI	Hospital survival
Kahn 1995	7/11	6/11
Bendz 2004	38/40	29/40
Quintero-Moran 2006	56/63	47/63
Gorjup 2006	102/108	88/108
Total	203/222 (91%)	170/222 (77%)

It is very important to notice that the main determinator of hospital survival in these patients is the degree of postresuscitation brain injury. In patients, who regain consciousness after re-establishment of spontaneous circulation, survival to hospital discharge is comparable to patients without preceding cardiac arrest [35]. On the contrary, in patients who are still comatose in CCL, survival hardly exceed 50% with good neurological recovery in less than 30%.

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Chapter 13
Interventions in Patients after CABG

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ < 100.00 $US extra; **$$** > 100.00 $US extra
⌛ < 10 minutes extra; ⌛⌛ > 10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

Patients who experience recurrence of ischemia after coronary artery bypass graft surgery (CABG) have lesions in diverse anatomic distributions (saphenous vein graft (SVG), native arteries, internal mammary, radial, gastroepiploic graft, or proximal subclavian artery). The results of percutaneous coronary interventions (PCI) depend on the types of conduits (native artery, arterial or saphenous vein grafts) or the locations on the conduits (proximal, mid-, distal or at the anastomotic sites) and the age of the grafts [1]. The clinical and technical problems encountered during PCI of SVG are listed in Table 13-1.

Early Postoperative Ischemia (<1 month): The most common cause of ischemia within hours or days of surgery is acute vein graft thrombosis (60%). Other causes are incomplete surgical revascularization (10%), kinked grafts, and focal stenoses distal to the insertion site and at the proximal or distal anastomotic sites, spasm or injury, insertion of graft to a vein causing AV fistula, or bypass of the wrong vessel [2].

Table 13-1 Clinical and Technical Problems during PCI of SVG

Problem	Corrective Measure	Adverse Outcome
Diffuse disease	Long stent	High rate of restenosis
Thrombus	Thrombectomy	Distal embolization
Degenerated SVG	Distal Protection	Distal embolization
Restenosis	Drug-Eluting Stent (DES)	? % restenosis
Retrograde embolization in ostial lesion	???	CVA, distal organ emboli

Early Postoperative Ischemia (1 month–1 year): Recurrent angina between 1 month and 1 year after the surgery is most often due to peri-anastomotic stenosis, graft occlusion, or mid-SVG stenosis from fibrous intimal hyperplasia. Recurrence of angina at about 3 months postoperatively is highly suggestive of a distal graft anastomotic lesion and should, in most cases, lead to evaluationfor PCI.

Late Postoperative Ischemia (>3 years after surgery): At this stage, the most common cause of ischemia is due to formation of new atherosclerotic plaques in the SVG. However, these plaques have less fibrocollagenous tissue and calcification, so they are softer, more friable, of larger size, and frequently associated with thrombus.

INDICATIONS

Indications for Percutaneous Interventions: PCI offers a less invasive alternative for revascularization in symptomatic post-bypass patients including many who were not candidates for repeat surgery because of contraindications (pulmonary and renal failure, old age, malignancy). Other patients who can undergo PCI with acceptable risks are patients with patent arterial grafts that would be jeopardized by reoperation, patients with relatively small amount of ischemic, symptom-producing myocardium, and patients with no arterial or venous conduit available for graft.

The status of the left anterior descending artery (LAD) and its graft significantly influences the selection process because of its impact on long-term outcome and lack of survival benefit of repeat surgery to treat non-LAD ischemia [3]. A patent left internal mammary artery (LIMA) to LAD improves the safety and so favors the selection of PCI in the right coronary artery (RCA) or left circumflex artery (LCX) distributions.

Therefore, selection of lesions for PCI must be based on careful analysis of the probabilities of initial success, complications, and for long-term safety and efficacy compared with competitive surgical strategies and medical therapies.

Indications for Surgical Revascularization: Reoperation is frequently recommended for severe disease of vein graft to the LAD.

Table 13-2 Anatomic Factors Influencing Revascularization Decisions in Post-Bypass Patients

Often lead to PCI	Often lead to CABG
Patent arterial graft (especially LAD)	Diseased SVG to LAD
> or = 2 patent grafts	Ejection fraction: 25–35%
1–3 culprit lesions	>3 culprit lesions
Inadequate conduit	Multiple SVG lesions
Near normal left ventricle	Available arterial conduits
Difficult surgical access	
*Posterior lateral target vessel	
*Mediastinal scarring secondary to radiation, infection, or pericarditis	
*Prior muscle transfer closure of unhealed sternotomy	
Future cardiac surgery anticipated	
*In-situ prosthetic valve	
*Mild to moderate aortic or mitral valve disease	

Multiple vessel involvement, small number of patent grafts, severe vein graft disease, and a damaged ventricle are factors more likely to lead to repeat surgery (Table 13-2) [1]. In the past, PCI was not preferred for bulky atheromatous lesions or thrombus-laden grafts. With the advent of distal protection devices, safer results are achieved for PCI of these lesions.

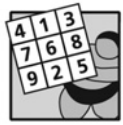
NATIVE CORONARY INTERVENTIONS

One year after bypass surgery, patients begin to develop new atherosclerotic plaques in the graft conduits or to show atherosclerotic progression in the native coronary arteries. Whenever possible, native artery lesions are targeted first because of their lower rate of restenosis. Approaches to native vessel sites in post-bypass patients include the treatment of protected left main disease, recanalization of old total occlusion, or native artery via venous or arterial grafts.

SAPHENOUS VEIN GRAFT INTERVENTIONS

One to 3 years after surgery, patients begin to develop atherosclerotic plaques in the SVG and after 3 years, these plaques appear with increased frequency. At the early stage, dilation of the distal anastomosis can be accomplished with little morbidity and good long-term patency (80–90%). Dilation of the proximal and mid-segment of the vein graft was highly successful at 90%, with a low rate of mortality (1%), Q-wave myocardial infarction (MI) and CABG (2%). The rate of non-Q-wave MI was 13%. The length of time since surgery was an important factor for restenosis as was the location of the lesion.

STRATEGIC MAPPING



Complex SVG Interventions: When evaluating the SVG lesions for intervention, the operators must consider possible consequences of atheromatous embolism, considering that the entire lesion and accompanying thrombus may be fragmented, dislodged, and embolized. If the risk of major atheroembolization, which could be decreased by distal protection devices, is acceptable, compared with other therapeutic opions, PCI may be appropriate [1]. Also, the relatively high subsequent coronary event rate and restenosis potential must also be factored into this decision.

Intervention of the Aorto-Ostial Lesion: There is not much difference in the technique of PCI for aorto-ostial lesion of the SVG. However, because there is increased fibrotic change and more spasm, there is a question about need of prior debulking followed by stenting or stenting alone of the aorto-ostial lesion. The technical concern during PCI of large and bulky aorto-ostial lesion is the antegrade and retrograde embolization [4].

Intervention in Degenerated SVGs: The lesions that are bulky or associated with thrombus are considered to be high risk. The complications include distal embolization, no reflow, abrupt closure, and perforation. So different preventive approaches such as mechanical thrombectomy and distal protection are devised because there is much to lose from the standpoint of distal embolization causing non-Q MI and increasing long term mortality (Table 13-3). In the case of perforation of SVG, usually there is contained perforation rather than cardiac tamponade (which still can happen) due to the extrapericardial course of the grafts and extensive post-pericardiotomy fibrosis.

Table 13-3 Advantages and Disadvantages of Distal Protection Devices

Advantages	Disadvantages
Balloon Occlusive Devices	
Easy to use	No antegrade flow
Aspirate large and small particles	Balloon-induced injury
Reliably trap debris	Not as steerable as PTCA wire
More tolerable with intermittent occlusion	Difficult to image during procedure
	Balloon can move during PCI
Distal Embolic Filter Devices	
Preserve antegrade flow	May not capture all debris
Contrast imaging possible throughout the procedure	Difficult to evaluate the retrieval of debris during procedure
	Filters may clog
Delivery catheters may cause embolization before filter deployment	
Cannot remove emboli intermittently in order to relieve overload	

Interventions of the Saphenous Vein Graft: In general, in search for the location of the insertion site, the more posterior the destination of the left-sided grafts, the higher they are located on the aorta. The top graft generally goes to the distal LCX, and the lowest graft goes to the LAD. Most left-sided grafts arise in a cranial direction from the aorta. Right coronary grafts are usually in the most caudal and rightward position on the aorta [1].

TECHNICAL TIPS

****Guides for Left Bypass Grafts:** The Judkins right coronary or left venous bypass or hockey stick catheters are effective guide for grafts arising anteriorly (the LAD and diagonals). However the left Amplatz and the hockey stick guides often provide the best backup for grafts arising in the inner curvature of the aorta (to the LCX). Engagement is best achieved by advancing the guide into the ascending aorta at the level of the aortic cusps then gently withdraw with clockwise rotation of the guide to orient the tip to the ostium in the LAO view. When the tip of the guide catches the ostium, the guide is then advanced to obtain optimal backup [5].

****Guides for Right Bypass Grafts:** For the grafts arising from the outer curve of the aorta, (usually to the RCA), the Multipurpose guide is the best to provide excellent co-axial alignment. Engagement into the ostium is achieved by advancing the guide into the aorta while making a clockwise rotation in order to point its tip towards the right, in the LAO projection [5]. In the case of a right Judkins or small Amplatz right, the guide is turned clockwise so it will point toward the outer curvature then it is slowly turned counter-clockwise to engage the graft with its tip pointing down. If the aorta is relatively large, a posteriorly located RCA graft may be difficult to reach with a Multipurpose or right Judkins guide but an Amplatz left will usually be successful in this situation.

***Balloon Angioplasty for Vein Grafts:** Balloons are generally sized 1:1 to venous grafts and slightly oversized for suboptimal initial results, or when dealing with restenotic lesions. Long (30–40 mm) balloons are frequently used when the lesions are long and bulky or when thrombus is present. The extra fibrosis of mature vein graft lesions often requires dilation to higher pressures >12 atm.

****Stenting for Vein Grafts:** The SVGs have a high degree of elastic recoil that can be overcome by stenting. Aorto-ostial vein graft lesions are most often treated with placement of stents. Nondilatable aorto-ostial or distal anastomotic lesions have been successfully treated with rotational or directional atherectomy then stent with drug eluting stent (DES).

****Size and Length of Stent:** If a small stent is deployed in a friable and fragile atheromatous segment of a SVG, further attempts to recross it with large a balloon would increase the risk of stent

embolization. Deployment of a too-large stent would cause distal embolization due to excessive plaque extrusion. It is the same reason that overdilating a balloon should be avoided. The length of the stent should be longer than the measured length of the lesion. The reason is the soft atheromatous content of a plaque will be squeezed farther and rearranged along its length due to pressure by a stent. When multiple stenting is planned, the distal stent is deployed first then the proximal one. This strategy is to avoid recrossing newly deployed stent. However, if there is very tight proximal lesion, then crossing it can cause distal debris embolization. Proximal tight lesion can decrease distal contrast flow and so hampers the optimal visualization of stent position and deployment. The risk and benefit of each strategy, (to stent a distal or a proximal lesion first), should be assessed well before embarking on a selected path.

CAVEAT

Mismatch and Risk of Stent Dislodgment during PCI at the Insertion Site: Problems with interventions at the anastomotic site include: tortuosity of the arterial segments proximal and distal to the insertion site, difference in diameter of the segment proximal and distal to the target lesion, the degree of size mismatch between the distal vein graft and native vessel, and the angle of insertion of the vein graft into the native vessel. In case of stenting at the anastomotic site, the proximal part of the stent can be bigger, so the stent can have a funnel shape and is easier to dislodge backwards. The size of the balloon is the size of the native segment distal to the insertion site [1].

PCI IN THE ARTERIAL GRAFTS

Balloon angioplasty and stenting are feasible in arterial *in situ* (left or right internal mammary artery (IMA)) or arterial grafts removed from the radial site. In PCI of IMA grafts, hydrophilic steerable wire is helpful in the presence of tortuosity. Care must be taken to ensure that there is short guide length (80 cm) to reach distal sites with extra-long (145 cm) balloon catheters, or the guide can be shortened and capped with a flared, short sheath one size smaller [5].

TECHNIQUE Cannulation of the Left Internal Mammary Artery Graft: On many occasions, the Judkins right guide can more easily engage the subclavian artery because its primary curve is less acute. Then it is exchanged for the LIMA guide over a 0.038" wire. The usual view for cannulation of the LIMA is the anterior-posterior (AP) view, with the patient's arms down by the side. Selection of the subclavian artery is achieved by placing the guide, with or without the wire

protruding, around the arch beyond the origin of the desired artery. The guide is then gently withdrawn and rotated counter-clockwise to direct the tip superiorly until the wire or guide tip enters the subclavian origin. An angled hydrophilic wire may facilitate passage through a tortuous subclavian. The guide can then be advanced over the wire beyond the origin of the IMA, which is usually situated inferior to the thyrocervical trunk and distal to the vertebral artery. Small flush injections of contrast and gentle withdrawal of the guide can identify the location of the ostium. Gentle counter-clockwise rotation of the tip directs it anteriorly and enables it to enter the vessel selectively. If it is difficult to see the ostium, a 60º LAO or 45º right anterior oblique (RAO) projection would elongate the aortic arch, allowing excellent visualization of the origin of the IMA, so the guide tip can be engaged with precision. If it enters the left carotid artery, just withdraw it gently, it will enter the subclavian artery.

TECHNICAL TIPS

*****Engaging a LIMA Guide with a Wire:** If the IMA is difficult to engage, a slippery hydrophilic or a very steerable soft wire can be used to superselect the IMA. It then functions as a rail for cannulation of the guide. This has been necessary more commonly in the right IMA with the tip of the guide nearby. Be very gentle when cannulating the IMA because it is prone to spasm and dissection. Intracoronary nitroglycerin or verapamil can be given generously. If the subclavian artery is very tortuous, guide cannulation can be achieved through the ipsilateral radial or brachial approach.

*****Engaging the right IMA (RIMA) with a Pigtail Catheter:** After failure of engaging the very tortuous right subclavian artery from the femoral approach and from the brachial approach with standard IMA catheter, a new approach with a pigtail was tried by Lapp *et al.* A 5Fr pigtail catheter (Boston Scientific, Boston MA) was placed distally to the RIMA ostium and a long 0.014" coronary wire (Choice PT Extra Support, Boston Scientific Boston MA) was advanced through the catheter. The more the catheter was moved distally, the more the loop of the pigtail catheter straightened. With this maneuver, in the LAO 50 view, the curvature of the pigtail could be adjusted to intubate selectively the RIMA ostium. The wire was advanced into the distal part of the vessel, across the lesion. Then the pigtail catheter was exchanged for an IMA guide for PCI. This technique using a coronary wire in a 5Fr pigtail catheter allows the tip of the catheter to be shaped according to any specific anatomy [6].

*****LIMA Guide:** The VB-1 catheter was developed as a modification of a pigtail catheter. The diagnostic version of the catheter has a shape that is similar to the proximal two-thirds of the curve of a standard pigtail catheter. Proper technique during use of the VB-1 catheter is important. After obtaining access of the left subclavian artery, typically with the catheter used to image the right coronary artery,

a 175 cm length, 0.035" guidewire is positioned well beyond the origin of the LIMA and used to exchange for the VB-1 catheter. The wire is removed and the catheter flushed in the usual manner. The curvature of the distal tip of the VB-1 catheter allows it to be directed inferiorly. Slow withdrawal of the catheter toward the origin of the LIMA will allow the tip to cannulate the LIMA passively, and because of its flexible design, the catheter tip characteristically assumes a coaxial position in the proximal LIMA. In the event that the LIMA arises anteriorly from the subclavian artery, gentle counter-clockwise rotation while withdrawing the catheter directs the tip anteriorly toward the ostium of the LIMA, again achieving a coaxial position in the vessel [7].

TAKE HOME MESSAGE

Intervention of IMA graft [8]

1. Check the subclavian or the IMA in patients who are anticipating to go for CABG if there is a 20 mmHg difference in blood pressure between the two arms [9].
2. Always check the subclavian artery in post-CABG patient with angina.
3. In evaluating the LIMA, check the 90° lateral view. It may be the only view that shows the distal insertion site adequately.
4. Watch out for spasm and pseudostenosis of LIMA when instrumenting that vessel.
5. Watch for guide deep intubation of the LIMA. Watch the pressure tracing. Do no inject contrast into the LIMA if you are not sure of the position of the guide. Too-deep intubation may cause dissection.

Femoral or Radial Approach: The LIMA or RIMA can be approached by the radial approach if the take-off and proximal course of the two IMA is descending vertically or internally. Use the femoral approach if the IMA take-off is descending externally [10].

Cause of Failure of PCI in LIMA Graft: The most common cause of failure of IMA graft angioplasty is mild, moderate, and severe tortuosity of the IMA graft [11]. Mild tortuosity is defined as an isolated curve or series of curves in the graft $<90°$, moderate tortuosity when the bends make an angle between 90° and 150°, and severe tortuosity is when the graft produced a series of angles in the graft of more than 150° or an isolated turn of 360°. In the majority of IMA graft angioplasty the tortuosity is mild to moderate and does not affect the outcome [12].

TECHNICAL TIPS

***How to Overcome the Problems with Pseudolesions in Tortuous LIMA:** During PCI of a lesion in the LIMA graft, the wire caused pseudolesion. As there was no flow through the LIMA,

the angiographic assessment of the angioplasty result was difficult. Sharma *et al.* suggested exchanging the angioplasty wire with a flexible-shaft Transit catheter. Once the Transit catheter was in place and the wire was removed, the LIMA assumed its normal tortuous contour, thus leading to resolution of the "accordioning" of the vessel. Injection of contrast though the guide (with the Transit catheter in place inside the LIMA in order to maintain a channel for wire access) permitted visualization of the entire LIMA and allowed angiographic assessment of the angioplasty lesion site [13].

TREATMENT AND PREVENTION OF DISTAL EMBOLIZATION

Which Cause the Most Emboli: Angioplasty or Stent? During PCI of SVG, the embolized particles recovered from the filter basket were seen under microscopy and consisted predominantly of a necrotic core with cholesterol clefts, lipid-rich macrophages and fibrin material. This suggests that the material consisted primarily of the soft acellular atheromatous material typically found under the fibrous cap. Semiquantitative analysis of aspirates found both balloon angioplasty and stenting showed significantly greater amount of debris following balloon predilation than subsequent stenting ($p = 0.43$). Direct stenting without balloon predilation was associated with less particulate material than seen with balloon predilation alone and less than that associated with the combination of balloon predilation and subsequent stenting [14].

TECHNICAL TIPS

****How to Assure There is Total Distal Protection?** After positioning and inflation of the distal balloon of the protection device (e.g. PercuSurge, Medtronic, Minneapolis, MN), before performing PCI inject a column of contrast into the lumen to mark the location of the balloon and the proximal end of the column should be around the lesion to be dilated.While performing PCI, if that the distal column of contrast does not move, and there is no contrast seeping distally then it is assured that the balloon is being kept immobile and the distal protection is intact.

****No Flow with Distal Filter Devices:** During PCI of SVG, the filter can be overloaded with thrombi and atheromatous material, and the distal flow can be cut off. After predilating angioplasty of a SVG lesion, repeat angiography may show no flow with the contrast holding up at the filtering device. The differential diagnoses are distal embolization despite use of distal protection device; or that the wire was "choked" with embolized atheromatous materials. Therefore intermittent aspiration should be performed to relieve the overloaded filter. The export catheter syringe is first filled with saline which is injected to "agitate" the filtered materials, then it is followed by vacuum aspiration.

CRITICAL THINKING

Technical Problems with distal protection devices (DPD): Two main problems remain with distal protection device (DPD) technology. The first is that the size of the protection devices requires either predilation or dottering of the target lesion with some degree of dislodgment prior to the deployment of the DPD. In complex lesions, the "buddy wire" technique and small-sized balloon predilatation may be required The second is the lack of solution for cases in which the lesion is very close to a major bifurcation. The device can be placed in only one branch. In such a case, there will be preferential flow diversion to the unprotected branch with exacerbated embolization to that branch. The placement of an occluding balloon in the side branch results in a reversal of this trend with the flow being forced through the filter [15].

TECHNICAL TIPS

*****Improvised Distal Protection Device:** In any laboratories without the dedicated DPD, Stein *et al.* suggested a deflated over-the-wire balloon to advance beyond the index lesion (e.g. the ACE balloon, Boston Scientific, Boston MA). Inflate it to block the flow. Perform angioplasty. Be sure the patient can tolerate the ischemia due to inflation of balloon. After angioplasty, advance a large transport catheter with many side holes, and aspirate the debris from the distal blood column [16]. This strategy cannot be applied to PCI with stenting because the balloon would be trapped if not removed prior to stenting.

EQUIPMENT Proximal Protection Device: The Kerberos Embolic Protection Technology System (KEPT) (Kerberos Proximal Solutions, Sunnyvale, CA) is designed to provide proximal embolic protection during percutaneous SVG intervention. The KEPT system consists of three components: a specially constructed occluding guide, a rinsing catheter, and a mechanical rinsing device. The specially constructed 8Fr guide incorporates a compliant balloon at its distal tip. A removable obturator is available, if needed, to facilitate introduction if necessary. A diameter lumen allows routine coronary intervention and aspiration. A second lumen allows inflation of the occlusion balloon. This is an over-the-wire 4Fr catheter with multiple perforations located between two distal marker bands. The catheter has two separate lumens, one for introduction over a standard 0.014" guidewire and the other to allow irrigation of rinse solution through the external perforations. This hand-activated device allows for simultaneous irrigation and aspiration within the treatment site. The device activates two 1.5 cc syringes, which move in opposite directions for isovolumetric rinsing and aspiration (rinspiration). Heparinized saline or Ringer's lactate is injected via the rinsing catheter and aspiration

occurs through the central lumen of the occluding guide. Aspirate is collected in a bag for later examination and disposal. An 8.5 or 9Fr femoral arterial sheath is utilized. The SVG is selectively cannulated and the specially constructed guide seated deeply in the graft ostium. The occluding balloon at the tip of the guide is inflated to approximately 2 atm pressure or until the arterial waveform obtained from the guide lumen is noted to dampen, confirming proximal occlusion. A standard angioplasty wire is placed distal to the lesion. If possible, this is accomplished following proximal guide occlusion with the aid of previously determined fluoroscopic landmarks or a small injection of contrast so as to avoid wire-induced embolization. Balloon dilation or stent implantation is performed. Subsequently, the rinse catheter is introduced over the coronary guidewire, positioned distal to the lesion, then slowly withdrawn. Simultaneously, the handheld rinspirator is manually activated at the rate of 1 squeeze/sec. The proximal occlusion balloon is then deflated, restoring coronary perfusion [17].

CASE REPORT Bifurcating Distal Protection Devices: A 55-year-old man was admitted for cardiac catheterization. Coronary angiography demonstrated double-vessel disease with a 70–90% stenosis in the proximal circumflex artery and a critical thrombus-containing lesion in a dominant right coronary artery immediately proximal to its bifurcation to the right posterior descending artery (PDA) and right posterolateral artery (PLB). An 8Fr Judkins Right 4 guide was positioned and an intermediate wire was passed to the distal right PDA. A 1.5 mm X-Sizer thrombectomy device (Endicor Medical, San Clemente, CA) was placed proximal to the lesion, and aspiration was performed with only minimal benefit, as evidenced by limited thrombus extraction and improvement of the angiographic stenosis. At this point, an EPI filter wire (Boston Scientific, Santa Clara, CA) protection device was passed through the lesion and deployed in the large right posterolateral artery distal to the target lesion. Prior to the angioplasty in the target lesion, an ACE 1.5 $\times$ 20 mm balloon (Boston Scientific Scimed, Maple Grave, MN) was placed in the right PDA and inflated at low pressure, occluding the right PDA immediately distal to the bifurcation. Angioplasty was then performed to the RCA lesion with the inflated right PDA balloon preventing flow to that vessel with all flow diverted to the right PLB, which was protected with the prepositioned filter. The ACE balloon was removed and the distal RCA stented with excellent angiographic result. The filter wire was then removed. As the PDA balloon was placed slightly distal to the bifurcation upon its removal, some slow flow was noted and successfully treated with i.c. adenosine with TIMI 3Flow documented at the completion of the procedure in both branches of the RCA [15].

Treatment of No-Reflow: Angiographic no-reflow must be differentiated from other causes of diminished antegrade flow due to epicardial obstruction caused by dissection, thrombus, prolonged focal epicardial spasm, distal macroembolism, air embolism, and competitive flow from persistent collaterals obscuring satisfactory antegrade

Table 13-4 Treatment of No-Reflow

Adenosine	10–20 mcg bolus
Verapamil	100–200 mcg boluses up to 1000 mcg with temporary pacer standby
Nitroprusside	50–200 mcg bolus, up to 1000 mcg total dose

opacification. Deep seating of a guide may also cause flow diminution that mimics no-reflow. Although no-reflow is a diagnosis of exclusion, from a practical standpoint eliminating all of the possible etiologies may be difficult. An organized and systematic method is therefore the best approach to diagnosis. Multiple angiographic views to exclude an occult coronary dissection are necessary. Intravascular ultrasound can be helpful in excluding epicardial causes of delayed filling or emptying of the vessel. Some operators find measurement of the translesional pressure gradient useful to exclude significant epicardial obstruction. Others suggest the use of contrast injection into the distal vessel through a subselective catheter to delineate the angiographic status. In some cases, the diagnosis is made only after serial treatment of all of the possibilities, including additional stenting and balloon inflation, fails to resolve the impaired distal perfusion.

TECHNICAL TIPS

****Effective Injection of Drug for No-Reflow:** The operator should be aware that agents administered through the guide may preferentially distribute to areas with retained flow rather than at the site of activity. Therefore, when practical, drugs should be administered either through an infusion catheter placed distally or through the central lumen of an over-the-wire balloon catheter. Intracoronary nitroglycerin is usually suggested as the first-line agent, mainly to reverse epicardial vessel spasm, even if the blood pressure is reduced. Theoretically, nitroglycerin should have little impact on arteriolar tone and hence on no-reflow since physiologically it produces little effect in the microvasculature [18] (Table 13-4).

*****Treatment of No-Reflow with Distal Blood Aspiration:** Aspirate slowly using a 50 mL syringe, while the guide is deeply advanced into the graft. The aspiration was effective in this patient because the slugging contrast from the vein was removed into the catheter. The result was excellent, the no-reflow aspect disappeared and the ECG changes normalized, concomitant with pain dissolution [19].

TREATMENT OF THROMBUS IN SVG

In the presence of thrombus in SVG, the first option is medical treatment with drugs. If there is equipment available, then thrombectomy can be performed. Which method is better and more cost-effective?

Medical treatment of thrombus in SVG includes aspirin, clopidogrel and glycoprotein 2b3a inhibitor (GPI) such as abciximab (intragraft bolus plus intravenous infusion over 12 hours) plus fibrinolytic drug such as intragraft rtPA (100 mg over 20 minutes). The patient then is maintained on intravenous heparin over the next 48 hours for an active clotting time (ACT) between 250–300 seconds. However, data from large randomized trials are lacking. Prolonged infusion of GPI prior to PCI may allow for endogenous lysis of thrombus prior to intervention. However, this strategy is more costly because long term infusion of GPI. If a repeat angiogram at 48 hours after GPI does not show adequate resolution of the thrombus then mechanical thrombectomy is another option.

If mechanical thrombectomy is intended from the beginning, in cases of proximal thrombi, then they can be aspirated by a large guide. Smaller distal thrombi can be aspirated by a Pronto or Export catheter. AngioJet atherectomy is the most effective method for clot removal if the thrombus is large.

Distal Embolization of Thrombi: The administration of GPI or fibrinolytic agents does not modify the rate of myocardial reperfusion nor the risk of distal embolization [18]. The reason is the presence of a large burden of atheroembolic material in the SVG which may embolize any times despite the presence of GPI or fibrinolytic drugs. This is a likely explanation for the negative clinical results of GPI in SVG interventions.

In the case of mechanical thrombectomy devices, distal embolization remains a concern during wire and device passage while all disrupted thrombi may not be aspirated. Distal protection, with an occlusive balloon or filter, may be desirable but is associated with a significant risk of embolization during initial passage of the wire or device across the lesion. An additional concern would be the tendency to redirect flow (and therefore emboli) up the unprotected proximal LAD. Proximal protection with the the Velocimed Proxis™ and the Kerberos™ embolic protection systems which occlude the proximal vessel with a balloon prior to distal wire passage and intervention. Embolic material is aspirated prior to restoration of flow, potentially addressing some of the limitations of alternative approaches [17].

Currently, the only modality proven to decrease (not totally eliminate) the events in PCI of SVG is the use of distal protection devices, including the PercuSurge GuardWire and the Filter Wire [20]. Details on thrombectomy devices are discussed below.

The Aspiration Thrombectomy Device: To date, the series of devices that have been tested in clinical studies and are available on the market includes the acolysis ultrasound thrombolysis device, the excimer laser, the transluminal extraction catheter, the angiojet rheolytic thrombectomy device, the hydrolyser hydrodynamic thrombectomy device, the X-sizer helical thrombectomy device, and the rescue thrombectomy catheter. All these devices are rather complex to use and not every one is based on a rapid-exchange strategy, thus

rendering their use problematic in emergency procedures, in particular in low-volume centers.

EQUIPMENT The Pronto Device: A dual lumen rapid exchange aspiration thrombectomy catheter with a 0.056″ diameter extraction lumen. It has a rounded distal tip with a sloped extraction lumen to protect the vessel wall during advancement, and is connected to a 30 mL locking vacuum syringe for extraction of the thrombus by the Venturi effect The negative pressure should be maintained till aspiration catheter is removed from the guide. Back-bleeding will remove eventual residual thrombus fragments in the guide. Before the next passage, the aspiration catheter has to be flushed to remove thrombus fragments within the catheter [20].

EQUIPMENT The Rescue Catheter: A negative pressure suction catheter consisting of a dual lumen catheter attached to a tubing assembly. When connected to the rescue console, suction is delivered to the distal tip of the catheter, which shears the thrombus into small particles and draws them down the catheter and into a collection bottle. It is recommended that the device is positioned just proximal to the thrombus before aspiration starts. Slow movement across the thrombus with a pecking motion is suggested. If blood removal stops, the catheter should be withdrawn until blood flow resumes. If this fails to happen, the catheter should be removed, large debris should be dislodged by injection of saline into the catheter tip and the procedure repeated as necessary. This device is not recommended for patients with left main stem stenosis >50% [20].

EQUIPMENT Rheolytic Thrombectomy: AngioJet rheolytic thrombectomy (RT) is a catheter-based method for thrombus removal. The catheter is attached to a drive unit with a piston pump that generates a high-pressure pulsed flow rate of 10 000 psi at 60 cc/min through a hypotube. The hypotube ejects its saline at a loop in the catheter tip. The jets of high-velocity saline are directed back into an exhaust lumen. This creates a vortex, or Venturi effect tip (Bernoulli effect) that fragments and aspirates thrombus and loose debris. Transient bradycardia develops with RT catheter activation, particularly in right coronary or dominant circumflex lesions, and a right ventricular temporary pacemaker is recommended. The bradycardia is thought to be caused by the active release of adenosine from hemolyzed red cells. Transient ST elevation noted on the electrocardiogram during pump activation is usually due to the release of potassium from red cells, and not active ischemia. However, based on the AngioJet in Acute Myocardial Infarction (AIMI) trial, concern with the use of this device for STEMI patients has been raised, primarily due to the increased mortality and larger infarct size in the AngioJet treated patients [20].

EQUIPMENT The X-Sizer Catheter System: The X-sizer catheter system consists of a helical cutter and dual lumen catheter connected to an external passive vacuum source allowing removal of intracoronary thrombotic material. Once activated the cutter is spun

at 2 100 RPM by a motor drive unit. As the catheter is advanced in a slow fashion, the thrombus load is aspirated through various ports at the head of the catheter [20].

EXOTIC COMPLEX INTERVENTIONS FOR THE URBAN WEEK-END WARRIORS

1 PCI for Patients with STEMI within Hours of CABG: Sometimes, shortly after returning from the operating room (OR) after CABG, the patient is found to have ST segment elevation in one of the areas just bypassed. There is strong suspicion of acute occlusion of one of the bypass grafts. The patient could be taken back to the OR to recheck the patency of the bypass grafts, or the patient could go to the cardiac catheterization laboratories for emergency angiography. If there is a need for PCI, full dose of unfractioned heparin (UFH) could be given. The reason is that during CABG with the chest open, the patient was fully heparinized without extra bleeding because all the bleeding sites are well cauterized. After the chest was closed, anticoagulation was reversed with protamine. Now, when there is a need for short term anticoagulant therapy for PCI, the patient could tolerate it without problem. Urgent coronary angiography may reveal a compromised graft. During intervention, extreme care is warranted and balloon sizing should be conservative because of the possibility of suture line disruption and severe hemorrhagic complications [1]. Once a graft is thrombosed, opening of the native vessel is preferable. However, if the native vessel is not a reasonable target, balloon interventions on the graft are also effective if thrombus formation is not extensive. Intracoronary thrombolytic therapy, although technically feasible, is reported only in rare cases with one third requiring mediastinal drainage due to bleeding [21]. Therefore, removal of thrombus by a thrombectomy device is preferred. In general, no PCI could ever be done without the patient being anticoagulated [22].

2 PCI in the Subclavian Artery: On many occasions, in symptomatic patient after CABG, noninvasive studies point towards ischemia in the distribution of a left or right IMA graft. The usual sites of obstruction include the IMA graft itself, the lesion at the insertion site, or the subclavian artery. Obstructive lesion of the subclavian artery proximal to the origin of the LIMA graft can happen even it is rare (Figure 13-1). The lesion can be corrected by stenting the subclavian artery in order to relieve the ischemia in the territory supplied by the LIMA [11].

3 Perforation in SVG – No Pericardial Effusion with Hypotension: In a case report of Bretelle *et al.*, after direct stenting the angiogram revealed a diffuse rupture of the SVG with contrast leakage into the mediastinum. The patient became hemodynamically unstable with a very low blood pressure (50/30 mmHg). Volume resuscitation along with blood transfusion was used to stabilize the situation.

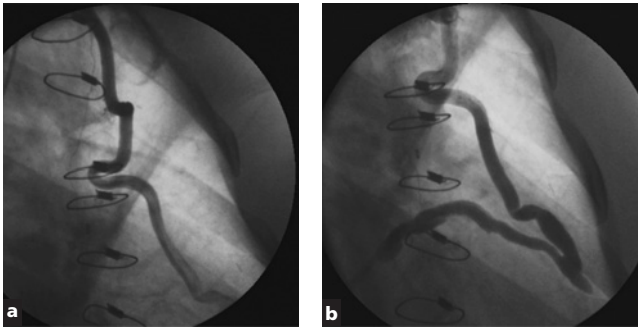


Figure 13-1 LIMA to large cardiac vein. (a) LIMA at its origin. (b) LIMA inserted into the large cardiac vein which goes into the atrium. (Courtesy of the Cardiac Catheterization Laboratories of Community Heathcare System, St Mary Medical Center, Hobart IN.)

Transthoracic echocardiography did not show any pericardial effusion. At the same time, 3 PTFE-covered Jomed stents were deployed at 14 atm. Contrast extravazation was then decreased. The CT scan showed a stable hematoma in the mediastinum without any active bleeding. Even so it compressed the RV causing hypotension [23].

4 LIMA Entrapped by Surgery: In a case report by Ternay *et al.* one month after CABG, a patient presented to the emergency room with sterile dehiscence of the sternum. The sternum was reapproximated with Robby Check Weave and closed with double wires. Four days later, he presented to the emergency department with chest pain, shortness of breath and syncope. The electrocardiogram showed diffuse ST-segment depression. His cardiac catheterization showed the native LAD to be occluded in the mid segment, and the distal LAD filled via retrograde flow from the first diagonal which was supplied by a patent saphenous vein graft. The LIMA injection revealed abrupt interruption of flow in the mid segment of the LIMA graft. It appeared that the LIMA was entrapped under the recently placed sternal wires. A balloon catheter could not be advanced beyond the point of wire entrapment. In the cardiac catheterization laboratory, the cardiothoracic surgeon removed the entrapping sternal wires under fluoroscopic guidance. Repeat angiography showed persistent interruption of flow in the LIMA graft. So the mid and distal LIMA segments were successfully dilated and stented resulting in distal TIMI 3Flow [24].

5 Luxation of Stent During Aneursym Exclusion: The SVG to the LAD was patent, however, the sequential SVG showed a 95% ostial lesion followed by a saccular post-stenotic aneurysm which measured 12 $\times$ 13 mm^2. An 8Fr Judkins Right guide and a 0.014" BMW wire (Guidant, Temecula, Calif.) were chosen for support and device delivery. The lesion was pre-treated with a 2.5 $\times$ 10 mm

followed by a 4.0 × 10 mm cutting balloon (Boston Scientific/Scimed, Maple Grove, Minn.). Repeat angiography showed enlarged entrance to the SVG and aneurysm without dissection, perforation or rupture. The ostial SVG lesion, along with the post-stenotic aneurysmal segment, were stented with a 16 mm PTFE-covered stent (Jostent, JOMED GmbH, Germany), which had been free-mounted onto a 4.0 × 20 mm Maverick 2 balloon (Boston Scientific/Scimed). After stent deployment, the distal end of the covered stent was seen to have dislocated into the aneurysm proper and the outlet of the stent was not in direct alignment with the rest of the SVG. In order to realign the PTFE-covered stent, further stent coverage extending beyond the distal margin of the first stent was clearly necessary. Another free-mounted PTFE-covered stent was deemed risky due to its relative rigidity, bulk and potential for dislodgement. Decision was made to place a conventional, low-profile, pre-mounted stent. The choice for a longer stent was to secure mid-segment axial strength where there was no mechanical support from the adjacent vascular wall. Post-dilation was performed with an excellent final angiographic outcome and minimal contrast extravasation at the distal end of the aneurysm [25].

TAKE HOME MESSAGE

Following successful SVG intervention, there is a high cardiac event rate for most patient subgroups. Success rate and long tem patency of PCI in the distal anastomotic lesions is an exception. The restenotic process in vein grafts does not plateau as it does in native coronary arteries, and mild-to-moderate non-target vein graft lesions are associated with recurrent ischemic events in about one-third of patients. If one moves from these relatively ideal candidates to the treatment of diffuse disease, recent total occlusions, the prospects for long-term patency and clinical stability diminish, while the acute risk of thromboembolic myocardial infarction, bleeding, and costs escalate. While continued study is needed to develop methods (membrane covered stent, distal protection device, thrombectomy, brachytherapy, etc.) to prolong the functional life of degenerating venous grafts, and after thoughtful cost-conscious consideration of risks and benefits and of resource consumption, day-to-day application of percutaneous strategies to these difficult problems must be approached with caution [1].

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Chapter 14
Bifurcation Lesion

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complication

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GENERAL OVERVIEW

Optimal treatment of bifurcation lesions poses a challenge in coronary intervention and is generally associated with lower immediate procedural success and worse late outcomes compared to treating nonbifurcation lesions. There is no consensus on a best strategy for treating this lesion subset. Several factors are likely to contribute to these poorer outcomes. Plaque and endothelial characteristics of ostial lesions appear to lead to increased recoil and increased risk of dissection with compromise of side and main branch (SB and MB). Intervention in either branch of a bifurcation lesion frequently leads to "snow plowing" with shifting of plaque, compromising the opposing branch. Because of these concerns, such lesions are often treated less aggressively, leading to greater residual stenosis and therefore greater restenosis. At the present time, when drug eluting stent (DES) solves the problems of instent restenosis in the MB, despite several proposed techniques of bifurcation stenting, restenosis at the ostium of the SB remains a stubborn problem [1].

STRATEGIC MAPPING

In bifurcation lesion, a soft plaque distributed toward the origin of the SB is more prone to shift toward the SB upon MB dilatation than a fibrous plaque. How will the SB behave following MB stenting? Will this behavior be influenced by the presence of disease at the origin and inside the SB? How will the plaque composition in the MB and SB affect the final result and influence the immediate and long-term result following a specific approach? Is the angle between the main and the SB important in affecting the immediate results and the fluid dynamics to

act on the long-term results [2]? Some of these factors are unrecognizable at the time of angiography and therefore cannot be quantified in a mathematical formula predicting and comparing the results of different percutaneous coronary intervention (PCI) techniques on bifurcation lesion [2].

MB Stenting with Provisional SB Stenting: After evaluating the lesion in the MB and SB and the angle of the SB, the first and probably the most important issue is when to stent only the MB and when to treat the SB with simple balloon inflation and reserving the option to stent the SB when faced with a suboptimal result. Which technique is best to use when it becomes necessary to place a second stent in the SB? The major challenge in any technique is to ensure that the SB is protected and has a satisfactory final result. Deploying a SB stent through the side of a MB stent can be technically challenging and may not be successfully achieved every time [3].

When the SB is very angulated or there is a high probability of closure with MB stenting, leaving the SB wire in place while deploying the MB (at relatively low pressures) may facilitate rewiring the SB. If the SB is dilated through the struts of the MB stent, a kissing inflation is mandatory, as this helps restore the MB geometry and expansion. Although a provisional T-stent strategy is most often utilized when SB stenting is required, there are true bifurcation lesions where the selected use of more complex bifurcation approaches (the Crush technique, the T-stenting, the Culotte technique etc.) seem appropriate, particularly when the MB and SB are larger vessels, having more diffuse SB disease [4].

Two Stent Stenting: When an operator decides to stent both the MB and SB, which is the best technique to use? The decision to implant two stents from the very beginning of the procedure depends on various factors such as the size and extension of myocardium supplied by the SB. In addition, it is important to evaluate whether the disease involves only the ostium of the SB or it extends into the proximal segment of the vessel, this last condition will more likely demand a second stent [5].

GUIDE

With the current generation of low-profile balloons and stents, bifurcation intervention may be performed with 6–8Fr guide depending on the technique employed. Provisional SB stenting can be performed through a 6Fr guide (0.07″), which is also adequate for simultaneously delivering a monorail stent and balloon or two balloons for kissing balloon (KB) postdilatation. Guides of 7Fr (0.08″) or 8Fr (0.09″) are required for two-stent techniques. These will also allow adequate KB postdilatation: up to three balloon shafts can be delivered through

an 8-Fr guide [1]. Required minimal guide lumen diameter may be calculated by adding 0.006" to the combined diameters of the largest portion of the shafts of the two balloon catheters.

WIRE

Wiring is decided by the lesion anatomy. A wire may be left "jailed" in the SB during MB ballooning and stenting to act as a landmark for rewiring through the side of the MB stent. In this situation a non-hydrophilic wire is to be used, as there is a potential risk of shearing the hydrophilic coating on wire removal. Deploying the stent at nominal pressure and subsequent postdilatation after wire removal may also be helpful [1].

TECHNICAL TIPS

****Which Branch to Wire First?** To avoid wire crossing, the most difficult branch (which is usually the SB), because it needs more manipulation, should be wired first. The second wire tends to track along the first so it should then be used to cross the MB lesion with gentle torquing (no more than a wrist rotation). An easier way is to identify each wire by having torque device with different color.

PRE-STENTING BALLOON DILATION

The best intervention of a bifurcation lesion is to stent the MB without compromising any SB. In any situation that can cause plaque shifting even if there is no lesion at the ostium of the SB, preventive low pressure balloon inflation of the SB would help to simplify the PCI at the bifurcation lesion. Usually both branches are wired and both balloons are advanced into the MB and the SB. If plaque shift from the MB is anticipated, the SB balloon is inflated first at low pressure (e.g. 2 atm). Then full inflation of the MB balloon is done. The role of the SB balloon is to prevent plaque shifting from the MB lesion. After successful inflation, both balloons are deflated, and removed proximally so a good angiogram can be done in order to evaluate the result. The major advantage of the strategy is that the atherosclerotic material in the MB is well re-arranged, and further MB stenting may not cause further plaque shifting even the MB stent would jail the SB. This strategy will also help to avoid the need of subsequent SB dilation through the side strut. The disadvantage would include denudation of the ostium of the SB or potential rupture of a plaque in the proximal part of the SB.

TECHNICAL TIPS

****Diameter of the Combined Two Balloons:** Since both balloons will inflate together in the MB proximal to the bifurcation, it is important that these balloons are not oversized for the vessel diameter or inflated to high pressures. The dilating diameter of the two balloons together will be less than the nominal diameters of both balloons together, depending on balloon and vessel compliance and inflation

pressure, and this must be considered on a lesion to lesion basis. A rule of thumb is that the diameter of the two simultaneously inflated balloons is equal to the diameter of the larger balloon plus one third of the diameter of the smaller one (i.e. 3 mm MB balloon and 2.5 mm SB balloon become: 3 mm + 2.5/3 = 3.8 mm in the proximal MB when both balloons are inflated).

PROVISIONAL SIDE BRANCH STENTING

Provisional SB stenting is elective stent implantation in the MB and when needed, the SB. If during the dilatation procedure the SB occludes, dissects, or has an impaired flow, the threshold for stenting lowers significantly. Bailout stenting of the occluding SB appears reasonable if its diameter is 2.5 mm or larger. The choice of technique is determined by the angle of the bifurcation depending on whether it is $<90°$, approximately 90°, or retroflexed and $>90°$ to the antegrade MB.

TECHNIQUE: After placement of the MB stent, if the results in the SB are poor and appear to require stenting, a wire is passed through the side struts of the MB stent into the SB. After the origin of the SB is dilated, a stent is passed into the SB and deployed with its proximal margin just at the origin of the SB. Final KB inflation is needed to correct the deformation of the main stent after SB dilation.

TECHNICAL TIPS

****Stent Design and Sidestrut Dilation:** Initial profile of the stent (crossability through MB stent struts) and visibility for achieving ideal stent positioning (good stent radioopacity or visible markers at both ends of the balloons) are other two important criteria in stent choice strategy for the branch. With rescue of side branches "jailed" within a stent, it is important to recognize differences in stent design and the result of dilation of stent cells. In open cell design, the stent struts are stretched and displaced with side lumen dilatation, resulting in a progressively larger lumen as the balloon size increases, although not all stents respond equally.

****Main Stent Deformation after Sidestrut Dilation:** It has also been demonstrated that SB dilation through a stent consistently results in narrowing of the main stent lumen immediately downstream from the SB; this narrowing increases in severity with the increasing size and inflation pressure of the balloon used for SB dilation. Redilation of the MB stent lumen alone then results in some reduction of size of the SB lumen. This problem can best be avoided by ending the procedure with "kissing" balloon dilation of both the MB and SB, although with caution to keep the proximal balloon margin within the stent and to avoid overdilation of the proximal segment of the MB stent [6].

****Where to Cross the Stent Struts:** Access to the SB through the strut of a stent is usually possible through two or three different cells (proximal, mid and distal). The cell choice influences stent deformation and the best approach is to open a more distal cell.

Therefore, the best strategy is to cross the more distal cell of the stent during wiring of SB.

****How to Manipulate a Wire to Cross a Sidestrut:** The wire should be shaped to approximately 90°; after the tip is engaged within the struts at the origin of the SB, a slight backward movement with careful steering allows crossing into the SB. In case of no success, reshaping of the tip with a wider, >90° curve should be attempted. Hydrophilic-coated wire might find less friction in crossing the struts, but the risk of dissecting the SB increases. In case of no success, a 1.5 mm, over-the-wire balloon or an open-end catheter can be advanced close to the origin of the SB to increase the support of the wire crossing the struts. This technique is especially useful for a reverse (>90°) angle of origin of a SB. The J tip is shaped manually depending on the angulation of the SB and the length of the tip on the diameter of the MB. In an "extreme angulated" lesion, it is usually impossible to enter the SB directly. In these cases, crossing first the MB lesion and then pulling back the wire orientated towards the SB so that it "jumps" into the SB ostium. Gentle torque maneuver helps to feel the lesion and cross it progressively. In case of failure to enter the SB, inflating a small balloon in the MB (with two wires in the MB) may be useful in order to facilitate wire manipulation and enter the SB.

POST STENTING KISSING BALLOON DILATION

To achieve optimal results in both vessels, a "kissing balloon" inflation can be done with simultaneous dilatations in both branches (Figure 14-1).

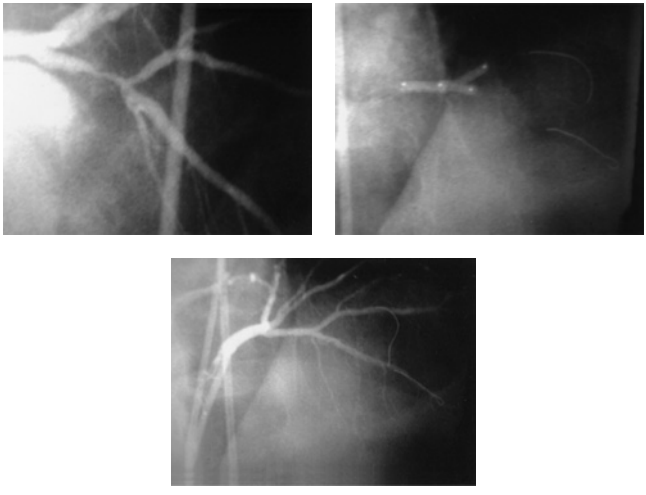

Figure 14-1 Severe lesion in proximal LAD involving first diagonal branch. Angioplasty of LAD alone would likely have compromised the diagonal. A 3.5 mm balloon in the LAD and a 3.0 mm balloon in the diagonal were inflated simultaneously, giving a good final result in both branches.

This technique may, therefore, not be suitable if the proximal vessel diameter is not larger than that of the branches involved. In this case, both branches may be dilated more aggressively sequentially with a final low-pressure kissing dilation to limit stent deformation.

TAKE HOME MESSAGE

The decision whether to implant one or two stents is an important one and has to be made early during the course of the procedure. In cases of relatively small SB, the main goal is to treat the MB; stenting the SB may not be recommended. The SB should be stented in the following circumstances: (1) significant (>50%) ostial stenosis of the SB and/or flow compromise has been observed at the initiation of the procedure; (2) symptomatic compromise (chest pain or ECG changes) of the SB observed during the course of the procedure; (3) the SB diameter is 2.5 mm or larger and supplies a significant myocardial region; (4) the SB can not be treated effectively using balloon angioplasty alone. In other words, if the SB is of adequate size and heavily diseased with an atherosclerotic lesion extending from the main lesion into the proximal portion of the branch, the strategy of using two DES may be preferred with higher chances of long-lasting vessel patency. In other cases, a provisional stenting approach should be attempted [7].

THE T-STENTING TECHNIQUE

In case of a bifurcation lesion with a SB at a 90 degree angle, the technique of T stenting is best. A wire is placed in both the MB and the SB and a stent is deployed first in the SB with the proximal stent edge at the origin of the SB, being careful this does not protrude into the MB. The wire is then removed from the SB and a stent is deployed over the wire in the MB across the origin of the SB [8–11]. The following step consists of recrossing from the MB stent into the SB and dilating the SB ostium to provide a larger stent cell opening, before performing final kissing balloon inflation. T stenting can provide excellent results when the SB originates at a right angle from the MB (Figure 14-2a). With other than a right angle takeoff, there will be either an unstented gap at the origin of the branch, or protrusion of a portion of the SB stent into the MB (Figures 14-2b,c) as happens with the Crush technique (see below).

TECHNICAL TIPS

****Which Branch Should be Stented First?** Generally the larger, more important branch is stented first, although consideration must also be given to angulation at the bifurcation. If there is marked angulation, it is preferable to stent the more angulated branch first to permit easier access into the opposing branch. If an important dissection or occlusion is present in one branch, this branch should be

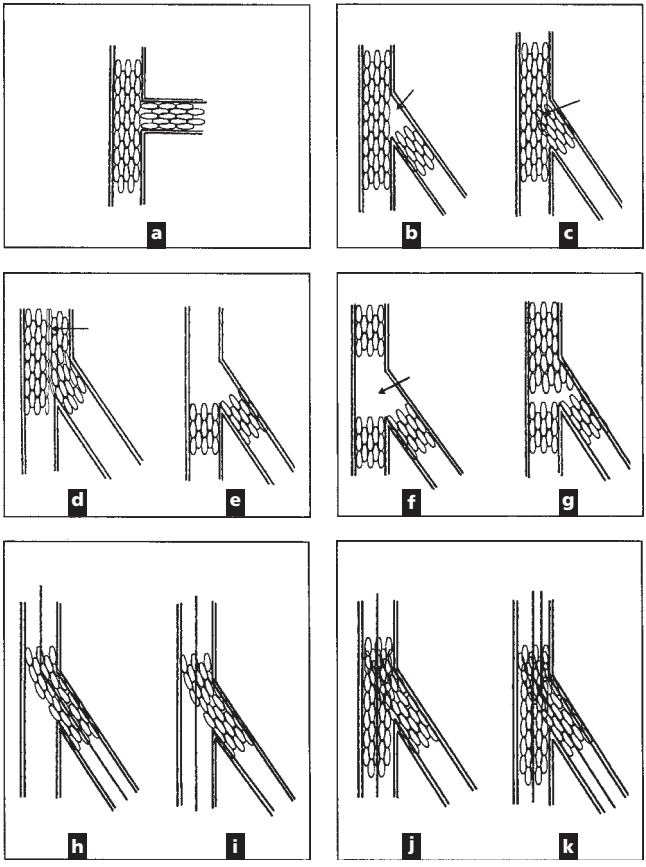

Figure 14-2 Types of bifurcation stenting. (a) T stenting with the right angle takeoff of SB providing good coverage of lesion. (b) T stenting with acute angle takeoff of SB leaving an unstented gap at origin of branch (arrow). (c) T stenting with a similar acute angle takeoff of the SB with coverage of origin but protrusion of the stent into the MB (arrow). (d) "Kissing" stenting with stents in MB and SB and creation of double-barrel lumen in proximal vessel (arrow). (e) V stenting, providing good coverage of MB and SB just beyond the bifurcation but leaving the bifurcation itself uncovered. (f) Y stenting similar to (e), but with placement of a third stent in the proximal vessel, the bifurcation itself remaining unstented (arrow). (g) "Trousers stenting" with the third stent advanced over two balloons into both distal branches overlapping the distal stents. (h) "Culotte stenting" with placement of first stent in the most angulated branch, followed by advancing a guidewire through this stent into the opposing vessel (i), stenting this vessel through the first stent after predilatation (j), finishing by recrossing the initial stent with a guidewire and doing simultaneous "kissing" balloon dilatations in both branches (k).

stented first, since wire removal might be risky. Accordingly, when the lesion remains T-shaped (which means that the bifurcation angle is >70º) after wiring, if the access to the SB is difficult or if the SB lesion is long or dissected, the SB should be stented first. If the SB is going to be stented first, it should be kept in mind that visualization of the SB ostium is sometimes difficult and before stent deployment it is therefore important to check stent position using adequate and multiple views, to avoid a too distal or too proximal SB stent deployment [12].

****Risk and Benefits of a "Jailed Wire":** In some cases, a wire can be voluntarily "jailed", left in the SB while stenting the MB. In case of occlusion, this wire can be a valuable landmark to re-cross with another wire into the SB. This technique is very useful because it helps to maintain the SB open and to favorably modify the angulation between both branches. Access to the SB after MB stenting is, therefore, facilitated. After stenting the MB, the wire in the MB (secured by the stent) is pulled back and, ideally, the most distal cell of the stent is crossed and the wire is pushed distally in the SB. Then the "jailed" wire is withdrawn from the SB (pull the guide back so there is no risk of deep intubation of the guide) and pushed into the MB [12].

THE MODIFIED T-STENTING TECHNIQUE

In order to best position a SB stent, the modified T-stenting technique is suggested.

TECHNIQUE: First, both branches are wired and alternately dilated. Then a first stent is advanced into the SB, but not expanded, and a second stent is advanced in the MB, covering the ostium of the SB. The first stent is carefully positioned at the ostium of the SB and expanded. The balloon and wire are removed from the SB and then the stent in the MB is expanded. The SB is rewired and kissing balloon dilatations of both branches are performed.

This technique allows exact positioning of the first stent at the ostium of the SB. In case of slight protrusion of the first stent into the MB while dilating the second stent, the first stent might be pushed into the SB. The second stent can be any slotted-tube stent, possibly with large struts to facilitate rewiring and dilatation of the SB. An 8Fr guide is needed for this technique. This procedure is limited to near 90° bifurcations. If the stent in the SB is implanted too distally, an uncovered gap might remain at the ostium of the SB. The other disadvantage is that both stents are positioned at the same time. Movement of one stent can disturb the exact location of the other. Then the lumen of the artery has to be well pre-dilated to accommodate two stents without causing ischemia and to have good contrast flow in order to have good evaluation of the position of the two stents including the proximal end of the SB stent at its ostium [13].

It must be noted that there is still opportunity for the SB not to be fully covered with the modified T-stenting technique as more than

three-quarters of all bifurcation lesions having angles <70° rather than the ideal 90° for this technique [13].

THE CRUSH TECHNIQUE

This technique requires a minimum 7Fr guide. Both stents in the MB and SB are positioned, with the proximal part of the SB stent lying well within the MB. Importantly, it must be ensured that the edge of the stent in the MB is more proximal than the SB stent. The SB stent is deployed first, and the balloon and wire are withdrawn while carefully ensuring the MB stent remains in position. The MB stent is then deployed thereby crushing the proximal part of the SB stent. To optimize the final result, it is recommended that the SB is rewired and a final kissing balloon dilatation is performed. It is always important to perform a "two-step" kissing balloon inflation: (a) the side branch balloon (usually a non-compliant one) is inflated first at high pressures (18–20 atm) (b) subsequently the two balloons are inflated together at medium-high pressures (15 atm). A potential limitation of the technique is the dislocation of the stent in the MB during withdrawal of the balloon and the wire after stenting of the SB. Repositioning of the MB stent might be difficult because of protrusion of the struts of the SB stent in the MB [14].

CRITICAL THINKING

When There is no Perfect Apposition of Stent at the Sidebranch Ostium in the Crush Technique: In a bench study by Ormiston *et al.*, when stents were deployed using the crush technique in a bifurcation phantom with the SB angled at 45°, the suboptimally expanded stent at the SB ostium was fully expanded by kissing balloon inflations with appropriately sized balloons. When deployment was in the bifurcation with an 80° angled SB, underexpansion at the SB ostium was more marked and not always corrected by kissing balloon inflation, especially if the SB balloon remained bent and not fully expanded at the ostium. Full expansion at the ostium was achieved with sequential (SB then MB) inflations if the SB balloon extended only several millimeters into the MB to allow it to inflate fully in a straight line without being bent. The SB balloon needed to be of the same diameter or larger than the deploying balloon to expand fully the SB stent at the ostium [15]. However, the introduction of the two-step kissing balloon inflation, with the SB balloon inflated at high pressure first, followed by simultaneous inflation with the MB balloon seems to have eliminated this problem.

TAKE HOME MESSAGE

Perfect Kissing: What is of paramount importance is to perform, before kissing, a high-pressure balloon inflation in the SB in order to be sure to expand the stent fully at the ostium of the SB. Following this step comes the kiss, which needs to be performed at medium pressure, usually 15 atm (when the two balloons are inflated together), to avoid any proximal dissection. Deflating the balloons simultaneously may also help to prevent any further distortion of the stent struts, avoiding incomplete apposition [16].

THE MODIFIED OR STEP CRUSH TECHNIQUE

The stent in the SB is positioned as above with a balloon (of similar size to the vessel reference diameter) within the MB. Again, it is important to ensure that the end of the balloon in the MB is more proximal to the SB stent. The SB stent is deployed, and its balloon and the wire are withdrawn. The SB stent is then crushed using the balloon in the MB. The SB is rewired and crossed by a balloon to open its ostial segment. Finally, the MB is stented in the usual manner, and the result was optimized with kissing balloon dilatation as described above.

The advantage of this strategy is that the initial use of a balloon in the MB, rather than a stent that is naturally more bulky, allows the use of a smaller (6Fr) guide. The modified crush technique with double kissing balloon inflation is designed to split the final kissing balloon inflation procedure into two, so that the wire and balloon have to cross only one layer of stent struts each time [17].

With the use of the modified crush, the potential limitation of stent displacement in the MB with the conventional technique is addressed. Thus, the modified crush is the technique of choice, when very accurate placement of the stent in the MB is needed or smaller catheters are required [18].

The main advantage of this modified approach is to simplify the recrossing into the SB following the initial crush of the protruding segment of the SB stent into the MB. This new approach requires a second crossing into the SB and further final kissing inflation. The advantages are a shorter time needed to recross into the SB and possibly a decreased need to use a second new balloon to perform final kissing [18].

THE REVERSE CRUSH TECHNIQUE

In the reverse crush technique, a stent is already deployed in the MB, and the SB is wired through the stent struts. After predilatation of the SB ostium, a stent is positioned in a way ensuring that the proximal part lies well within the MB. A balloon of the same size or larger than the MB stent is positioned in the MB. The stent is deployed, and the SB balloon and wire are withdrawn. The stent is then crushed with the balloon within the MB. The SB may then be rewired to enable kissing

balloon postdilatation. This technique is useful in two situations: (1) if it is felt that it might be possible to avoid routine SB stenting, but the result of the SB is poor after the MB is stented; or (2) for patients who come back with on-going problems related to restenosis at the ostium of the SB with MB stenting [18].

CRITICAL THINKING

Bench Study on the Reverse Crush Technique: This bench study, performed by Ormiston *et al.* [19], has shown that a slotted-tube stent such as the Bx Velocity stent, when internally crushed within a stent of the same (or different) design, was well applied to the wall and did not protrude into the main-stent lumen. This was confirmed by endoscopy. In contrast, the modular design (Driver/Endeavor stent, Medtronic Inc., Minneapolis, MN) when used in the SB was not well flattened inside the SB stent and struts protruded into the lumen. The internal crush strategy using the slotted-tube design stents (Bx Velocity and Express) provided coverage of the SB ostium with freedom from gaps. The SB stent was well expanded at the ostium even without postdilatation. After internal crushing and in contrast to other stenting strategies, kissing balloon postdilatation caused distortion. The distorted portions of stents at the distal margin of the ostium were pushed into the main-vessel lumen, potentially causing obstruction. This finding was true of all stent designs tested and not related to the sequence of balloon deflation. Kissing balloons did, however, retain or improve the expansion of the SB stent at the ostium. In addition, they released the SB from jail by widening the gaps between struts, potentially improving subsequent SB access. The MB distortion could be repaired by MB redilatation with an appropriately sized balloon. An alternative postdilatation strategy after internal crush that releases the SB from jail and prevents MB distortion is sequential SB and then MB post dilatation [19].

THE V STENTING TECHNIQUE

The V stenting technique is performed by placing two stents in both branches of a bifurcation with overlapping of the proximal stent portions [8,9]. Wires are placed in both branches and, with or without predilatation, stents are deployed in both branches with proximal overlap. This may be done with either simultaneous stent deployment with equal pressure in both balloons, or with sequential stent deployment followed by final simultaneous inflations of both stent balloons (Figure 14-2d). This provides good access to both branches of the bifurcation but should be used only in larger arteries and where the size of the proximal vessel will permit simultaneous high-pressure balloon inflations. With this technique, a double-barreled lumen is created in the proximal vessel with a metallic carina not opposed to

any vessel wall. This technique provides good access into both of the branches but leaves uncovered any disease in the bifurcation itself or proximal to the bifurcation [8–10].

The main advantage of the V-technique is that the operators will never lose access to either of the two branches. In addition, when a final kissing inflation is performed, there is no need to recross any stent. Different operators allow a variable amount of protrusion, creating sometimes a rather long (5 mm or more) double barrel in the proximal MB (a difference between V and simultaneous kissing technique (SKS)). It is necessary to recognize that the "V" or SKS cannot be applied in every bifurcation where two stents are needed. Important limitations are the extent of disease proximal to the bifurcation and the angle (close or larger then 90º) of the SB versus the MB.

One of the problems with this technique is that the proximal stent struts may puncture the opposing balloon. The most appropriare stents for the "V" technique are two slotted tube stents of equal design with good radial strength to preserve the best configuration of the original carina. This technique is relatively safe to perform since access to both branches is always maintained. Lesion coverage is also complete: "V" technique has more limited applications, it is best suited for very large branches with a narrow angle of origin.

THE SIMULTANEOUS KISSING STENTS TECHNIQUE

The SKS technique is best suited to easily accessible bifurcations with large proximal reference diameter containing plaque and when both branches are of similar diameter. The procedure involves wiring the MB and SB which maintains access to both during the entire procedure. Both stents are positioned side by side creating a "double barrel" configuration and are deployed simultaneously which also helps to minimize plaque shift. By having both stents parallel, this extends the carina of the bifurcation proximally. In the V-stenting technique the proximal parts of the stents are positioned to be just abutting each other thereby creating the classical "V" configuration. Both techniques have limited applicability because of the need for relatively narrow bifurcation angles and the use of large-sized guides (7Fr minimum) [2].

There is no question that the SKS technique remains the simplest and most immediate approach to treat bifurcations with DES when two stents are needed. A difference from other approaches is that this technique does not allow provisional stenting and always forces the operator to use two stents from the very beginning of the procedure. What is important is to utilize this technique when the anatomical setting is appropriate [2].

THE CULOTTE TECHNIQUE

TECHNIQUE: In "culotte" stenting, a stent is placed in one branch with a second stent placed through a cell of the first stent into the branch vessel with overlapping of the proximal portions of both stents

CRITICAL THINKING

Bench Study on the Culotte Technique:
Deployment of the SB stent through the side of the MB stent caused distortion of the latter. Balloon inflation in the MB through the side of the SB stent enlarged gaps between struts but distorted the SB ostium. Kissing balloon postdilatation repaired distortion and fully expanded the stent at the SB ostium. Examination of the interior of the stents showed that kissing balloon postdilatation produced full luminal caliber in both branches with full scaffolding and potential drug application at the ostium [19]. This technique is likely to have a good results with DES because it is a provisional SB stenting strategy that can provide full SB scaffolding and potential drug application with wide patency of the MB and SB. A limitation of all provisional stenting strategies is that it may be impossible to pass a second stent through the first stent struts, although this is likely to be easier with modular-ring designs such as the Driver stent (Endeavor) or large-cell slotted-tube designs such as the Liberté stent (Taxus, Boston Scientific, Natick, MA). In addition, cell size limits the SB diameter achievable at the bifurcation: with a close cell design stent, such as the Bx Velocity (the platform of the Cypher stent), the operator should be aware that the maximum opening of cells will be no more than 3 mm [19].

CAVEAT

Main Branch and Side Branch Stent Distortion:
Stent distortion occurred with kissing balloon inflations if the MB balloon was smaller in diameter than the delivery balloon. The distortion occurred with all stent designs and with all bifurcation strategies (crush, culotte, and single stent). The distortion was corrected by redilating the MB with an appropriately sized balloon either with or without simultaneous SB dilatation [15]. However, postdilatation, which may not be easy, is needed to expand the stent fully in the SB ostium, to widen gaps between the two layers of stent covering the SB facilitating access, and to prevent or repair SB distortion. With side branches angled at 70°, kissing balloon inflations can produce these outcomes. In contrast, with side branches angled at $>70°$, kissing balloon inflations may not expand the SB stent at the ostium. To expand the stent fully when the branch is angled at $>70°$, it may be necessary first to postdilate the SB with a balloon that protrudes only a few millimeters into the MB so the balloon is not bent and stays fully straight. After deflation of this balloon, the MB should be dilated with an appropriately sized balloon to repair any SB distortion [15].

(Figures 14-2h-k). The wires are initially placed in both branches and predilatation is performed, either sequentially or simultaneously. A stent is then placed in one vessel covering a segment proximal and distal to the bifurcation across the opposing branch. Another wire is then advanced across the deployed stent into the unstented vessel. Some operators prefer leaving the initial wire in the branch vessel during stenting of the first vessel as a guide to recrossing, although this has the disadvantage of possibly entrapping this wire. Once the unstented branch has been crossed with a wire, this is dilated with a balloon to open the stent cell in preparation for stenting of the branch. The balloon is then removed and the second stent advanced over the branch wire and positioned so as to cover the branch lesion and widely overlap the proximal portion of the previously placed stent. The wire in the first branch, having been pulled back prior to deployment of the second stent, is then readvanced across the struts of both stents into the first vessel and balloons advanced over both wires to finish with a "kissing" balloon inflation. During this final inflation, it is important to be certain that both balloons are within the proximal stent and that they are inflated at relatively low pressure, being careful not to oversize the overlapped balloons.

INTERVENTIONS OF SIDE BRANCH

Frequency and severity of SB compromise may be decreased by balloon angioplasty, rotational atherectomy, or directional coronary atherectomy (DCA) of the SB prior to stent placement, although re-dilation is frequently required following stent deployment. Compromise of small side branches of <2 mm diameter is frequently of no clinical significance and may require no particular treatment. With loss of a larger SB, a second wire can usually be passed through the side struts into the compromised branch. Some operators leave a wire in the SB at the time of stent deployment as a marker for the site of origin of the branch. The wire may occasionally, however, become entrapped with stent deployment and this technique is not all the times necessary in locating the origin of the branch occlusion. Care must be taken to avoid passing the wire behind the stent rather than through a cell into the SB. Proper positioning of the wire within the stent lumen is confirmed by easy, unobstructed passage beyond the stent. Positioning of the wire under the stent may be indicated by inability to advance a balloon catheter for SB dilation. In the case of some severe SB lesions seeming unlikely to respond to balloon dilatation, rotational atherectomy through the stent struts into the branch can be performed. When this is done, a small burr should be used initially with gradual increase in burr size to prevent burr entrapment in the branch. Frequency and severity of SB compromise may be decreased by balloon angioplasty, rotational atherectomy, or directional coronary atherectomy (DCA) of the SB prior to stent placement, although redilation is frequently required following stent deployment. Whenever restenosis occurs, this narrowing is very focal (<5 mm in length) and most of the time not associated with symptoms or ischemia [20].

TECHNICAL TIPS

****Risk of Side Branch Occlusion:** Side branches are at risk of occlusion when they are diseased at their origin and when the lesion in the MB is very close to the ostium of the SB, with possible plaque shift. Any SB of >2.0 mm should be preserved; therefore, the very first step treating a bifurcation is to decide: (1) Does the SB need wire protection? (2) Does the SB need balloon dilatation? (3) Does the SB need debulking? (4) Does the SB need a stent?

****Balloon Angioplasty of a Sidebranch:** In general, the balloon should not be advanced completely through the stent into the SB for inflation because this increases the risk of balloon entrapment. The inflation pressure should also be kept well under the rated burst pressure because balloon rupture within a stent strut can also cause balloon entrapment. If it is impossible to advance a balloon across a side strut, there is an alternative for crossing the stent strut with a balloon on a fixed wire. There is minimal transition between wire and balloon with low profile, preserving optimal "pushability," which allows comfortable strut crossing. Furthermore, this method is quick, because only one device needs to be inserted. If the balloon fails to advance, repeated, quick forward and backward movement ("dottering") of the balloon, and adjusting the guide catheter position by intubation from time to time, may help the balloon to cross [12].

****Which Balloon to be Inflated First During Kissing Balloon Inflation?** After deploying a stent through the side strut of a MB stent, there is a need of kissing balloon inflation. The SB balloon should be inflated first in order to facilitate rotation of the stent towards the right direction followed by simultaneous balloon inflation.

DIRECTIONAL CORONARY ATHERECTOMY

The removal of plaque with DCA has potential advantages in treatment of bifurcation lesions in decreasing the amount of plaque shifted toward side branches, possibly decreasing the incidence of dissection, and providing a larger, smoother lumen that could facilitate the deployment of DES.

ROTATIONAL ATHERECTOMY

Lesion debulking with rotational atherectomy rather than DCA may be of particular benefit in smaller vessels, vessels that are heavily calcified, and those with significant ostial SB lesions. Use of rotational atherectomy may decrease the incidence of dissection and SB loss.

TECHNIQUE: A guide of sufficient size to permit passage of the largest anticipated burr should be used. A Rotablator wire is generally passed first across the lesion in the MB into the distal vessel and rotational atherectomy with the smallest burr to be used performed in the MB lesion. With progressive increases in burr size, it is generally preferable to treat first one branch and then the other with the same burr, redirecting the wire from one branch to the other. In some situations in

which it is felt that access to the SB with the wire will not be difficult, the MB may be treated first with progressively increasing burr sizes with subsequent treatment of the SB.

TECHNICAL TIPS

*****Strategies for Rotational Atherectomy of Two Branches:** In general, the largest and most important limb is wired first and has rotational atherectomy with a small burr [for example, a 1.5 mm burr into a 3.0 mm limb] to secure a reliable lumen. If the lumen of the second limb is readily accessible, it can usually be accessed by withdrawing the burr just proximal to the bifurcation, placing it on "Dynaglide," and manipulating the wire directly into the branch. This maneuver is not recommended if the lumen of the second limb appears difficult to wire. After the lumen of the second limb has been secured with rotational artherectomy, a decision has to be made about the next burr size. An ideal final burr-to-artery ratio is 0.6 or 0.7.

****Wire Bias:** Wire bias resulting in dissection and/or perforation are of particular concern in side branches with an acute angle of takeoff. This may be reduced by use of a small burr and careful attention to wire bias. Wire bias in pulling or pushing a burr into or out of a lesion must be taken into account. A small burr pulled up into a lesion by a guide wire will ablate much more tissue than a small burr directed away from the same lesion.

****Side Branch Protection:** Because SB compromise is seen so infrequently after rotational atherectomy, protection of branches with a second guide wire or predilatation of side branches is not necessary. Indeed, balloon angioplasty before rotational atherectomy should be avoided because predilatation can cause dissection. Since rotational atherectomy alone usually results in a suboptimal lumen, the procedure is generally completed with a "kissing" balloon inflation that can usually be accomplished with low-inflation pressures (Figure 14-3).

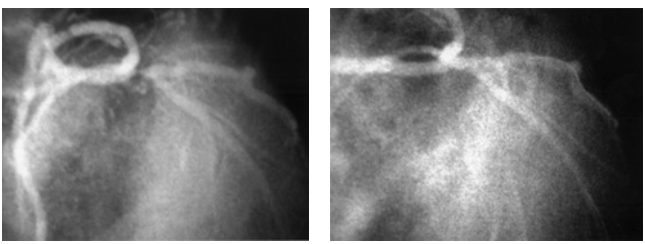

Figure 14-3 Severe ostial stenosis of the circumflex artery with ostial involvement of a large marginal branch (not well seen). PTCA alone or stenting into either branch would likely have compromised the opposing branch. Because of the ostial location of the lesion, PTCA would have given a suboptimal result and the angle of origin was into both circumflex branches, followed by a low-pressure balloon inflation, giving a good final result.

CRITICAL THINKING

Debulking for Bifurcation Lesions: The hypothesis that plaque removal is advantageous prior to stent implantation using directional atherectomy in noncalcified lesions and rotational atherectomy in calcified lesions has been attractive. However, the encouraging results of many single-center experiences have failed to be reproduced in the context of randomized studies.

The main problem of DCA is that the technique is highly operator-dependent and the amount of tissue removal varies depending on the commitment of the operator to perform extensive debulking. Atherectomy and DES can be combined when the anatomical setting is appropriate – such as a left main stenosis with a large plaque burden demonstrated by intravascular ultrasound – and plaque characteristics are suitable for removal with current directional atherectomy devices [20].

As opposed to DCA, which can be considered an optional procedure, the usage of rotational atherectomy could be, in some lesions, the only procedure to allow lesion dilatation and hence stent delivery. In most catheterization laboratories, the use of this procedure accounts for less than 5% of all interventions. Early reports found an advantage with the procedure in facilitating stent delivery and expansion with a suggestion of clinical benefit when used in lesions that demanded the use of this technology. In the majority of cases, rotablation is performed not only on the main branch, but occasionally (rarely) also, or only, on the SB. So lesion preparation of a calcified lesion can substantially facilitate stent delivery and symmetrical stent expansion with more homogeneous drug delivery [20].

A number of single-center studies reported the beneficial combination of stenting preceded by cutting balloon dilatation. In bifurcation lesions, where there is a large fibrotic plaque at the ostium of the SB, the usage of the cutting balloon as a predilatation strategy before stenting seems reasonable. Currently, cutting balloon is suggested in moderately calcified and fibrotic lesions, especially the ones involving the origin of the SB. In heavily calcified lesions, the cutting balloon could be the second step following small burr rotablation instead of using a larger burr with the goal of minimizing any distal embolization. Symmetrical stent expansion, avoidance of SB recoil, and stent compression are all attractive hypothetical results of cutting balloon atherectomy [20].

EXOTIC COMPLEX INTERVENTIONS FOR THE URBAN WEEK-END WARRIORS

1. Bifurcated Covered Stent for Coronary Aneurysm: A patient presented with exertional angina. Coronary angiography revealed single-vessel coronary artery disease and a large aneurysm of

the proximal left anterior descending (LAD) coronary artery immediately before the bifurcation with the first diagonal branch. There was severe stenosis in the artery immediately proximal to the aneurysm. The stent system was prepared as follows: a 3 × 19 mm JoStent Coronary Stent Graft (Jomed, Helsingborg, Sweden) was expanded and with a surgical blade, and an opening was created in the middle part of the stent. The opening involved a full cell of the stent. Through this opening, a 1.5 mm coaxial balloon was sandwiched with a 3.5 mm delivery balloon. The 1.5 mm balloon slightly protruded with its tip from the just-created side opening in the PTFE-covered stent. The system was advanced over the wires at the level of the bifurcation until the side balloon started to protrude into the diagonal branch. The stent was then delivered by inflating each balloon separately and then together. The coverage toward the diagonal was then completed by advancing a stent premounted on a balloon into the diagonal, creating a T-fashioned bifurcation system. Due to the nonavailability of dedicated bifurcation systems with PTFE-covered stents, a custom-made PTFE-covered stent with a custom-made side opening was used. It should be noted that extreme caution must be used not to disrupt the geometry of the PTFE-covered stent during the preparation of the described system [21].

2. Crossing the Crushed Stent With a Magnetic Wire: In the crush technique, the wire has to cross the crushed SB stent. In certain cases this can be difficult and may result in a prolonged procedure with possible complications such as contrast nephropathy and increased radiation exposure. In this patient, wiring the SB was unsuccessful despite various techniques employed, such as an over-the-wire balloon, to support the wire or by changing the angle of the wire tip manually. When a stiffer wire (PT Graphix Super Support) was used, it did not enter the true lumen but went subintimally. This was apparent after dilatation with an over the wire balloon as it resulted in a dissection at the proximal end of the deployed stent in the diagonal. Therefore, using the magnetic wire system, a three-dimensional virtual vessel roadmap was created from the angiographic images by using reconstruction software. A vector was then created that navigated a Titan™ Soft Support angled magnetic tip wire between the crushed stents' struts and avoid the false lumen. Subsequent use of kissing balloons, in the diagonal and in the LAD resulted in full apposition of the diagonal stent [22].

3. Trifurcation Stenting: A patient with history of coronary artery bypass surgery was admitted with unstable angina. The angiography showed a patent left internal mammary artery (LIMA) graft and occluded both vein grafts. There was a severe trifurcation lesion of the distal left main (LM) stem involving the ostia of the LAD and circumflex arteries and less so the intermediate vessel. It was elected to treat his trifurcation stenosis using a double-crush technique. Three wires were advanced into LAD, intermediate, and circumflex arteries respectively, via an 8Fr XB 3.5 guide catheter. The lesions were sequentially

predilated with a 2.5 mm balloon. A 3.0 × 8 mm Taxus stent was then deployed in the distal LM/LAD at 12 atm. The proximal end of this was crushed with a 3.5 mm balloon in the distal LM/intermediate. A 3.0 × 16 mm Taxus stent was then deployed in a similar manner at the ostium of the LCX (18 atm), followed by a 3.5 × 32 mm Taxus stent in the distal LM/intermediate artery at 10 atm. Sequential double kissing balloon postdilation was performed with a 3.5 mm balloon in the intermediate and a 3.0 mm balloon in the circumflex, both at 10 atm, followed by a 3.5 mm balloon in the intermediate and a 3.0 mm balloon in the LAD at 16 atm. The final result was satisfactory [23].

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Chapter 15
Complications

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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- **Management of air embolism
- **Management of massive air embolism

Intramural Hematoma
- **Take home message**

Coronary Perforation
- **Caveat**
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GENERAL OVERVIEW

During percutaneous coronary interventions (PCI), there are three possible major mechanical complications: acute or threatened closure, perforation, and no-reflow. These events can cause prolonged ischemia, hemodynamic instability, collapse, and death. The causes of acute or threatened closure are: dissection, thrombotic formation, air embolism, extra-luminal compression, antegrade aortic dissection etc. Other complications include retrograde aortic dissection, emboli to the central nervous system and reaction to contrast agents. The incidence of complications depends on the operator's skill, the technology available, and patient selection. Rigorous preventive measures preempt the appearance of complications. Operator experience, although it may be difficult to define, is extremely important in minimizing and treating the complications of PCI. With the use of current low-profile balloons and high torqueable wires, most patients with "simple" stenoses will have good results, even in the hands of relatively inexperienced operators. However, in patients with complex anatomy or when simple cases become complicated, experienced operators (who perform at least 75–150 cases per year) are likely to have superior outcomes [1]. With better equipment, stronger antiplatelet medications, and higher levels of operator experience, the incidence of complications from stenting is very low: less than 0.4% in mortality, abrupt closure, or emergent coronary artery bypass graft surgery (CABG). This is why the level of the operator's experience should dictate the case selection in patients with multiple risk factors and complex anatomy. Prevention is always the first priority because it is better to stay out of trouble rather than to get out of trouble.

THREATENED OR ACUTE CLOSURE

Threatened closure is defined as narrowing of the artery >50% during PCI with evidence of active ischemia (chest pain or electrocardiographic (ECG) changes). There are many causes of acute closure which include dissection, small side-branch occlusion, coronary spasm, air embolism, and distal embolization of plaque and/or thrombus. However, the most common cause is dissection.

DISSECTION

Dissection causes threatened closure due to excessive iatrogenic plaque fracturing from balloon inflation or device manipulation with subsequent separation of the layers of the vessel wall. A dissection can be very benign without any hemodynamic effect or it can occlude the arterial lumen, effectively stopping the blood flow. The transition between a therapeutic fracture and a threatening dissection is ill-defined [2]. The National Heart Lung and Blood Institute (NHLBI) classification of dissection is shown in Table 15-1 [3]. The incidence of coronary dissection by angiography was 10%, while it was higher with IVUS [4].

Table 15-1 Grading of Coronary Artery Dissection

Type A:	Minor radiolucency within the coronary lumen with minimal or no persistence after dye clearance
Type B:	Parallel track or double lumen separated by a radiolucent area during contrast injection with minimal or no persistence after dye clearance
Type C:	Extraluminal cap with persistence of contrast after dye clearance
Type D:	Spiral shape filling defects
Type E:	New persistent intraluminal filling defects
Type F:	Dissection leading to total occlusion without distal antegrade flow

CAVEAT

Recognizing Dissections from its Look-alikes: An intraluminal flap, or an extraluminal linear or spiral extravasion of contrast media, would suggest dissection. An intraluminal lucency with smooth contour, in an oval shape or an area with haziness, or a flat, rounded cut-off would suggest a thrombus. Spasm would have a more tapering end. Under IVUS, spasm is seen as narrowing without plaque. Other possible causes of pseudo-dissection are listed in Table 15-2.

Table 15-2 Differential Diagnoses of Dissection

Causes		Corrective techniques
1	Streaming of contrast	More forceful and steady injection
2	Deep guide intubation	Withdrawal of guide
3	Stiff wire straightening the vessel	Withdraw the wire with the flexible tip proximal to the new lesion
4	Overlapping of radiopaque wire	Withdraw the tip proximal to the new lesion
5	A thin branch running parallel to the artery	Change of camera projection

Management of Dissection: The factors defining prognosis after a dissection are: (1) the length of compromised vessel; and (2) the integrity of the antegrade flow. Minor dissection that does not compromise the coronary flow does not need treatment. If the vessel diameter is <2.5 mm, then the best strategy is to repeat prolonged low-pressure balloon inflations with a slightly oversized balloon. Dissections that are long (result in more than 50% residual stenosis), and impair flow are considered severe, and should be stented promptly, especially if the vessel diameter is >2.5 mm. Precautionary measures and tactics for prompt reversal of acute closure are listed below. Securing and maintaining wire access across the occluded artery is the single most important consideration in managing acute abrupt vessel closure. In case of spiral dissection, stenting the distal end to stop further propagation

of the dissection and the entry site to stop the source of dissection. However, some dissections cannot be stented (2–3%) because of severe proximal tortuosity, small size of the vessel, etc. The majority of dissections not resulting in acute ischemic complications heal with time, leaving no negative impact on restenosis rate [5].

TAKE HOME MESSAGE

Precautionary Measures and Tactics for Prompt Reversal of Acute Closure

1 Maintain wire across lesion.
2 Prompt balloon reinflation.
3 Prompt placement of stent to stop the dissection.
4 Ready access to contralateral artery and vein with 5F sheaths, in case hemodynamic support is needed.
5 Prior to PCI, perform lower abdominal aortogram to identify patients who may not tolerate intra-aortic balloon pump (IABP) placement.
6 IABP may be inserted prophylactically in selected patients or on standby for immediate insertion in case of hypotension or ischemic complication.

During a diagnostic angiogram, the tip of a catheter can cause dissection at the ostium. If a wire is inserted from the same catheter tip position, the wire would enter the false lumen of the dissecting plane. So look carefully at the angiographic picture when the dissection starts, then change the direction of the tip of the catheter. After that, advance the wire to the true lumen. During PCI, a dissection can happen locally due to excessive plaque fracture from balloon angioplasty or it is originated proximally at the LM or RCA ostium by a guide and propagated distally to the angioplasty site. The management of the locally dissected lesion is prompt local stenting, while the management of the ostial dissection that propagates distally is by stenting of the ostial left main (LM) or right coronary artery (RCA) first, then the distal dissected segment next. In these two situations, the wire has to be maintained across the lesion.

The antegrade propagating dissection could possibly stop at a previously stented area due to compression of the three layers of the arterial wall by the stent. If a dissection happens in the proximal and mid-segment of the left circumflex artery (LCX) that is encased inside the atrio-ventricular (AV) groove, the dissection may not commonly be propagated very far distally. However, because dissection is confined in a tight space (narrow corridor), so the luminal encroachment by the dissection is more severe (Figure 15-1). It is different from the left anterior descending artery (LAD) or right coronary artery (RCA) that lie on the epicardial surface and so dissect freely to the distal segment.

The third scenario happens when a wire is manipulated to cross a stented area and in fact is advanced outside a stent, behind the

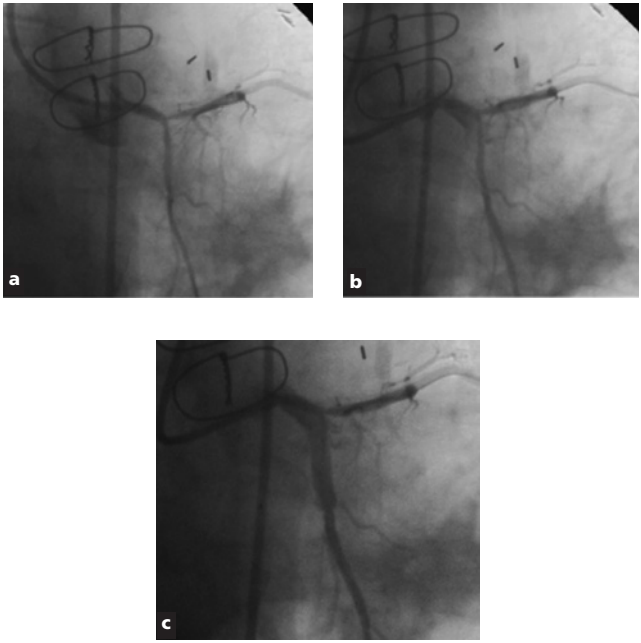


Figure 15-1 Anatomy of a LM dissection by a guide. (a) An injection that opacifies the LM and the LCX. (b) There is a lift of the entry site that propagates distally. (c) A complete dissection that encroaches the flow to the distal segment. It stops at the mid-segment of the LCX. (Courtesy of the Cardiac Catheterization Laboratories of Community Healthcare System, St Mary Medical Center, Hobart IN.)

stent struts. When the balloon is advanced behind the stent and is inflated distally, a dissection can occur and can be propagated in a retrograde or antegrade fashion. In this situation, a new wire has to enter the true lumen in order to secure a persistent true lumen access. In any situation, do not remove the wire across the lesion unless there is strong evidence that it is in the false lumen. Careful review of the angio film would show the origin of the dissection (local or ostial) and whether or not the wire is in the true lumen. A summary of these three scenarios is shown in Table 15-3.

Besides antegrade dissections from the LM, the RCA, the left internal mammary artery (LIMA) and the saphenous vein graft (SVG), a dissection can propagate distally in a retrograde fashion. The LIMA can be easily dissected at the time of performing selective angiogram or guiding catheter cannulation. This kind of dissection can be prevented by non-selective cannulation: drive the guidewire first, advance the balloon through the ostial LIMA and adjust the guiding catheter to a coaxial position with the balloon and guidewire as support devices.

Local dissection of SVG at proximal (especially SVG to RCA) or ostium part, which was caused by guiding catheter manipulation,

Table 15-3 Management of Dissection According to Site of Origin

Site of origin	Wire	Management
Ostium	Keep in place	Stent the ostium first
Local (non-ostial)	Keep in place	Stent local dissecting area
Local, in false lumen	Keep in place	*Insert second wire in true lumen *Remove first wire only after firm evidence that it is in a false lumen *Stent the narrowing area of true lumen

looks benign but can progress rapidly to ACS to AMI. When there is suspicion of retrograde dissection, pull the guide back and an injection of contrast in the coronary sinus would help to confirm the presence of retrograde aortic involvement. The management includes prompt stenting at the entry site of the dissection.

TECHNICAL TIPS

***Stent Edge Dissection:** If there is only minor dissection, there is no need for treatment. In the case of edge dissection following stent deployment, it is not imperative to cover all the edge dissections that are considered minor, with a residual lumen by IVUS larger than 50%, or not in a strategic location (not in the LM or at the ostium of a major branch) [6].

***Preventing Dissection:** In order to prevent dissection, usually the patient would have low-pressure 6–8 atm balloon predilation [7]. However, in cases of a lesion with unexpected heavy calcification, due to inadequate balloon predilation some stents cannot be deployed completely. So the key point is that for predilation, the small balloon (e.g. 2.5 mm diameter or a shorter length balloon) needs to be fully inflated without a waist at its middle. A high pressure small size balloon with a same size as predicted stent could be used both for predilation (low pressure) and postdilation (higher pressure), so that the stent could not be oversized too much to cause dissection, especially for calcified lesions. Other strategies include minimal manipulation of any devices prior to stent placement in order to limit the occurrence of dissection at the ostium or at the segment proximal to the lesion [7]. Then prompt stenting would prevent further propagation of dissection.

****Recrossing the Dissected Segment:** Once the wire position is lost, try to recross the lesion with a very soft wire rather than a stiff wire. The post-angioplasty angiogram should be reviewed carefully. Look for the plane of dissection and the most likely location of entry to the true lumen by many different orthogonal views. Then the tip of the wire is positioned at that location and manipulated to enter the true lumen. If there is a problem with recrossing the segment, or entry in the false lumen, an IVUS study should be done and the artery recrossed by a second wire parallel to the IVUS so it can be advanced

under IVUS guidance. The dissection is identified as the two lumens separated by a large tissue flap. The true lumen is confirmed by the presence of contrast agent during injection while the false lumen has no flow during injection of contrast. Many side branches can be seen coming out from a true lumen while there are none in the false lumen. A stent is to be deployed and seal the entry of the dissecting plane.

CRITICAL THINKING

Causes of Acute Occlusion after Stenting:
If stenting is the best strategy for prevention or treatment of occlusion, how can occlusion happen after stenting [8]? The usual causes of occlusion after balloon angioplasty and stenting were distal dissection (13%), and thrombus (13%). However, after stenting, 8% of patients had protrusion of tissue compromising the lumen and caused thombotic occlusion. The common denominator of these occlusions was a compromised distal blood flow promoting thrombotic formation. Then a perfect TIMI 3Flow after stenting is the best way to prevent any thrombotic complications [8].

CAVEAT

Precautionary measures that can minimize the occurrence of iatrogenic dissection include:

1. Avoid deep engagement of guide.
2. Pull the guide back a little while any interventional or diagnostic device is withdrawn from the artery.
3. Do not keep the guide too long in deep engagement position inside the LM.
4. Prompt recognition of dissection to prevent further both retrograde and antegrade extension. Always check proximal segment or ostium of instrumented artery before finishing a case.
5. In the presence of hemodynamic instability, prepare/insert IABP and call the surgeon.
6. Check the pressure waveform before every coronary injection. If a ventricularized pressure waveform is observed, the guide should be pulled out, or torqued slightly until a normal arterial pressure waveform is observed.

LEFT MAIN DISSECTION

LM dissection is the forerunner to catastrophic vessel closure. It can be precipitated by manipulation of interventional hardware in the LM ostium or during intervention of the ostial lesion of the LAD artery.

Sharp angulation at the LM-LAD junction appears to be a risk factor for LM dissection when the inflated balloon partially covers the LM [9]. The usual management of LM injury is CABG. However, it is necessary to keep the patient stable while waiting for emergency surgery. Unprotected LM revascularization is not a common procedure for the majority of operators in the US. Even so, to save the life of the patient, the acutely occluded LM has to be opened as a bailout emergency procedure, similar to pericardiocentesis in cardiac tamponade. The strategy is to open the LM even before inserting the temporary pacemaker and the intra-aortic balloon pump. The whole emergency procedure should be finished in a matter of minutes in order to reverse the process of hemodynamic collapse, shock, or impending death. Once the patient is stabilized, the decision about CABG can then be entertained (see Figure 15-1a-c).

CAVEAT

Can the LM Dissection be Missed by a Small Guide? On many occasions there is a clear discrepancy between the dramatic clinical presentation (severe chest pain, hypotension, ST-T change) with the paucity of the coronary artery findings. In these situations, additional orthogonal views need to be taken to confirm the noninvolvement of the coronary system or the presence of aortic dissection or LM dissection masquerading as acute myocardial infarction (AMI). In a report by Sakurai *et al*., a small guide could cross the ostial lesion of the LM without causing ventricularization of pressure, so the ostial lesion or dissection of the LM was missed. In situations with strong suspicion of LM dissection by ST elevation in the anterior leads and the hemodynamic collapse, repeat angiogram is to be done with a larger guide in order to detect the ostial lesion caused by dissection. Another way to detect the LM dissection is to pull the guide to barely outside the LM ostium and take an angiogram of the whole LM segment [10].

CASE REPORT Emergency Stenting of the LM Dissection: During the course of a LAD PCI, the patient developed chest pain. ECG showed ST segment elevation and an angiogram showed dissection extending into the aortic root approximately 8 cm above the coronary sinus. Subsequent angiography revealed no flow into the LAD or the LCX arteries. The patient rapidly developed severe chest pain and hypotension, and circulatory collapse appeared imminent. A new wire was rapidly advanced across the dissected LM into the LAD with rapid balloon dilation (2.5×20 mm balloon) and stenting (2.5×23 mm stent). With the stent positioned in the distal LM artery, extending into the proximal LAD, flow into the LAD was restored and the patient's hemodynamic parameters stabilized. To contain the expanding aortic dissection, a 4.0×13 mm stent was positioned at the LM ostium,

overlapping the first distal LM/LAD stent, and was deployed at 14 atm, with an excellent result.

Subsequent angiography revealed restored flow across the LM, LAD and LCX arteries. Concurrent with salvage of the LM artery, dopamine and atropine were administered. A new wire was advanced across the LM into the LCX. Balloon dilatation was performed using a 1.5 × 9 mm and a 2.0 × 20 mm balloon. Final angiography revealed a stable angioplasty result in the mid-LCX, with 30% diffuse residual disease. The two overlapping stents extending from the LM ostium to the proximal LAD were widely patent. There remained a visible dissection plane in the LM, which appeared to be stable, with no further extravasation of contrast into the ascending aorta. The patient remained hemodynamically stable [11]. The patient recovered well without surgery.

RETROGRADE AORTIC DISSECTION

Retrograde aortic dissection secondary to coronary dissection is a nightmare for any operator. Usually it happens after inflation of the proximal RCA (more common) or the LAD (Figure 15-2). Even though it is rare, it must be positively ruled out when there is unexplained chest pain or hypotension after angioplasty or stenting of any ostial or proximal lesion. If it is detected early, prompt corrective measures including

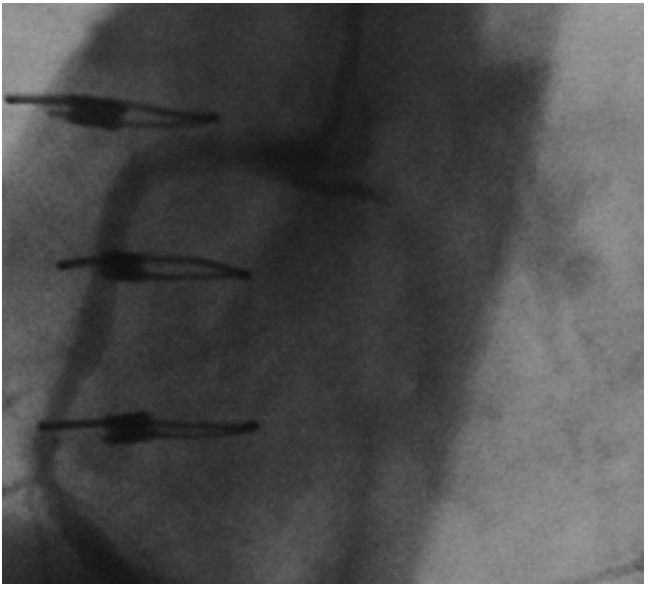


Figure 15-2 After inflation of the proximal RCA, there was persistent contrast staining at the ostium of the RCA on the aortic wall area. (Courtesy of the Cardiac Catheterization Laboratories of Community Heathcare System, St Mary Medical Center, Hobart IN.)

prompt stenting of the ostial lesion which is the entry site to seal off the dissection. Surgical consultation is needed, when there is significant aortic regurgitation, involvement of the supra-aortic vessels, and progression of the index dissection. If none of these problems is present, a watchful waiting is the best attitude [12]. Follow-up CT scan of the chest may identify the medically stabilized patient who need no further treatment or the complicated patient who may need surgery [13].

CASE REPORT Retrograde Abdominal and Thoracic Dissection from the Iliac Artery: A patient with suspected coronary disease was prepared for cardiac catheterization via the femoral approach. A standard right femoral artery puncture was performed and the wire passed easily to the mid-aorta. However, there was difficulty advancing the wire and the catheter beyond the aortic arch and the procedure was abandoned. Half an hour later, the patient complained of severe back pain and became temporarily hypotensive. She was transferred to the intensive care unit, where she was pain free but tachycardic (120 beats/minute), and blood pressure was 90/50 mmHg in both arms. A CT scan confirmed the aortic dissection, with the entry point in the iliac artery extending to the aortic arch. Initially, the patient was managed conservatively but she had recurrent transient episodes of severe back pain associated with transient hypotension (75/40 mmHg). So under fluoroscopic guidance, a 10 mm $\times$ 4 cm stent was placed in the right common iliac artery via the contralateral femoral approach. The new stent occluded the entry point. Follow-up CT showed thrombosis of the false lumen and sealing of the dissection flap. The favorable outcome in this patient was likely due to two factors: (1) the retrograde direction of the dissection, in contrast to the antegrade direction of the usual spontaneous aortic dissection; and (2) the absence of re-entry, which contributed to stagnation of blood flow in the false lumen, resulting in the formation of thrombus and the rapid disappearance of the retrograde dissection [14].

CASE REPORT Retrograde Ascending Aortic Dissection: A patient with unstable angina and carotid stenosis was prepared for PCI. A 3.0 $\times$ 20 mm balloon was advanced into the distal part of the RCA and was inflated twice to 6 atm. Subsequent angiography revealed a significant coronary dissection at the site of the lesion.

Three different types of stent were attempted to be delivered without success. An attempt to pass a perfusion balloon was also unsuccessful. In the course of our efforts to advance the stents, wire position was lost. When position was reestablished, the lesion was dilated several more times using a combination of balloons. The patient developed significant chest pain and ST segment elevation. At this time, it also became apparent that there was a guide-induced dissection of the origin of the RCA that extended proximally 7 cm into the ascending aorta. The blood pressure was 160/90 mmHg without signs of cerebrovascular (or other end-organ) compromise.

The case was discussed with a cardiothoracic surgeon. It was concluded that the risks associated with emergent CABG and aortic arch

replacement in the context of the patient's carotid disease and evolving myocardial infarction were prohibitively high and it was decided she should be treated conservatively. Therefore, the patient was transferred to the Coronary Care Unit where intravenous sodium nitroprusside was started in order to optimally manage her blood pressure. The following morning, a transesophageal echocardiogram showed an ascending aortic dissection involving the origin of the RCA and stopping at the posterior margin of the LM, extending 70 mm superiorly. Most of the false lumen appeared thrombosed apart from the superior third. There was no communication detected between the true and false lumen. There was mild central aortic regurgitation, which was known to be longstanding. A very small region of infero-posterobasal akinesia was demonstrated [15].

CRITICAL THINKING

In Patients with Retrograde Ascending Aortic Dissection, When to Send for Surgery and When to Keep Patients for Medical Treatment?

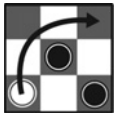
An "extensive" aortic dissection has arbitrarily been defined in the largest reported series to date as one that extends >40 mm up the aorta from the coronary cusp [15]. Applying this definition, seven of eight extensive aortic dissections reported in the literature to date have been surgically managed, with two deaths. One patient with an extensive dissection was deemed unsuitable for surgery (due to the extent of dissection and history of prior cardiac surgery) and was successfully managed with a conservative strategy. However, on this occasion it was possible to deploy stents in the culprit coronary artery, which restored vessel patency and normal anterograde flow at the time the complication was recognized, thus preventing myocardial infarction and sealing the entry site for the aortic dissection. Of the thirteen patients with dissection limited to <40 mm of the ascending aorta, twelve have been medically managed, with no deaths. Surgery was required in one patient who had suffered a limited dissection, but in this case the procedure was indicated primarily for emergent coronary bypass and not aortic repair [15]. Stenting at the site of the presumed entry site for aortic dissection in the culprit coronary vessel has been attempted in thirteen patients. The stent was successfully deployed angiographically in all of these cases. All patients survived to hospital discharge, and only two patients required subsequent surgery (in one case due to the development of hemopericardium, in the other due to the very extensive nature of the dissection, which had progressed to the aortic bifurcation) [15]. If the patient has extensive retrograde aortic dissection of >40 mm then surgery is suggested. If the dissection is <40 mm, medical therapy is suggested.

ACUTE THROMBOTIC CLOSURE

Even when the technical aspect of a PCI is almost flawless, the possibility of acute closure by uncontrolled platelet aggregation and new occlusive thrombotic formation still exists, not infrequently with superimposed vascular spasm. A thrombus is recognized as a progressively enlarging or mobile intra-luminal lucency, surrounded by contrast. Its incidence is low in stable angina patients. However, in patients with acute coronary syndrome, lesions with thrombus, long and diffuse lesions, or in degenerated vein grafts, the probability of having an acute occlusion due to thrombotic formation or distal embolization is high [16]. After stenting, acute closure due to subacute thrombosis happens if there is incomplete apposition of stent struts against the vessel wall and unrecognized mechanical obstruction proximal or distal to the stent.

To prevent thrombotic formation, in the case of a short procedure with minimal injury to the endothelium, besides anticoagulation with heparin or bivalirudin, prior treatment with oral antiplatelet drugs such as aspirin plus clopidogrel is effective. A bolus of 300 mg of clopidogrel should be given for at least 24 hours prior or 600 mg for at least 6 hours prior to the procedure. In the case of extensive injury to the endothelium by interventional hardware, the prospect of recurrent thrombosis could also be preempted by prior infusion of glycoprotein 2b3a inhibitors [17]. This is why minimal manipulation of the artery lumen prior to stent placement limits the depth and extent of vessel wall injury at the segment proximal to or around the lesion.

TACTICAL MOVE



Dissolution or Removal of Occlusive Intracoronary Thrombus

During an interventional procedure, if there is mild haziness at the lesion site or at the proximal segments, this is the early sign of thrombotic formation. At that moment the main goal is to have TIMI-3Flow, because a perfect flow is the best prevention against thrombotic formation and against shear stress which activates platelet aggregation.

1. **FIRST Best Maneuver:** When there is new haziness (small thrombi) occluding the proximal or mid-segment of an artery, some operators would make forceful injection of contrast. The goal is to dislodge the small and soft thrombi.
2. **$⧖ SECOND Best Maneuver:** Use the balloon to squeeze it, spread it to the wall.
3. **$$⧖⧖ THIRD Best Maneuver:** Removal of thrombus with thrombus extraction catheter or devices.
4. **$$⧖ FOURTH Best Maneuver – Rule Out Dissection:** Occult dissection at the lesion site or in its proximal segment has to be ruled out. If there is dissection that impedes the flow, prompt stenting is best.

5 **Treatment of Underlying Causes:** During PCI, any instrumentation of an arterial segment could break the integrity of the endothelial barrier and cause formation of thrombus. The appearance of thrombus is not the cause of the problem, it is the effect of any mechanical injury. So definitive treatment needs to address these mechanical problems such as plaque rupture, dissection, intimal denudation due to forceful jamming of stent, distal thrombotic embolization, distal traumatic push by a tip of a wire which rolls into a ball or intramural hemorrhage with opening into the lumen.

While the thrombus is being taken care of, usual emergency measures have to be taken to keep a decent blood pressure with IABP, temporary pacemaker, IV fluid, etc. activated clotting time (ACT) should be around 250 seconds.

NO-REFLOW

No-reflow is defined as stagnant contrast agent in the distal vasculature without apparent proximal obstruction. The incidence is 2% with plain balloon angioplasty (PTCA), 7% in patients undergoing rotational atherectomy, 12% for primary angioplasty, and much higher at 42% for PCI of degenerated SVG. The causes are mainly embolization of atheromatous material (gruel) and aggravated by microembolization of platelet-rich thrombi that release vasoactive agents (e.g., serotonine), leading to intense arteriolar vasospasm in the distal vasculature. The mortality of patients who developed no-reflow was 8% [18].

TECHNICAL TIPS

****Differential Diagnoses of No-Reflow:** The differential diagnosis of an apparent no-reflow phenomenon is dissection or acute thrombotic formation in the proximal segment, which is not well appreciated by conventional angiography. If in doubt, a transport or infusion catheter can be inserted through the wire and advanced to the distal segment of the no-flow area. Then the wire is removed. Pressure gradient between the tip of the microcatheter and guide is measured, and contrast injection through the end-hole will help to make the distinction between no-reflow or proximal obstructive lesions. Then injection of 3–5 cc of contrast agent with slow withdrawal of the catheter into the guide is useful to reveal any proximal disease, however hemodynamically insignificant. The results and the management are summarized in Table 15-4 [19].

Table 15-4 Differential Diagnoses of No-Reflow

Diagnosis	Proximal lesion	No-reflow	Distal lesion
Pressure gradient	(+)	(−)	(−)
Distal flow	Patent	No flow	Slow flow due to distal lesion

Results

1. If there is a pressure gradient, the cause could be due to proximal vessel obstruction or extensive intragraft pathology. The injection of contrast in the distal vasculature will show a patent distal artery. The treatment is correction of the proximal obstructive lesion.
2. If there is no pressure gradient and no single large embolus to explain the reduction of the flow, and the contrast washout remains poor in the distal bed, then the patient has no-reflow. This diagnosis of distal microvascular spasm and obstruction is a diagnosis of exclusion.
3. If there is no gradient, however, the pullback angiography could show a distal severe lesion that was not seen by conventional antegrade angiography through the guide because the contrast could not reach the distal segment due to no-reflow. The absence of the pressure gradient suggests that the disease is not flow-limiting. Correction of the lesion should resolve the no-reflow phenomenon and the symptoms of the patient.

Management of No-Reflow: The treatment includes forceful injection of blood through the guide in order to raise driving pressure across the capillary bed. Another approach is to inject small boluses of nitroglycerin (100–200 mcg: very quick try) and/or calcium channel blockers (100–200 mcg of verapamil) or adenosine (12–18 mcg). Verapamil is effective in 67% of cases in alleviating arteriolar spasm and restoring antegrade flow. Nitroprusside 40 mcg bolus up to 100–200 mcg can also be given with action to be seen in 2 minutes [20]. Epinephrine can be given especially in patients with hypotension. The dosage ranges between 50–200 mcg and multiple doses can be given and adjusted according to the presence and severity of hypotension [21]. It is important to deliver these agents into the distal artery through a balloon catheter or drug delivery catheter. Glycoprotein 2b3a inhibitors can be given as a bolus and a maintenance dose.

Preparation of Nitroprusside: One ampule of 100 mg nitroprusside (Nipride) is diluted with 250 mL of 5% DW. With a 20 cc syringe, withdraw 1 cc of the above solution and fill it with 19 cc of 5% DW (400 mcg of nitroprusside). Then give patient bolus of 3–4 cc (with 1 cc = 20 mcg) [20].

AIR EMBOLISM

The incidence of air embolism should be virtually none if meticulous safety measures are practiced. Once it happens, the patient will experience pain and hypotension similar to occlusion of a coronary artery in AMI. A small air embolus would dissipate quickly.

TECHNICAL TIPS

****Management of Air Embolism:** Strong hand injection of contrast may help to dissipate the air bubble into the distal microvasculature. Chest pain will disappear in less than 1 minute. However, if

it is a large air bubble, with an over-the-wire balloon catheter already in the guide, the operator can advance the catheter to the air bubble and aspirates the air embolus through its central lumen [21].

****Management of Massive Air Embolism:** In a case report by Colombo *et al*., 35 cc of air was injected into the LV during LV angiogram. The patient received cardio-pulmonary resuscitation (CPR) for 45 minutes then recovered with percutaneous cardio-pulmonary support (CPS) [22]. In case of air embolism in the right atrium or ventricle due to air entry during the subclavian or jugular vein cannulation, the patient should be put in the left lateral position so the air can be moved to the top of the right ventricle (RV) or right atrium (RA). A catheter then is inserted in the area and the air sucked out. In case of air embolism in the LV, then the patient should be put in the right lateral position, a pigtail catheter advanced into the LV, and the air withdrawn while having CPR.

INTRAMURAL HEMATOMA

Not infrequently, after inflation of a balloon, there is fracture of the atherosclerotic plaque, including rupture of the vasa vasorum causing formation of intraplaque, periplaque, and extra-luminal and intramyocardial hematoma. The degree of compression these hematoma have on the blood flow depends on their size. The obstruction is evident as the flow is obviously impeded although there is no sign of endoluminal dissection or thrombotic formation. The cause is to be evidenced by IVUS. The management is to stent the hemodynamically significant obstructed segment. Intramural hematoma tends to be further down toward the junction of the larger branches. In cases where intramural

TAKE HOME MESSAGE

When ECG changes and/or chest pain occur without evidence of abrupt closure at the site of the target lesion, a number of possibilities should be considered. LM dissection can be very difficult to exclude and may require multiple projections to be visualized adequately. Resistance to injection, pressure damping, severe pain, and ischemia seemingly out of proportion to lesion stenosis and hypotension, to an insignificant degree, are indirect signs of LM obstruction. IVUS may be helpful to ascertain this diagnosis when the results of the angiogram are equivocal.

Small branch occlusions can often be discovered after a detailed review comparing angiograms performed prior to the procedure side by side with those at the time of the event. Spasm usually appears at the site of the balloon inflation but may appear more diffusely in the same vessel, and even in other vessels distant from the site of mechanical interventions. Although often nitrate-responsive, spasm may be refractory and require therapy with calcium channel blockers.

Air embolism typically creates a column of contrast that terminates at a different site than that of the original stenosis; 100% oxygen is the best immediate therapy. The aspiration of air via a small intracoronary catheter may be possible if the patient does not improve in one or two minutes.

Distal embolization and the no-reflow phenomenon is a common problem to be ruled out and treated aggressively. It can happen in the distal of the index artery or at a branch proximal to the lesion or even a contralateral left coronary artery.

The appearance of a dissection of any severity is usually considered a sufficient reason to place a stent, if feasible. However, prior to the introduction of stents, operators were expected to recognize the morphology and extent of the dissection and its implications.

If none of the techniques and approaches offered above is effective in opening the artery, the operator must determine the value of emergency CABG on an individual basis. Since medical therapy is usually a euphemism for allowing the patient to sustain a myocardial infarction, this judgment is among the most difficult the interventionist must make. Before making this decision, a final attempt at redilation with a balloon catheter and perhaps the placement of a stent, if a wire remains across the occlusion, may be successful and should be considered.

The following considerations should be taken into account. Generally, the patient's overall clinical status, hemodynamic state, and the size of the jeopardized area are the primary characteristics that influence this decision. Choosing not to operate may well be an appropriate choice; if so, the reasons for this decision should be clearly explained to the patient and family [23].

hematoma at RCA is too large, an experienced CTO operator could, using stiff guidewire, make an exit point from the intramural hematoma into RCA branches to prevent enlargement of this hematoma.

CORONARY PERFORATION

Perforation of a coronary artery with a wire can be innocuous as long as the perforation is not inadvertently enlarged by a balloon. With new devices and attempts to cross chronic total occlusions (CTO), stiffer wires and laser wires harbor the risk that the wire will be forced through unrecognized subadventitial pathways into the true distal lumen. The subsequent dilation may then lacerate the adventitia and cause coronary perforation. In the most devastating scenario, there are actual rents or lacerations of the epicardial arteries with free communication of blood into the pericardial sac. This vessel rupture almost universally results in immediate hemodynamic collapse. Without control of bleeding and drainage of the pericardial sac, fatality may result [24].

Table 15-5 Risk Factors of Perforations

1	Oversizing balloon (balloon-artery ratio >1.2)
2	High-pressure balloon inflation outside the stent
3	Stenting of tapering vessel
4	Stenting of contained perforations from other devices
5	Stenting of lesions that are recrossed after severe dissection or abrupt closure
6	Stenting of total occlusion when there has been unrecognized subintimal passage of the wire
7	Stenting of small vessels (<2.6 mm)

Table 15-6 Classification of Perforation

Class	Definition	Risk of tamponade
I	Extraluminal crater without contrast extravasion	8%
II	Pericardial or myocardial "blush" without contrast agent "jetting"	13%
III	Contrast agent "jetting" through a frank (>1 mm) perforation A: Directed toward the pericardium B: Directed toward the myocardium	63%

The manifestation of perforation was delayed (5–24 hours) in 20% of patients, as seen in PCI with cutting balloon [25]. Angiographic features associated with higher risk of perforations are listed in Table 15-5 [25].

In spite of the use of glycoprotein 2b3a inhibitors, the risk of perforation and tamponade did not increase. A classification scheme is showed in Table 15-6 [26].

The treatment includes immediate inflation of the balloon at low pressure for 10 minutes (artery : balloon ratio 0.9 : 1.1) at the site of the type III perforation. For the type II perforation, without tamponade, some operators would inflate a perfusion balloon for 10–15 minutes to seal the perforation. Because of the catastrophic effect of perforation, it is critical for any operator to be experienced with the pericardiocentesis technique. If bleeding continues, inflate a perfusion balloon for 15–30 minutes. Prolonged balloon inflation is successful in 60–70% of perforations [27]. If sealing is not successful, start giving protamine in incremental doses of 25–50 mg over 10–30 minutes until ACT is <150 seconds; this should also be done in cases of jet extravasion and cavity spilling. Pericardiocentesis is to be performed if there is hemodynamic compromise. A covered stent to seal the perforation is now available in the US (JoStent Graft-JoMed International, Helsingborg, Sweden) [28]. Once there is no further dye extravasion, the patient is admitted for observation, and echocardiography should be repeated to check for further effusion. Detailed management of perforation is listed in Table 15-7.

Table 15-7 Strategies in the Management of Coronary Perforation: A Step-by-Step Approach

1	First: prolonged balloon inflation at low pressure, 2–6 atm for 10 mins. If bleeding continues, inflate perfusion balloon for 20–30 mins
2	Pericardiocentesis with a side hole catheter inside the pericardial space if tamponade
3	If bleeding continues: reversal of anticoagulation: (a) 1 mg of protamine for every 25 units of heparin given in the previous 4 hours: maximum 25–50 mg IV over 10–30 mins until ACT <150 sec
4	Covered stent for proximal or mid-segment of the perforated artery
5	Coil (material) embolization for perforation of distal end

CAVEAT

In most cases of perforation, when reversal of anticoagulation is needed, it can be done by giving protamine to reach an activated clotting time <150 seconds. Platelet transfusion is useful to reverse the antiplatelet effect in patients treated with abciximab, but not with tirofiban or eptifibatide. However, reversal of anticoagulation could lead to acute arterial occlusion or stent thrombosis. So the risk and benefit of anticoagulant reversal should be considered carefully.

When a perforation is large enough to require more than conservative treatment, two major options are available: (1) distal vessel embolization (as in some wire perforations) performed with gel foam particles, polyvinyl alcohol particles, subcutaneous fat tissues or with coils; or (2) sealing of the wall of the ruptured vessel with a covered stent. Unfortunately, the current PTFE-covered stent has limited flexibility and a large profile requiring 8Fr guide, and is not available in diameters smaller than 3.0 mm. This stent also has high restenosis rate which is treatable in the future [29].

TECHNICAL TIPS

****Preventive Measures Against Perforation by a Wire:** To avoid perforation, the tip of a wire is advanced gently without forcing against resistance. It should move freely. Once in the distal segment avoid placing the tip in small branches, for it can be inadvertently moved forward and perforate the artery. Its position should be checked frequently, especially when advancing or withdrawing any bulky devices such as long balloon or stent, or atherectomy devices.

****Preventive Measures Against Perforation due to Balloon Inflation:** After inflation of a balloon keep the deflated balloon in place, watch for the ECG to see whether it reverses to baseline and ask the patient to check the relief of chest pain caused by balloon inflation. Then make a small injection to check for severe dissection

and perforation. If there is good flow distally and no obvious extravasion of blood, then pull the balloon back into the guide. If there is any problem, dissection or perforation, then the balloon is ready to be re-advanced and re-inflated. Do not remove the balloon unless everything is clear. Wait for more than two minutes before the next inflation for ischemic precondition to kick in. If there is perforation, then inflate the balloon at low pressure. Once the patient is stabilized or has ischemia, then change to a perfusion balloon if there is an old one on the shelf (perfusion balloons are no longer produced because of little daily use).

****Management of Perforation at the Proximal and Mid-Segment:** The treatment with prolonged balloon inflation, in some fortunate cases, may permanently cover the defect with a tissue flap and solve the problem. Nevertheless, in patients with substantial tears or lacerations, a covered stent offers a viable option. Tamponade can still happen even rarely after PCI in patients with previous CABG. The reason is that there is scar formation in the pericardial area after the pericardium was opened and removed during CABG, so there is more contained perforation, with intramuscular or mediastinal hemorrhage rather than frank bleeding or effusion. If there is covered stent available, a large perforation can be successfully stopped by deploying a covered stent. Because the PTFE-covered stent is bulkier so the proximal segment should be predilated, the guide position should be optimal and extra buddy wire may be needed. The hand-crimped PTFE-covered stent should not be pushed too hard because it can be embolized.

****The Disadvantages of Perfusion Balloon Catheter:** In case of perforation, in order to stop the extravasion, the perfusion catheter can seal the lesion and permit distal perfusion. However, the perfusion catheter has important disadvantages.

1. Since they rely on intrinsic blood pressure to maintain perfusion, perfusion catheters are of limited used in patients with systemic hypotension. In order to have effective perfusion through the balloon, the systemic blood pressure should be at least above 80 mmHg.
2. They have high profile so they are not easy to advance through tortuous or calcified segments.
3. If the balloon covers a large side branch, inflation time will be limited by ischemia [30].

*****How to Make a Covered Stent with Balloon Material:** In a case of perforation where there is no PTFE covered stent, Pienvichit *et al*. suggested cutting both ends of an lightly inflated balloon in order to have a cylinder of balloon material. Then crimp a stent over another premounted stent with the balloon cylinder in between. This results in a makeshift covered stent [31].

****Reversal of Glycoprotein 2b3a Inhibition:** The degree of platelet inhibition by the small molecule inhibitors (Eptifibatide, tirofiban) is maintained through high plasma concentration which is proportional to platelet inhibition. So its effect disappears with discontinuation of the drug. In contrast, abciximab is mostly platelet-bound

with low plasma level. In order to reverse the effect of abciximab, platelet infusion is needed.

CASE REPORT Treatment of Perforation with Polyvinyl Alcohol Foam Embolization Particles: During PCI of a RCA, the tip of the Conquest wire was buckled at the proximal segment, and minimal extravasation of contrast dye was noticed from the proximal part of the RCA with peri-arterial staining without free-flowing of the dye. The patient complained of chest discomfort and became progressively hypotensive.

Immediate fluoroscopy revealed considerable dye collection in the pericardial space. Protamin 50 mg was given intravenously and emergency pericardiocentesis resulted in aspiration of a total of 200 cc of blood. Inflation of the balloon proximally to the perforation in order to seal the lesion did not result in cessation of blood flow to the pericardial space. On the contrary, further dye collection was noticed. A 2.3French TRANSIT catheter (Cordis Neurovascular Inc., Miami, FL) was advanced through a 0.014-inch Balance Middleweight 190 wire (Guidant, Santa Clara, CA) immediately proximal to the perforation site. Consequently, a 3 cc solution of polyvinyl alcohol 300 particles (300–500 microns) mixed with dye was slowly injected through the central lumen of the catheter. No more staining was observed at the distal arterial bed, suggesting thrombotic occlusion [32].

TECHNICAL TIPS

How to Embolize a Distal Vessel to Close a Perforation: The infusion of the particles can be performed through an over-the-wire balloon catheter while keeping the balloon inflated to prevent further leak. In this last circumstance, it is important to utilize a small-sized PVT particle (<300 microns) which can pass through a 0.014-inch lumen. Extreme caution is needed to prevent reflux of embolizing materials in the more proximal vessel or even to intracranial arteries in case of guide dislocation. For this specific reason, the infusion utilizing the central lumen of an over-the-wire proximally inflated balloon may be a most appropriate approach [32].

CASE REPORT Occlusion of the Distal Artery with Subcutaneous Tissue: During PCI of a RCA, after the wire was pulled back, contrast medium leaked from a small vessel of the posterolateral branch. A balloon was advanced to the posterolateral branch and inflated for 5 minutes in an unsuccessful attempt to stop the leak. After the administration of protamine sulfate reduced the ACT from 264 to 129 seconds, the balloon was reinflated for 5 minutes. However, the leak continued. Therefore, the wire was carefully and gently advanced into the penetrated vessel, and the tip of the microcatheter was placed distally to obstruct the vessel. However, obstruction for 5 min did not stop the bleeding. To embolize or to form thrombi in the penetrated vessel, subcutaneous tissue was planned to deliver to the vessel. Subcutaneous tissue was extracted from the incision site

of the right femoral region, where a sheath had been inserted. The tissue contained much fat. While the tip of the microcatheter almost completely obstructed the vessel, the tissue was put into the microcatheter and pushed with a wire until the tip of the wire protruded about 1 cm from the microcatheter. The resistance to pushing the tissue gradually decreased as the wire was advanced. After the tip of the wire had protruded from the microcatheter for 2 min, it was pulled back into the microcatheter, and the microcatheter and the wire were withdrawn from the guide. Coronary angiography showed that the small vessel was obstructed with no sign of bleeding. Subcutaneous tissue could be used as an embolization material because an autologous clot could not be created due to full dose of heparinization or the cardiac catheterization laboratories has no (microcoils, gelatin sponges, polyvinyl alcohol, microfibrillar collagen, and thrombin) material for embolization [33].

TECHNICAL TIPS

*****Dual Guide for Closure of Perforation:** In a case of PCI of the LAD, a perforation was seen in the mid-LAD. A balloon was inflated to stop the perforation. However, every time the balloon is deflated, the blood pressure dropped. The balloon was reinflated and left femoral arterial access was achieved with an 8Fr sheath. Heparin was reversed. A 7Fr or larger diameter guide was advanced to the coronary ostium. As the second guide was brought into position, the first guide had to be gently moved away from the ostium without disturbing the occluding balloon. A second wire was advanced through the second guide beyond the perforation. The occluding balloon was briefly deflated to allow passage of the wire. The covered stent was prepped and mounted, then advanced to the tip of the second guide. Then the occluding balloon deflated and withdrawn. Quickly, the covered stent was advanced to cover the perforation, the first wire withdrawn, and the covered stent then deployed at high pressure. Repeat angiography was performed to ensure closure of the perforation [34]. The Jomed hand-mounted covered stent will not track well through a 6Fr guide, especially one with moderate angulation. Covered stents >3 mm in diameter will need an 8Fr guide [35].

TACTICAL MOVE

Management of Perforation at the Distal End of an Artery

If the perforation is at the distal end of a large vessel or a branch, the treatment is by reversing the anticoagulation state, balloon tamponade of the more proximal segment if the patient can tolerate the ischemic injury, injection of thrombin to the distal branch, closure of the perforated branch with embolized material (including platelet infusate, clotted blood, or coil material).

1 $⌛ **FIRST BEST Maneuver – Balloon tamponade:** Prolonged balloon tamponade at proximal segment, if tolerated, then perfusion balloon tamponade if needed
2 $⌛ **SECOND BEST Maneuver:** If sealing is unsuccessful, then: reversing anticoagulation with protamine
3 $⌛ **Add New Drug – Platelet Product:** If the distal end needs to be sealed off, then injection of 3–4 cc of platelet infusate at the distal perforated end through the transfer catheter or through the lumen of an over-the-wire balloon. Do not inject platelet infusate at the ostium of the main artery. The whole artery can clot. There is risk of delayed bleeding (investigational).
4 $$⌛⌛♦ **Add New Device:** Covered stent can be used to block the opening of the sidebranch or closure with coil material any perforation at its distal end.
5 $⌛ **Add New Drug**s**:** Injection of thrombin, gelatin sponge (Gelfoam) or polyvinyl alcohol form (PVA) to distal segment to thrombose the distal vessel (investigational).

CRITICAL THINKING

In Case of Perforation, Which one is Better: Covered-Stent or Prolonged Balloon Inflation?
After perforation, there are choices either to inflate a regular balloon with reversal of anticoagulation by heparin or to deploy a PTFE-covered stent. If the patient still needs anticoagulation to keep the just dilated or stented lesion open then covered stent is best. If anticoagulation may not need after PCI, then reversal of heparin and prolonged inflation of a balloon to seal off the perforation is an acceptable choice. In a case report by Colombo *et al*. [36], full reversal of heparin and prolonged ischemia due to continuous balloon inflation caused slow flow in the distal bed. Later they contributed to the early thrombosis of the covered stent. So it is better to deploy a covered stent without full reversal of heparin and glycoprotein 2b3a inhibition. However, the covered stent is of higher profile, bulkier and so cannot be always advanced to distal or through tortuous segment.

CASE REPORT Left Anterior Descending Artery to Right Ventricle Fistula and Left Ventricular Free Wall Perforation: A patient with AMI underwent PCI of the LAD. After stenting, angiography revealed staining in the myocardium, contrast extravasation to the pericardium, and contrast leakage from the mid-LAD via a fistula to the right ventricle. Protamine was given intravenously to reverse the heparin effect. Pericardiocentesis was performed to relieve the cardiac tamponade. The perforation persisted after prolonged balloon inflation and was finally sealed by implanting

a 2.5–5.0 × 19 mm covered stent. Although angiography did not reveal contrast extravasation from the LAD, the patient was in persistent hypotension with continuous drainage of blood from the pericardial drain, and was therefore taken to the operating room. There was a large amount of fresh blood and clots in the pericardial space with tamponade effect. A large hematoma was present in the left ventricle (LV) over the proximal and mid-LAD territory, with arterial bleeding from two free wall perforations. The tissues were friable and attempted repair of the bleeding sites resulted in further tear at the adjacent sites. New perforations appeared after applying sutures to control the initial bleeding sites. Bleeding could not be stopped despite almost complete covering of the anterior LV wall by Teflon strips. The patient succumbed shortly after weaning off cardiopulmonary bypass. Postmortem examination showed a fistula from a small perforation in the posterior wall of the mid-LAD to the RV 3 cm below the pulmonic annulus. The anterior LV wall could not be assessed because of multiple patches and sutures.

Although the perforation in this case was apparently sealed on angiography after implanting a covered stent, there was ongoing severe bleeding into the pericardial space, as evidenced by the continued drainage of blood from the pericardial drain and persistent hemodynamics of cardiac tamponade. It was only noted in the operating room that the source of bleeding was from perforations of the LV free wall, but not from the artery, which was not suspected pre-operatively. The free wall perforations may have resulted from ischemia, leading to myocardial infarction from prolonged balloon inflation. But this case is unusual in that the ventricular wall integrity was so severely damaged that sutures could not be taken for repairing the perforations, leading to the fatal outcome despite surgical intervention [37].

CAVEAT

The Decision for Sending the Patient for CABG after Perforation: After perforation, if there is no covered stent and no embolizer, and if the bleeding does not stop after long local tamponade, the patient may need surgery. However, it is not an easy and simple procedure. Many times, the area around the perforation has intramural hematoma so the whole myocardial area is edematous and swollen. In that situation, it is almost impossible to locate the perforated branch, so the surgeon just ligates the more proximal segment and bypasses any other diseased arteries. Surgery does not reperfuse the perforated branch we try to save. Surgery may not be needed if the patient can clinically tolerate the closure of a small branch or the distal end of a large vessel. Important procedural considerations for prevention of perforation are listed in Table 15-8.

Table 15-8 Important Procedural Considerations for Prevention of Perforation

1	Constantly monitor of distal wire
2	Treat suspected perforation seriously, especially in patients on glycoprotein 2b3a inhibitors
3	Do pericardiocentesis in the CCL. Insert a 6F pigtail catheter for drainage
4	Local measures to seal perforation before leaving the CCL
5	Admit to ICU, frequent echocardiographic follow-up

Table 15-9 Cardiac Tamponade Characteristics

Clinical signs of cardiac tamponade
Tachycardia
Hypotension
Elevated jugular venous pressure with a blunted Y-descent
Pulsus paradoxus
Distant heart sounds
Physical features/signs of the underlying etiology (e.g., connective tissue disorders)
Enlarged cardiac silhouette on chest x-ray
Hemodynamic changes in cardiac tamponade
Hypotension elevation of filling pressures in all four cardiac chambers
Diastolic equalization of pressures
Blunted Y-descent in RA pressure waveform
RV and LV peak systolic pressures out of phase
Peak aortic pressure varying more than 10–12 mmHg
Decrease in cardiac output

CARDIAC TAMPONADE

In the classic model of cardiac tamponade, the pericardial space fills to a critical hemodynamic point, at which time hypotension and decline in cardiac output ensue. This schema divided tamponade physiology into three phases. In phase one, pericardial pressure equilibrates with the right ventricular filling pressure, but the cardiac output does not change. During phase two, the contents in the pericardial space continues to accumulate and the pericardial and right ventricular filling pressures rise simultaneously and equilibrate with left ventricular filling pressure, a point when there is depression of cardiac output but pulsus paradoxus is still absent. In phase three, further accumulation of pericardial fluid results in a simultaneous elevation of the pericardial, right ventricular, and left ventricular filling pressures, with further decline in the cardiac output and appearance of pulsus paradoxus. (See Table 15-9.)

Pressure Waveform: The normal pressure waveform in the right atrium displays three positive waves, A, C, and V, and two negative waves, X- and Y-descents. The A-wave is caused by atrial systole; the C-wave is caused by tricuspid valve displacement toward the right

atrium during early ventricular systole. The A- and C-waves are followed by a negative deflection, the X-descent, associated with decline in intrapericardial pressure at the beginning of ventricular systole and movement of the tricuspid apparatus toward the ventricular apex. The V-wave reflects passive atrial filling in late ventricular systole and is followed by Y-descent, coinciding with the opening of the atrioventricular valves and early ventricular diastole.

In cardiac tamponade, the RA pressure waveform has an attenuated or an absent Y-descent. Absent Y-descent is secondary to diastolic equalization of pressures in RA and RV and lack of effective flow across the tricuspid valve in early ventricular diastole. The caval (venous) flow also becomes monophasic and is confined to ventricular systole, corresponding to the X-descent. Similar to RA, the Y-descent in venous pressure waveform (during ventricular diastole) is also absent or transformed into a positive wave. The vena cava flow during ventricular systole is maintained by RA transmural pressure (RA pressure minus the intrapericardial pressure). Specifically, as the cardiac volume is reduced during ventricular systole and blood is ejected into the great arteries, the right intra-atrial pressure falls in excess of the intrapericardial pressure, producing a pressure gradient for forward venous blood flow. During ventricular diastole, the cardiac volume increases, limiting the early diastolic surge of venous return [36].

Non-Invasive Testing: Transthoracic echocardiography plays a vital role in the diagnosis and management of pericardial effusion and tamponade. As the effusion enlarges, various M-mode and 2D echocardiographic signs are used to determine hemodynamic significance and compromise. Specifically, early diastolic collapse of the RV, late diastolic collapse of the RA exaggerates respiratory variability in mitral inflow velocity (lowest velocity during inspiration) and tricuspid inflow velocity (highest during inspiration) [36].

Low Pressure Tamponade: Low-pressure tamponade may occur in a patient with hypovolemia during the setting of compressive pericardial effusion. Clinical findings may demonstrate a low to normal blood pressure with an absence of jugular venous distention or pulsus paradoxus. If a low-pressure tamponade is suspected, a fluid bolus prior to invasive hemodynamic measurement may help unmask occult tamponade or constrictive pericarditis hemodynamics [36].

Tamponade with RV and LV Dysfunction: The clinical diagnosis of tamponade in a patient with preexisting significant LV dysfunction can often be difficult. In such patients, the left ventricular end-diastolic pressure may be elevated higher than the right ventricular end-diastolic pressure and the intrapericardial pressure. Similarly, in patients with isolated right heart failure (e.g., COPD) and elevated right end-diastolic pressure, the intrapericardial pressure will increase to equal the LV end-diastolic pressure but remain lower than the RV filling pressures. Both RV and LV dysfunction may lead to absent pulsus paradoxus. The hemodynamic diagnosis of tamponade in patients with LV dysfunction

can be made when the RA and intrapericardial pressures equilibrate and track each other throughout the respiratory cycle. Likewise, in a patient with predominantly right heart failure and a high RV diastolic pressures, the pulmonary capillary wedge pressure (PCWP) and intrapericardial pressure track each other throughout the respiratory cycle [36].

Management of Tamponade: The treatment of cardiac tamponade is based on clinical presentation and may involve pericardial fluid removal by percutaneous pericardiocentesis. Pericardial space is entered via subxiphoid approach unless the effusion is loculated. A bedside echocardiogram may be performed simultaneously or immediately following the pericardiocentesis. In patients with poor LV function after pericardiocentesis, monitoring should focus on development of pulmonary edema mainly due to an abrupt increase in the pulmonary blood flow and left heart filling. If the PCWP remains elevated after complete drainage, the operator should consider preexisting cardiomyopathy. Large-volume pericardiocentesis has been reported to cause transient LV systolic dysfunction and severe RV dysfunction leading to cardiogenic shock [38].

CASE REPORT Delayed Abrupt Tamponade by Isolated Left Atrial Compression: The angiogram diagnostic showed a patent LAD stent without significant restenosis and an occlusive in-stent restenosis of a small LCX artery and second obtuse marginal artery. After pretreatment with clopidogrel and aspirin, the patient was given 70IU/kg of unfractionated heparin before angioplasty. The LCX and second obtuse marginal arteries were the site of a successful kissing balloon angioplasty. A contained type II mid LCX artery perforation, located in the atrioventricular sulcus, and type II coronary perforation of a small atrial branch induced by wire position were noted. For the following 30 minutes, the angiogram showed a contained pericoronary contrast staining without any leak. After the procedure, the patient remained asymptomatic with no hemodynamic compromise until 4 hours later, when he suddenly developed severe bradycardia and hypotension partially corrected with intravenous administration of fluid, atropine, and catecholamines. A bedside transthoracic echocardiogram demonstrated a large hematoma adjacent to the left atrium without any pericardial effusion. Cardiac tamponade secondary to a localized compression of the left atrium was suspected. The patient remained unstable and was transferred immediately to the operating room. A second echocardiogram performed immediately prior to surgery showed a marked increase in the size of the hematoma. Surgery was performed via median sternotomy and revealed a large hematoma located in the atrioventricular sulcus with compression of the left atrium. Biological glue and patch were locally applied to repair the coronary perforation. The patient had a normal postoperative recovery.

It is believed that tamponade with hemodynamic compromise occurs rarely in patients who have previously undergone cardiac surgical procedures partly due to partial pericardiectomy and secondary pericardial adhesions. The partial pericardial adhesions probably favor

migration of the hematoma with expression at a distance from the site of perforation. So the lesson is that a hematoma can compress any cardiac chamber causing any symptom of tamponade without the presence of pericardial effusion [35].

CASE REPORT When Stenting and Surgery is of no Use for Treatment of Perforation: During PCI of a LAD, angiography showed a type III B coronary artery perforation near a large septal perforator. The balloon was re-inflated proximal to the site of perforation, with a modest rise in blood pressure. An echocardiogram showed a small to moderate sized pericardial effusion without signs of cardiac tamponade. A PTFE covered stent over a second wire could not be safely positioned as even a few seconds of balloon deflation led to hemodynamic collapse. The patient was taken for emergency CABG. The site of perforation was sought but could not be identified and approximately 150 mL of blood was evacuated from the pericardium. The patient was discharged 10 days postsurgery with no deficits [39].

CASE REPORT RV Hematoma Causing Shock: Five hours after PCI of the RCA, a patient exhibited shock (60/40 mmHg) and tachycardia.Coronary angiography and right heart catheterization were done for further diagnostic clarity. The RCA was patent and without evidence of extravasation of contrast. Passage of the Swan-Ganz catheter to the pulmonary artery (PA) was very difficult, unlike the previous right heart study. Right heart catheterization revealed a pressure gradient between the PA and the RV; PA pressure = 23/15 mmHg; RV pressure = 53/25 mmHg; low cardiac index (CI; 1.90 L/min/m^2; previously 2.93 L/min/m^2), high mean RA pressure (26 mmHg), and cardiogenic shock with a systemic blood pressure of 48/36 mmHg. This hemodynamic collapse was treated with inotropic agents and adequate hydration, and a right ventriculogram was performed, which revealed compression of the right ventricular out-flow tract (RVOT). Thus, this hypotension was found to be a result of compression of the RVOT caused by hematoma. Pericardiocentesis was performed next to the left edge of the sternum into the hematoma. After inserting a 6Fr sheath, contrast medium was injected through a pigtail catheter and identified a closed cavity. A total of 411 mL blood was aspirated. Following this procedure, the pressure gradient between the PA and RV became negligible (28/17 and 30/14 mmHg, respectively; pressure gradient = 2 mmHg), RA pressure fell (26 to 12 mmHg), and CI increased (4.17 L/min/m^2) with a rise in systemic blood pressure (104/68 mmHg) [40].

CASE REPORT Fatal Dissecting Subepicardial Hematoma: Five months after stenting of the insertion site of a SVG to the LAD, a patient developed recurrent angina and repeat angiography showed diffuse in-stent restenosis. He was referred for angioplasty and vascular brachytherapy. The stenosis was crossed and dilated with a balloon at 8 atm. The stent appeared relatively underexpanded. Subsequent dilatations with another balloon at 16 atm and then a 3.0 × 10 mm Cutting

Balloon (Boston Scientific) at 14 atm resulted in complete expansion of the stent. Angiography after the final dilation revealed a large perforation at the vein graft anastomosis. The perforation was immediately sealed by inflating the Cutting Balloon inside the stent. A 2.5 × 20 mm Crossail RX balloon over a second wire was then introduced and rapidly exchanged with the Cutting Balloon to provide further sealing. Protamine 50 mg was administrated to lower the ACT to 131 seconds. After a 15-minute balloon inflation, the patient was hemodynamically stable and angiography showed that the perforation was sealed.

Over the following 5–10 min, the patient developed chest pain and bradycardia. Angiography showed thrombosis in the body of the SVG with TIMI 1Flow distally. A temporary transvenous pacing wire was placed and thrombectomy was performed with a 4Fr Angiojet device (Possis Medical, Minneapolis, MN). Heparin 5000 U was readministered to raise the ACT to 248 sec. Despite the thrombectomy, there was still evidence of thrombus in the proximal portion of the SVG, which was then treated with a 3.0 × 23 mm stent. Final TIMI 2Flow was obtained after distal delivery of 100 μg of sodium nitroprusside.

A transthoracic echocardiogram performed in the catheterization laboratory showed an echo-free space around the left lateral wall. Right heart catheterization did not reveal equalization of pressures. At this time, the patient was still experiencing chest discomfort and became hypotensive. The ECG showed diffuse ST elevation. Repeat angiography showed a patent SVG with persistent slow flow without evidence of perforation. An intra-aortic balloon pump was placed and the patient transferred to the coronary care unit.

Upon transfer to the coronary care unit, contrast echocardiography showed persistent localized collection with global severe hypokinesis. The patient was returned to the cardiac catheterization laboratory, and repeat coronary angiography was performed, showing that the LAD, which had had normal flow previously, now had markedly slow flow and did not fill distally to the apex. The SVG-D1 was patent with slow flow but no visible perforation.

Because of the patient's increasing hemodynamic instability and global ischemia, he was sent for cardiac surgery. Findings at surgery revealed neither fluid nor blood in the pericardial space. The heart was blue and distended. The epicardium was excised, showing a subepicardial hematoma cavity extending from the anterior wall to the posterior wall. Innumerable perforating branches from the epicardial vessels into the myocardium had been avulsed by the 2 cm hematoma. The hematoma was evacuated but there was persistent oozing from the subepicardial surface without any accessible vessels to cauterize. The epicardium was glued back onto the raw exposed myocardium with Bioglue and the bleeding was brought under control. Although there was no adequate site for insertion of a left ventricular assist device, the patient's hemodynamic condition had stabilized. However 3 days later the patient died.

An autopsy was performed. There was a subepicardial hematoma extending from the anterior to the posterior aspect of the heart,

compressing the native LAD. There was transmural left ventricular AMI involving the anterior, lateral, and the anterior septum and partially the posterior septum and the posterior wall. The cause of death was global AMI following a perforation of the SVG causing a dissecting subepicardial hematoma.

In the case described, the perforation was successfully controlled within seconds, but the patient continued to deteriorate until the diagnosis of the subepicardial hematoma was made in the operating room.

It is believed that the risk of subepicardial hematoma is increased in patients after cardiac surgery as a result of the adhesion of the epicardium to the pericardium [41].

CEREBROVASCULAR ACCIDENT

During the interventional procedure, embolized material to the central nervous system can cause transient ischemic attack (TIA) or disabling stroke. The lesser-known problems are transient or permanent blindness or seizure. The strongest independent predictors were use of thrombolytic prior to PCI, heparin before and after PCI, low creatinine clearance, past history of CVA and diabetes [42].

With the advent of intravenous thrombolytic therapy, direct injection of a fibrinolytic agent (investigational), or intracranial vessel angioplasty (investigational), the management of embolic stroke is changing drastically. Once embolic stroke is confirmed, fibrinolytic drugs can be given intravenously: tPA 0.9 mg/kg for a maximum of 90 mg, 10% as a bolus and the rest to be infused in 1 hour [43].

Transient and Permanent Blindness: Occipital blindness occurs rarely and usually disappears within a few hours. However, with emboli, the patient can develop permanent blindness. The MRI findings show contrast agent extravasion, without cerebral ischemia or hemorrhage. These findings are also seen in posterior leukoencephalopathy syndrome. The mechanism is a transient vasculopathy with disruption of blood–brain barrier as a cause of transient cortical blindness after contrast angiography. The main treatment is aggressive hypertension control and symptomatic treatment of headache [44].

VENTRICULAR TACHYCARDIA AND FIBRILLATION

Cardiac arrest can be caused by VT, VF or asystole. While the patient is being resuscitated with intubation, external cardiac massage, IABP, or pacemaker insertion, usually the blood pressure is sustained at an unacceptable level of 50–60 mmHg.

BEST METHOD

The One and Only Maneuver – Coronary Perfusion During CPR: During CPR, the blood pressure is low as evidenced by aortic pressure. If there is good LV function prior to cardiac arrest the chance of good recovery is high. A coronary angiogram

during CPR in a patient with asystole shows almost no flow to the coronary system. The interventional cardiologist can help coronary perfusion by keeping the guide inside the LM, gently pulling it out, withdrawing oxygenated blood to a large syringe connected to the guide and injecting this blood into the coronary system. Mix this blood with some contrast and inject to check the flow to the distal coronary vasculature and feeble movement of the LV. With good oxygenation from the ventilator (the patient is intubated), correction of electrolyte, acid–base imbalance, and opening of the acute occluded artery (which causes the cardiac arrest), coupled with forced coronary perfusion by injection of oxygenated blood, the chance of recovery is higher. Try not to cause dissection of the LM with movement of the guide.

TECHNICAL TIPS

*****How to Differentiate between Ventricular Tachycardia and Supraventricular Tachycardia by Intracardiac ECG:** To record an intracardiac ECG, a 0.014" Choice Floppy angioplasty wire (Boston Scientific/Scimed Maple Grove MN), is placed in the lumen of a 6Fr Multipurpose catheter with the wire tip minimally protruding. The proximal end of the wire is attached to a surface V1 ECG lead with a sterile alligator clamp. During continuous ECG and pressure monitoring, the catheter is manually withdrawn from the right ventricle and right atrium [45]. The RV ECG complex is wider and the RA ECG complex is smaller. VT is seen as RV wide complex with AV dissociation. RA ECG complex is seen in accordance with RV complex in SVT.

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Chapter 16
Interventions in Patients with Bleeding or Bleeding Tendency

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ < 100.00 $US extra; **$$** > 100.00 $US extra
⌛ < 10 minutes extra; ⌛⌛ > 10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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In general, percutaneous coronary intervention (PCI) is contra-indicated in patients with bleeding and those that bleed easily because during PCI the patient needs full anticoagulation to counter any thrombotic formation caused by introduction and manipulation of devices in the vascular system. The patients who currently bleed may not tolerate any short term anticoagulant effect. The patients who bleed easily may have annoying and prolonged bleeds especially at the surgical or vascular access site while on long-term antiplatelet drugs (clopidogrel or aspirin (ASA)). These patients in critical situation such as acute myocardial infarction (AMI) or unstable angina may need to undergo PCI, in spite of the fact that the operators have difficulty in predicting the risk of or controlling further bleeding before or during PCI. Any patients whose bleeding cannot be controlled afterwards should not undergo PCI because they will succumb to hemorrhagic shock. Such patients are listed in Table 16-1.

Table 16-1 Patients Having Problems with Anticoagulant or Antiplatelet Drugs

Major Bleeding Problems during PCI and Subsequent Non-Cardiac Surgery
1 AMI patients with active bleeding from compressible sources
2 AMI patients with active bleeding from non-compressible sources (gastrointestinal, cerebral, renal, etc.)
Major Bleeding Problems During PCI
1 AMI patients with recent surgery
2 AMI patients with recent stroke
Minor Bleeding Problems with Long Term Antiplatelet Drugs
1 AMI patients on warfarin
Major Bleeding Problem with Short Term Anticoagulant and Minor Problems with Long Term Antiplatelet Drugs
1 Thrombocytopenia from liver disease
2 Idiopathic thrombocytogenic purpura
3 Hemophilia A
4 Hemophilia B
5 von Willebrand's

There are many options for reperfusion of the infarct-related artery (IRA) in AMI patients with bleeding. These options differ from or complement each other. Their benefits and risks, advantages and disadvantages are presented and discussed in Table 16-2.

ANGIOPLASTY ALONE WITHOUT STENTING

In general, PCI is contra-indicated for patients with bleeding or bleeding diathesis because during PCI, full anticoagulation is needed. However, if the risk of mortality from AMI is higher than the risk of complications from bleeding during PCI, the AMI patient should undergo plain balloon angioplasty (PTCA) with or without stenting in the IRA. The benefits and risks of PTCA alone without stenting are highlighted in the case study below.

CASE REPORT Angioplasty without Stenting for AMI Patients with Recurrent Gastro-intestinal Bleeding: An elderly patient with AMI was admitted with chest pain and mild hematemesis. The electrocardiogram (ECG) showed ST segment elevation. The gastroenterologist refused to do gastroscopy because of ongoing AMI. As the risk of mortality from AMI is estimated to be higher than the risk of complications from further bleeding, the patient successfully underwent PTCA of the IRA with a single bolus dose of unfractioned heparin (UFH) and ASA. After PTCA, the condition of the patient was more stabilized, so the patient could successfully undergo gastroscopy for ligation of the bleeding artery with a Hemoclip device (Medtronic, Natick MA). Because the patient had only PTCA, the patient was given ASA, without UFH and clopidogrel, after the procedure.

The practical implication is whenever a patient has bleeding problems with prolonged anticoagulant or antiplatelet therapy, the patient should undergo PTCA without stenting. After the procedure, only ASA (without clopidogrel) is needed. This is the most appropriate strategy for AMI patients with: (1) recurrent and active bleeding; (2) after recent surgery; or (3) after an ischemic stroke. How long will the beneficial effect of an opened IRA by PTCA alone last? What is the main concern in the short term for AMI patient undergoing PTCA without stenting?

Re-Occlusion for AMI Patients Undergoing PTCA: The main concern for AMI patients undergoing only PTCA without stenting is

Table 16-2 Procedural and Pharmacological Options for PCI in Patients with Bleeding

1	Angioplasty (PTCA) without stenting
2	Angioplasty with bare-metal stent
3	Angioplasty with drug-eluting stent
4	Stenting with heparin (UFH)
5	Stenting with bivalirudin (DTI)
6	Stenting with fondaparinux
7	Stenting with ASA and clopidogrel

re-occlusion. This question was asked in the Controlled Abciximab and Device Investigation to Lower Late Angioplasty Complications (CADILLAC) trial. In this trial, 2,082 patients with AMI were randomized in a 2 × 2 factorial design to primary stenting or to balloon angioplasty, each with and without abciximab. At a median of 2 days (range 0 to 23), early re-occlusion occurred in 0.5% of patients who had been randomized to undergo stenting, versus 1.4% of those who underwent PTCA alone ($p = 0.04$). After PTCA, these patients should be followed-up closely for early re-occlusion because they require and benefit from repeat PTCA. Complex baseline lesion morphology and small vessel size are angiographic predictors of early re-occlusion if the patient undergoes only PTCA without stenting [1].

PTCA ALONE FOR AMI PATIENTS AFTER RECENT SURGERY

Besides the patient with bleeding, the strategy of PTCA alone without stenting could also be applied to patients in whom AMI occurs after a recent surgical procedure. The proximity of the surgery to PCI places this patient at high risk for hemorrhagic complications at the operative site.

CASE REPORT PCI for AMI Patient after Recent Surgery: A patient underwent knee surgery. One hour later, while the patient was in the recovery room, the patient complained of chest pain. An EKG showed ST elevation in V leads. An emergency coronary angiography showed complete occlusion of the LAD. The patient was then given an initial 0.75 mg/kg bolus of bivalirudin followed by a 1.75 mg/kg/hour infusion and underwent a successful PCI with ASA and clopidogrel [2].

This case study raises two questions: (1) Do all the patients benefit from PCI for AMI that occurs after a surgical procedure? (2) Do these patients have less local- and intra-cranial bleeding with UFH or with bivalirudin (direct antithrombins, DTI)?

Benefit and Risks of PCI for AMI Patients after Recent Surgery: Between 1990 and 1998, 48 consecutive patients at Mayo Clinics underwent emergency coronary angiography and interventions due to AMI within 7 days after a non-cardiac surgery. The types of surgery included intra-abdominal, orthopedic, vascular, urologic, and neurologic procedures. Shock was present in 21 patients, and cardiac arrest occurred in 12 before angiography. Of the 41 patients who underwent angioplasty, the procedure was successful in 30. None had bleeding at the surgical site in the catheterization laboratory. Thirty-one patients (65%) survived to be discharged. Of the 21 patients with shock before catheterization, 11 survived. Nine of 12 patients with cardiac arrest before angiography survived. Although nine patients required red blood cell transfusion, only one developed bleeding at the surgical site requiring treatment [3. Survival of 11 of 21 patients with profound cardiogenic shock is encouraging when compared to the outcome of patients receiving reperfusion therapy in the SHOCK trial [4]. Steps to

minimize bleeding at the surgical site, including weight adjustment of UFH or DTI, should be taken to decrease risk. Between UFH and DTI, which one causes less bleeding?

UFH vs DTI for PCI in AMI Patients: At this present time, the risks of any anticoagulation by UFH or bivalirudin, or of antiplatelet agents such as aspirin or clopidogrel, for AMI patients after a recent non-cardiac surgery, an ischemic stroke or with gastrointestinal GI bleed are clearly not quantifiable from the scarce data available in the literature [4]. For these patients, only anecdotal reports showed that bivalirudin and clopidogrel in the immediate post-operative period did not impose an increased risk of bleeding [4]. The understanding and rationale of treatment in these cases were translated from data of patients in large clinical trials in which the majority of high risk patients were excluded. In the REPLACE-2 trial of patients undergoing PCI without high-risk features, bivalirudin was tested against UFH and a glycoprotein (GP) 2b3a inhibitor. The primary end point at 30 days included major bleeding plus the usual end points of death, MI, and urgent revascularization. These events occurred in 9.2% of the bivalirudin group and 10% of the group given UFH plus GP 2b3a inhibitors (nonsignificant). The secondary end point was freedom from death, MI, and urgent revascularization and occurred in 7.6% of the bivalirudin group and 7.1% of the group given UFH plus GP 2b3a inhibitors (also nonsignificant), but bleeding (combined major and moderate bleeding) was significantly reduced in the bivalirudin group (from 7.1% to 2.4%, $p < 0.001$) [5]. So the rationale of using bivalirudin as antithrombotic is that DTI was not inferior to UFH in clinical outcomes, while it caused less bleeding. Bivalirudin also has a rapid onset of action, a short 25-minute half-life and a predictable dose response that allowed for early sheath removal [6].

As PTCA alone could be done successfully with DTI without the need of long term clopidogrel, for AMI patients after recent surgery, does UFH or DTI provide a better outcome for PCI in AMI patient with recent stroke?

Concern of Further Bleeding by Anticoagulant During PCI for AMI Patients with Recent Stroke: If the patient with recent ischemic stroke develops AMI, the patient could undergo PCI under the coverage of short term anticoagulant (UFH or DTI) and long term oral antiplatelet drug. However there are two concerns: (1) The risk of hemorrhagic conversion of the ischemic stroke with anticoagulant therapy; and (2) risk of cerebral emboli from the protruding plaque in the aortic arch if they were the cause of emboli stroke in the first place.

In the REPLACE 2 trial, AMI patients receiving bivalirudin did better than patients receiving UFH. However, no patients had recent stroke, so the question of hemorrhagic conversion could not be definitively answered [5]. Due to the risk of intracranial bleed from single or double antiplatelet agents, placement of a drug eluting stent (DES) or bare metal stent (BMS) should be avoided if possible. Both kinds of stent

obligate the patient to take ASA and clopidogrel. An adequate PTCA result would have accomplished the acute goal of restoring flow to salvage the myocardium without the need for clopidogrel. The higher rate of restenosis associated with PTCA in comparison to stenting would have been less of an acute concern than the increased risk of bleeding associated with the addition of clopidogrel [4]. However, the 1.4% rate of early acute and subacute thrombosis at the PTCA site is a *real* and great concern.

In the OASIS-6 trial, the goal was to evaluate the treatment with fondaparinux compared with control (UFH or placebo) among STEMI patients. Patients were randomized to either fondaparinux (2.5 mg/day for up to 8 days or hospital discharge; $n = 6036$) or control ($n = 6056$). Patients were classified as Stratum 1, meaning UFH was not indicated, or Stratum 2, meaning UFH was indicated. Patients in Stratum 1 received fondaparinux or placebo; patients in Stratum 2 received fondaparinux or UFH. The primary endpoint of death or MI at 30 days was lower in the fondaparinux group compared with the control group (9.7% vs 11.2%, hazard ratio (HR) 0.86, $p = 0.008$). Among the components of the composite at 30 days, mortality was lower in the fondaparinux group (7.8% vs 8.9%, HR 0.87, $p = 0.03$), and reinfarction trended lower (2.5% vs 3.0%, HR 0.81, $p = 0.06$). Guiding catheter thrombosis in the primary PCI cohort occurred significantly more frequently with fondaparinux (n = 22 vs. n = 0, $p < 0.001$), as did coronary complications (n = 270 vs. n = 225, $p = 0.04$). There was no difference in severe bleeding at 9 days by treatment group (1.0% with fondaparinux vs 1.3% with control, p = NS). Intracranial hemorrhage occurred in 0.2% in each group. Further investigations are needed to elucidate the effectiveness of fondaparinux in the subsets of AMI patients after recent surgery, stroke or having bleeding [7].

Even after successful PCI or only PTCA with UFH or DTI, with or without clopidogrel, the ideal scenario is still how PCI could be carried out without anticoagulation or without the concern of short or long term complications from UFH or DTI. Are there any reliable data addressing this question?

PCI WITHOUT ANTICOAGULANT

In some patients with AMI or with bleeding tendency (after recent surgery), even a short term anticoagulant could cause bleeding with long term sequelae. These patients could benefit from a new stent coated with GP 2b3a inhibitor. This strategy was verified in the Reopro-coated stent trial. In this prospective randomized trial comparing the outcomes of 96 patients with AMI treated with abciximab (ReoPro)-coated stents (n = 48) and BMS (control, n = 48). PCI was performed by standard technique without UFH in group 1 and with UFH as usual in group 2. The results showed that after PCI, one patient in group II had reinfarction and target lesion reintervention during the hospital stay. No AMI was seen in group 1. A year later, two patients in group II

(4.1%) had AMI, whereas no patient in group I suffered AMI. So abciximab-coated stent implantation was safe and effective in AMI patients without the need of an anticoagulant [8].

This strategy could be applied perfectly for patients with high risk of bleeding from even short term anticoagulation such as an AMI patient with recent surgery or a concurrent or recent stroke.

Besides the typical cases of AMI patients with bleeding or bleeding diathesis, there are many other complex, complicated non-cardiac conditions on top of the bleeding problems which need to be solved before, during or after PCI. The general strategy is to have temporary control of the bleeding so PCI for AMI could proceed. Once the IRA is recanalized and the patient general condition improves then the cause of bleeding could be definitively corrected.

COMPREHENSIVE ANGIOPLASTY AND STENTING FOR AMI PATIENTS WHO NEED SUBSEQUENT SURGERY

CASE REPORT Angioplasty and Stenting of AMI Patients with Bleeding and Need for Subsequent Surgery: An elderly patient had AMI when driving a car. The patient lost control of his car and hit the oncoming one in the opposite lane. The patient suffered a fracture in the left femur and tibia with profuse bleeding. In the emergency room, the orthopedic surgeon applied a pressure dressing to stop the bleeding and splints to stabilize the fractured bones. Because of ongoing AMI, the patient could not undergo extensive surgery. The patient was brought to the cardiac catheterization laboratories and had a bilateral femoral angiogram. The results showed no laceration in the arterial system, so the patient was prepared for PCI and successfully underwent angioplasty and stenting of the IRA under the coverage of one bolus dose of UFH (5000 units) and clopidrogrel. After PCI, the patient's condition was stabilized enough to undergo definitive surgery for the fractured leg.

Rationale of the Anticoagulation Strategy: For any patient with bleeding or bleeding tendencies, if there is no severe arterial bleeding or if the venous bleeding could be controlled by pressure dressings, then the patient can undergo coronary interventions. A diagnostic coronary angiogram would not require any anticoagulation. The preferred anticoagulant for PCI is UFH because its short half life (4 hours for a bolus dose of 5000 units) and its effect can be reversed by protamine. ASA can be given without immediate worsening of bleeding. A 300–600 mg bolus of clopidogrel is given after PCI and its effect does not start for at least 6–8 hours later. Its therapeutic effect (>80% of platelets blocked) does not reach its peak until 24 hours later. During the time window after the effect of UFH wears off and before the full therapeutic effect of clopidogrel starts, any surgery could be done with minimal concern of bleeding.

The strategy of using the window of low anticoagulation level during transition between UFH and clopidogrel was also applied in case of PCI followed by aortic valve replacement or carotid artery stenting followed by coronary artery bypass graft (CABG). In case of aortic stenosis, if the elderly (>75 years of age) patients undergo aortic valve replacement (AVR) and CABG in the same session, their mortality was estimated to be very high (>20%). So the strategy was to perform PCI and straight after the patient would go for minimally invasive AVR [9].

ANEMIA AND RE-OCCLUSION AFTER PCI

As seen in previous cases, many AMI patients with bleeding benefit from PTCA with or without stenting, with or without ASA or clopidogrel, with UFH or DTI: *who* are the patients who cannot tolerate PCI?

CASE REPORT Risk of Re-Occlusion after Transfusion for Patient Just Undergoing PCI: A patient with inferior wall myocardial infarction (IWMI) was treated with tenecteplase. The patient underwent successful rescue PCI of the right coronary artery (RCA). Due to a history of chronic anemia, the patient was pre-treated with aspirin and clopidogrel. After PCI, the patient was found to have persistent significant anemia (hemoglobin 6.5 mg/dL). Therefore, two units of blood were transfused with substantial increase of hemoglobin levels (up to 10.1 mg/dL). Nine hours later, the patient developed chest pain and ST elevation in 2,3, and a VF. Emergency angiogram showed reocclusion of the RCA stent. In order to reduce the risk of stent re-thrombosis, the patient received abciximab infusion and 300 mg of clopidogrel at the time of repeat PCI [10].

Stent Thrombosis after Blood Transfusion: Does blood transfusion carry increased risk of stent thrombosis? Acute and subacute stent thrombosis is a hard-to-predict complication of PCI. Anemia, (i.e., with the decrease in hematic concentration of antiplatelet agents) and a blood transfusion, (i.e., with the administration of fully functioning platelets),may act synergistically to increase the risk of stent thrombosis. The risk for stent thrombosis is highest in the first 14 days after insertion, particularly in the first 96 hours, and decreases thereafter [11]. With the latest data in the literature, bleeding after PCI is really a reliable marker of long term higher mortality. The patient recieving transfusion also has higher mortality and morbidity. One best option is to stent the patient with a Reopro-coated stent, if it is commercially available. However, the best management at this present time is to administer a second "load" of clopidogrel at the time of blood transfusion in anemic patients with recent PCI regardless of their hemorrhagic risk [11].

TAKE HOME MESSAGE

In general, the principle is that PCI can be performed if any bleeding can be stopped before, during, and mainly after PCI. The patient

Table 16-3 Advantages and Disadvantages of PCI of Patients with Bleeding or Bleeding Tendency

Strategy	Advantages	Disadvantages
DES	Best long term results	Need UFH, ASA, Clopidogrel
BMS	No UFH after PCI	1 month Clopidogrel, ASA forever
Only PTCA	No UFH after PCI	Re-occlusion rate of 1.4% no clopidogrel, only ASA
Reopro-coated stent	No need of UFH	1 month of Clopidogrel, ISR same as BMS
UFH	Short ½ life, can be reversed	Uncertain pharmacokinetic
Bivalirudin	Short ½ life	Cannot be reversed

also needs to tolerate 4 hours of anticoagulant without excessive further bleeding during PCI. If the bleeding cannot be stopped after PCI, then PCI is contra-indicated. If the patient needs surgery after PCI, then the appropriate time window is between the end of the effect of UFH and before the full effect of clopidogrel. If there is concern about long term effect of clopidogrel, then PTCA without stenting is the best option. If there is Reopro-coated stent available then this is the best scenario for patients who need PCI without anticoagulant. The advantages and disadvantages of each option are highlighted in Table 16-3. So in real life, each option should be selected according to the specific clinical requirement for a best expected outcome.

ANGIOPLASTY AND STENTING FOR PATIENTS ON WARFARIN

During PCI for a patient taking warfarin, coronary angiography, angioplasty and stenting can be performed in the usual fashion. Care is taken to access the femoral artery below the inguinal ligament and above the inferior margin of the femoral head while avoiding posterior wall puncture. When PCI is needed, an activated clotting time (ACT) should be measured before and after the administration of UFH. Target ACT is ⩾300 sec. Patients can receive ASA and clopidogrel as usual. Hemostasis by vascular closure device is preferred because only a platelet plug is formed to stop the bleeding at the vascular access site when the sheath is removed and manual pressure is held. Cardiac catheterization and PCI without interrupting warfarin is beneficial for patients at high risk for thrombotic and embolic complications such as those with prosthetic mechanical valves in the mitral position or those with a history of a recent embolic event [12].

Does Aspirin and Thienopyridine Cause More Bleeding for Patients on Warfarin after PCI? The main complication of oral anticoagulant therapy is bleeding, and the risk is related to the intensity of anticoagulation. In a meta-analysis, the relationship between international normalized index (INR) and major bleeding events was assessed; an INR $>$ 3, compared with an INR $\leqslant$ 3, was associated with an odds ratio for bleeding events of 3.21 (95% CI, 1.24–8.28) [13]. The Atrial Fibrillation, Aspirin, Anticoagulation Study [14] reported major bleeding annual rates of 1.1% during treatment with aspirin and adjusted-dose warfarin. Increasing INR value was an independent risk factor for bleeding complications. The Aspirin and Coumadin After Acute Coronary Syndromes study [15] demonstrated that by using a low-intensity warfarin regimen (INR 2.0–2.5) and aspirin, there was an increase only in minor, but not major, bleeding complications compared to aspirin-treated patients. In contrast, the Multicenter Aspirin and Ticlopidine Trial after Intracoronary Stenting trial, comparing antiplatelet with anticoagulant treatment in high-risk patients, aimed for a target INR of 2.5–3, and reported a higher incidence of bleeding complications with anticoagulation treatment [16]. So short-term triple therapy after PCI was not associated with prohibitively high bleeding complication rates, and thus should be favorably considered in patients with a clear indication for warfarin and clopidogrel [17].

PCI FOR PATIENTS WITH THROMBOCYTOPENIA

Thrombocytopenia is any disorder in which there are not enough platelets and it is sometimes associated with abnormal bleeding. Three major causes of low platelets are: (1) low production of platelets in the bone marrow; (2) increased breakdown of platelets in the bloodstream (intravascular); (3) increased breakdown of platelets in the spleen or liver (extravascular). Disorders that involve low production in the bone marrow include aplastic anemia, cancer in the bone marrow, infections in the bone marrow (rare), and drugs (very rare). Disorders that involve the breakdown of platelets include immune thrombocytopenic purpura (ITP), drug-induced immune thrombocytopenia, drug-induced nonimmune thrombocytopenia, thrombotic thrombocytopenic purpura, and disseminated intravascular coagulation (DIC). The breakdown of platelets by the spleen is called hypersplenism [18].

Catheterization in Patients with Thrombocytopenia Due to Liver Disease: In a study by Vaitkus *et al.*, an acceptably low risk of bleeding related to diagnostic cardiac catheterization in patients with advanced liver failure associated with thrombocytopenia and coagulopathy was demonstrated. Infusion of blood products is largely unnecessary in the pursuit of adequate hemostasis. The vast majority of patients in this study did not receive antecedent platelet or plasma infusions and nevertheless did not experience bleeding. So with appropriately careful technique during vascular access, cardiac catheterization can be safely performed in patients with severe

liver disease despite significant thrombocytopenia and coagulopathy. Adjunctive blood product transfusions are not necessary in the majority of cases [19].

PCI in Patients with Thrombocytopenia: If a patient has mild symptom of thrombocytopenia, then PCI can be performed with close follow-up of the platelet level and complications.

CASE REPORT PCI in a Patient with Thrombocytopenia Due to Myelodysplastic Syndrome: An old patient presented with unstable angina. Admission laboratories results showed severe thrombocytopenia (platelet count of 47,000 platelets/mm^3). Diagnostic angiography showed a severe lesion in the mid-left anterior descending artery (LAD). To minimize the risk of bleeding, PCI was performed with bivalirudin, instead of UFH. 300 mg of clopidogrel and 365 mg of ASA were given two hours before the procedure. He was given an intravenous (IV) bolus of bivalirudin followed by a maintenance infusion for the duration of the procedure. Baseline ACT was 120 seconds. Five minutes after the bivalirudin bolus and infusion, the ACT was 365 seconds. PCI was performed successfully in the mid-LAD without bleeding complication [18].

In patients with more severe symptoms of thrombocytopenia, PCI can still be performed under coverage of UFH or DTI. The oral antiplatelet agents (ASA or clopidogrel) can be given and empirically withheld when there is complication from low platelet level. In this situation, DES stenting is not favored because of uncertainty of long term complication profile with clopidogrel.

CASE REPORT PCI in a Patient with Thrombocytopenia Due to Myelodysplastic Syndrome: A patient with history of spontaneous gingival hemorrhage and petechiae came with chest pain. The platelet level was 20,000 platelets/mm^3. The patient underwent a bone marrow biopsy which confirmed the diagnosis of myelodysplastic syndrome involving both granulocytic and megakaryocytic series, with preservation of the erythroid series. The patient was then medicated with folic acid, pyridoxin, and cyanocobalamin. The number of platelets was maintained at approximately 40,000/mm^3, and no further hemorrhagic events were observed. Coagulogram results were within normal limit: INR was 1.09 (normal range = 0.90–1.26), and the APTT (activated partial thromboplastin time) was 28 seconds (normal range = 25–45 seconds). The patient underwent coronary angiography 2 hours after transfusion of 10 units of platelets. The result showed an obstruction of 80% in the RCA ostium. Six days later, after 75 mg/day of clopidogrel and 2 hours after new transfusion of 10 units of platelets, the patient underwent successful stenting of the RCA ostium with 6000 units of UFH. The hemogram and coagulogram done on the day of the procedure showed thrombocytopenia 40,000/mm3, an APTT of 31 seconds, and an INR of 1.03. After 2 weeks of clopidogrel, the platelet count remained stable at 40,000/mm^3 [20].

Immune Thrombocytopenia Purpura: ITP is an autoimmune disorder characterized by accelerated platelet destruction. Spontaneous mucocutaneous bleeding is common and death by hemorrhage can occur. Because of the risk of bleeding, aspirin and other pharmacological inhibitors of platelet function are generally withheld.

CASE REPORT PCI in a Patient with ITP: A 77-year-old man with history of prior CABG complained of angina and dyspnea with exertion. On admission, his platelet count was 70×10^9/L. Diagnostic coronary angiography revealed a significant lesion in the left circumflex coronary artery (LCX). ITP had been diagnosed 30 years prior to his admission at a time when he bled into his knee joint following strenuous exercise. His platelet counts through the years had always been above 50×10^9/L. At the time of his CABG, his platelet count had been around 100×10^9/L and he had not bled excessively. He had had multiple petechial bleeds and small conjunctival bleeds half a year after his CABG, while being on aspirin 81 mg per day. Platelet counts at that time were around 60×10^9/L. Aspirin was therefore discontinued. Otherwise, there was no history of increased bleeding. The patient opted to undergo PCI of the LCX and thus he was given aspirin (325 mg) the night before and on the morning of PCI. His platelet count was 64×10^9/L. UFH was administered at the time of angioplasty. The lesion in the LCX was treated using a cutting balloon (Boston Scientific, Nattick MA) without complications. The patient was discharged on 81 mg of daily aspirin in addition to his regular medical regimen. Five weeks later, he presented with 5 days of progressive chest pain that was now occurring at rest. ECG was unchanged but troponin I was mildly elevated. He had remained on chronic aspirin therapy (81 mg q.d.) and had not experienced any petechiae or abnormal bleeding. His platelet count was 78×10^9/L. Coronary angiography revealed severe stenosis at the site of the previous LCX angioplasty. Two BMS were deployed. There was no excessive bleeding during or after the procedure and the patient had prompt relief of symptoms. Three weeks later, the patient developed diffuse petechiae and a spontaneous nose bleed at a time when his platelet count had decreased to 84×10^9/L. Clopidogrel was discontinued and aspirin held for 4 days. Since that time, he has had no further bleeding problems and continues on aspirin (81 mg q.d.). His platelet count is 79×10^9/L. PCI in a patient with ITP presents a unique situation in which platelet function needs to be inhibited sufficiently to perform PCI safely but not to the extent that bleeding complications result [21].

Cutting balloon was selected because there is no need for long term clopidogrel after PCI. Stent is not favored as initial therapy because of the risk of subacute stent thrombosis in a patient in whom it is unknown whether he would be tolerant of aspirin plus clopidogrel therapy [21].

PCI FOR PATIENTS WITH HEMOPHILIA B

Hemophilia B is a severe inherited coagulopathy caused by mutations in the gene that encodes factor IX. Surgical and invasive procedures in

patients suffering from this congenital disease are to be considered as being at high risk of bleeding. Regularly, the patient with hemophilia was administered recombinant factor VIII pre- and post-procedure to maintain activity levels between 60–80% in order to prevent bleeding. When a patient with hemophilia B had AMI undergoing PCI, anticoagulation was maintained with a direct thrombin inhibitor, bivalirudin, a thrombin-specific anticoagulant. There were no complications. Bivalirudin can be safely used in patients with hemophilia B undergoing PCI [22].

PCI FOR PATIENTS WITH HEMOPHILIA A

Hemophilia A is a sex-linked genetic bleeding disorder resulting in deficiency of plasma Factor VIII coagulant activity. Patients with severe hemophilia A have FVIII levels of about 1% of normal and tend to bleed frequently on minimal or unrecognized trauma, especially into joints or muscle or less frequently intracerebrally. Modern management of hemophilia A includes safe and early treatment of bleeding and prophylactic use of FVIII concentrate in prevention of bleeding. Major surgery may be performed quite safely in hemophiliac patients if hemostasis is achieved by transfusion of FVIII, with the aim of achieving a target concentration of 100% FVIII activity during the perioperative period.

Whether FVIII transfusions are pro-atherogenic remains to be investigated. There are however, case reports of patients having AMI after receiving FVIII transfusions [23]. To avoid excess bleeding during surgery in patients with hemophilia type A, administration of FVIII is necessary. However, since FVIII is an important component of clot formation and high FVIII activity has been identified as a risk factor for thrombosis, effective, reliable anticoagulation is also required during PCI.

CASE REPORT PCI in Patients with Hemophilia A: A 64-year-old white male with a medical history of severe hemophilia A (factor VIII-dependent) presented with acute non ST elevation MI. After appropriate hematology consultation and prior to cardiac catheterization, the patient was administered 4,500 units of factor VIII (FVIII) to maintain FVIII activity around 100%. For PCI, the patient was anticoagulated with 11.3cc bolus of bivalirudin (5 mg/mL) via peripheral IV followed by 1.78cc bivalirudin (5 mg/mL) infusion. Coronary angioplasty of the LAD was performed successfully [23].

PCI FOR PATIENTS WITH VON WILLEBRAND'S DISEASE

von Willebrand factor (vWF) is a large glycoprotein encoded on chromosome 12 produced by vascular endothelial cells and megakaryocytes. It is also contained in alpha granules within the platelets and plays a crucial role in the formation of a platelet plug at sites of endothelial damage. It binds to exposed collagen-containing subendothelium and forms a bridge between the subendothelium and

platelets, allowing platelet adhesion. Platelet aggregation is also partly mediated by vWF, as it binds to platelets via the Ib/IX/V and IIb-IIIa glycoprotein complexes. vWF stabilizes the circulating procoagulant factor VIII by forming a noncovalent complex with it. Factor VIII is an essential cofactor in the activation of factor X, leading ultimately to the formation of thrombin and fibrin [24].

von Willebrand Disease (vWD) affects approximately 1% of the US population and is the most common congenital bleeding disorder [25]. It is subdivided into three types. The majority of cases are Type I (75%), which is inherited in an autosomal-dominant fashion. A mutation at the vWF gene on chromosome 12 impairs the export of vWF out of storage organelles, thus resulting in a reduction in circulating levels of vWF. This slows platelet plug formation and reduces circulating levels of Factor VIII (which rapidly degrades in the absence of vWF). Typical clinical manifestations include epistaxis, menorrhagia and difficult hemostasis following surgery. Type II vWD is caused by the production of flawed vWF due to point, insertion or missenses mutations. A moderate bleeding risk results and there are several subtypes. A major gene deletion causing a complete lack of vWF results in a severe bleeding tendency (Type III) [26].

Clinical Manifestations: The clinical manifestations of vWD vary from features secondary to platelet dysfunction or Factor VIII deficiency, according to the clinical subtype [26]. Typically, there is easy bruising and mucosal bleeding such as epistaxis and menorrhagia in milder forms of vWD. The latter may only occur associated with aspirin or nonsteroidal anti-inflammatory drug use or after minor surgery or dental extractions. In the most severe cases of vWD, such as in Types 3 and 2N there may be hemarthroses and hematomas secondary to the deficiency in factor VIII. Bleeding episodes in Type 3 are characterized by GI bleeding in 20%, hemarthroses in 37%, postoperative bleeding in 41%, muscle hematomas in 52%, menorrhagia in 69%, oral cavity bleeding in 70% and severe nosebleeds in 77% [27].

Laboratory Diagnosis: Initial screening tests typically include bleeding time, platelet count and the APTT. The bleeding time is prolonged in severe forms of vWD but may be normal or minimally prolonged in the milder forms. The platelet count is decreased in Type 2B. The APTT is prolonged in patients with vWD when Factor VIII levels are decreased. Normal values of the aforementioned test do not exclude vWD, especially in milder forms of the disease. Therefore, in patients with a bleeding history, especially with ASA-associated bruising or when there is a family history of vWD, more specific tests that include vWF antigen (vWF:Ag), ristocetin cofactor activity, and Factor VIII coagulant activity tests may be required to confirm or exclude the diagnosis. Plasma vWF:Ag levels measure the immunoreactive protein and are decreased in Type 1. The binding activity between vWF and platelet glycoprotein 1b is assessed via the ristocetin activity assay. Levels are decreased in Types 2A and 2M. Factor VIII

activity levels usually parallel vWF:Ag. There are also assays to assess the multimeric pattern of vWF protein but usually these are not necessary to make the diagnosis [28].

Replacement Therapy: Humate-P, an intermediate purity concentrate, is currently available for treatment. Typically 1 IU/kg will increase plasma VIII:c levels by 2 U/dL [29]. Very high plasma VIII:c levels (>200%) have been associated with deep venous thromboses post surgery in some rare instances [29]. Monitoring bleeding time is not necessary since no laboratory test accurately predicts hemostatic response. In general, there is agreement that the goal of therapy is to maintain ristocetin cofactor levels between 50–100% with replacement therapy for a period of 3–10 days following major surgery and less following percutaneous procedures. The length of time required post catheterization has not been studied and there are no evidence-based guidelines. Platelet transfusions may also be required to achieve hemostasis in cases where bleeding is not corrected by the preceding pharmacologic maneuvers and in severe cases of Type III vWD.

Treatment of Acute Coronary Syndromes: Aspirin is standard therapy in acute coronary syndromes (ACS). This has safely been administered and described in case reports documenting various approaches to management of ACS in patients with vWD. Thrombolytic therapy has been administered in the case of vWD and an ST elevation MI, but was associated with a significant decrease in hemoglobin requiring transfusion [30]. Successful PCI was reported without the use of anticoagulation in a patient with Type 1 vWD [31]. A 70-year-old female with a history of menorrhagia and recurrent epistaxis presented with an acute inferior MI. Aspirin was administered. Cardiac catheterization was performed and right coronary artery angioplasty, without stent placement, was performed without preprocedural heparin. In this patient, Factor VIII levels were 137 IU/dL (range 50–200 IU/dL), vFW:Ag was 40 IU/dL (50–200 IU/dL), and vWF activity was 51 IU/dL (range 50–200 IU/dL). There were no bleeding complications. Others have noted the safe use of full antithrombotic and antiplatelet therapy in a patient with vWD and an anterior infarct and ventricular fibrillation arrest [32]. Arjomand *et al.* reported their experience in performing primary PCI with the deployment of a BMS and the use of full anticoagulation with heparin to maintain an ACT greater than 300 seconds [33]. Glycoprotein 2b3a therapy with tirofiban was also administered during the procedure and continued for 24 hours after the procedure. Tirofiban was chosen secondary to its shorter half life as compared with other GP 2b3a inhibitors. The patient was also treated with aspirin and clopidogrel. The patient's Factor VIII activity level was 82% (normal 50–100%) and vWF antigen was 74% (normal greater than 50%). There were no bleeding complications.

PCI in Patients with von Willebrand's Disease: For elective procedures, most patients with vWD do not present a significant

risk of bleeding and require no prophylactic pharmacologic replacement therapy, especially if they have Type 1 disease and their ristocetin cofactor levels are more than 50% of normal. If the ristocetin cofactor level is less than 50%, patients should receive weight-adjusted doses of Factor VIII-vWF prior to and after the procedure to maintain their cofactor levels more than 50% of normal. This overall approach should be modulated by any prior history of spontaneous mucosal bleeding or excessive bleeding with any prior procedure. All patients who come to the cardiac catheterization laboratory have baseline coagulation studies performed. In addition, specific questions should be a routine part of the pre-procedure evaluation to assess a history of bleeding in all patients and their first degree relatives since vWD can be present even with normal aPTT and bleeding times. Although bleeding time is not generally a part of the routine screening process, it probably should be done in anyone with a "positive" or suggestive history. For patients who present with ACS there are a number of options to help decrease the risk of bleeding. The data suggest the use of aspirin, UDH o DTI with the adjunctive use of short-acting glycoprotein IIb/IIIa inhibitors at the operator's discretion based on the perceived risk for cardiac morbidity and mortality. In terms of catheterization approach, there may be some advantage to reduce the risk of bleeding that favors the radial compared to the femoral approach. Caution in sheath management with early sheath removal may also decrease bleeding complications. Finally, Factor VIII-vWF can be administered prophylactically in patients with an increased risk of significant clinical bleeding or in the treatment of peri- or postprocedural bleeding. For patients with Type 1 vWD, long-term management of the patients post percutaneous revascularization with aspirin and clopidogrel should continue with careful monitoring and clear instructions for immediate follow-up at the first sign of any mucosal bleeding. For patients with other subtypes, individual decisions in consultation with a hematologist is necessary to tailor the type of revascularization (balloon alone, BMS, DES) for the patient [26].

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Chapter 17
Removal of Embolized Material

Kirk Garratt, Thach N. Nguyen

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complication

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GENERAL OVERVIEW

Percutaneous techniques for treatment of coronary artery disease (PCI) have evolved and diversified dramatically, but they all have at least one common feature: all involve technical manipulation of complex equipment in the confines of coronary arteries, and are operated from a significant distance. When traversing severely diseased coronary arteries and manipulating equipment, particularly devices with detachable components, the opportunity for loss or embolization of material in the coronary circulation presents itself. In this chapter we will review and discuss the management strategies for embolized material.

STRATEGIC MAPPING

When a problem with defective equipment (unexpandable stent, uncoiling wire, asymmetrically bulging balloon due to metal fatigue, twisted guide, etc.) arises inside the coronary artery or the ascending aorta, it is ideal to remove the entire system below the level of the renal arteries so the problem can be corrected without risk of cerebral embolization or to any vital organ.

In the case of a stent that slips off the delivery balloon inside the coronary artery, it cannot be simply brought to the iliac artery by withdrawing the whole system, even as a unit. Pulling the indwelling angioplasty wire will leave a loose, free stent behind. So all efforts are concentrated on keeping the wire inside the stent and across the lesion for prompt access of rescue devices. As a stent slips off the delivery balloon, there are two options: either to retrieve it or to deploy it in a safe, non-target location. Retrieval should be attempted if threatening malposition occurs, or if the stent is loose in the aorta or in another location in which deployment cannot be undertaken safely. In the retrieval strategy, the stent should be brought safely below the renal arteries so there is no chance for cerebral embolization. Once below the level of the renal arteries, the next important step is to remove the embolized stent from the femoral sheath without injury to the femoral artery or need of arterial cut-down. Everything should be done within an acceptable time frame, with a wire still across the lesion. In the mean time, the patient has to be watched closely and the clinical condition remains stable, so the scheduled PCI can be continued and finished.

Sometimes, peripheral embolization of stents can be the best option. Systemic embolization does not cause any severe clinical sequelae, except to the cerebral circulation. Short wire fragments retained in a totally occluded artery do not pose any long-term side effects [1]. There are many reports of embolized stents into the lower extremities and periphery, without evidence of untoward long-term event affects. Any foreign materials that are retained more than 1 week should not be removed percutaneously because they may be covered and incorporated by fibrous tissue. Aggressive extraction of the embolized material may injure and perforate the vessel.

Different options in the management of embolized material are listed in Table 17-1.

All of the techniques discussed in this chapter are used only as references. They range from the standard methods with the commercial snares to the improvised techniques, which become lifesaving if the procedure is successful. The selection of a particular method or equipment depends on the patient's clinical condition, familiarity with retrieval equipment by the operators, and availability of the equipment in the cardiac catheterization laboratories. The discussion focuses more on coronary stent, but the retrieval technique may be applied to any embolized device or fragments.

RETRIEVAL OF EMBOLIZED CORONARY STENT

The majority of stents currently in use are of balloon-expandable design. They differ from the self expanding stents, which are generally constructed out of multiple interlacing strands of wire, or flexible coil stents, which are usually formed from a single compliant filament. Since coronary stents represent a detachable component of a PCI system, stents are, by their nature, prone to accidental release from the overall apparatus. Significant coronary calcification, tortuosity, and suboptimal guide position can contribute to stent embolization.

Table 17-1 Options in the Management of Embolized Material

1	No treatment for peripherally embolized small stents
2	Deploy the embolized stent in inconsequential location
3	Remove a tubular stent by a snare
4	Removal of a broken wire segment by a snare
5	Removal of embolized material by a snare made with a loop of angioplasty wire emerged from a transport catheter
6	Removal of tubular stent with two twisted wires
7	Secure a stent by inflating a small balloon distal to it and remove the whole system
8	Remount the stent by a balloon through a transport catheter
9	Grasp a stent by a biopsy forceps at the ostium of a coronary artery

Stent Embolization: Typically occurs in one of the three scenarios. First, the stent may be successfully introduced into the coronary circulation, but it cannot be advanced into the target area. This is usually due to proximal tortuosity, rigid and calcified proximal segment, or insufficient pre-dilatation of the target lesion. Second, in an attempt to direct stenting without pre-dilatation, unexpected difficulty in advancing a stent may be encountered. In these cases, the stent should be gently retracted back into the guide, removed and the lesion pre-dilated. If the distal tip of the stent has engaged the lesion, it is possible that manipulation to advance the stent may strip the stent off of the balloon, such that it remains embedded in the lesion when the balloon is retracted. In this case, the coronary wire is generally still in place, indwelling through the stent lumen and the lesion.

Most frequently, stents also become dislodged from the deployment balloon when they are retracted from the coronary artery back into the guide. At that time, the tip of the guide may catch the proximal edge of the stent, and strip it off the deployment balloon. The stent will be left dangling on the coronary wire at or near the ostium of the vessel under treatment.

TECHNICAL TIPS

****How to Withdraw a Stent Without Embolizing It [2]?** When a stent is unable to be delivered to the target area because of tortuous proximal segment or because it is unable to cross a tight lesion, it has to be withdrawn into the guide. Then the tip of the guide should be lined up well coaxially with the indwelling wire and its straddling stent. If the guide cannot provide an excellent co-axial relationship with the stent, then the guide should be retracted until a favorable alignment between the guide and stent can be achieved. Sometimes, removal may require retracting the guide to the tip of the femoral sheath in order to straighten the tip of the guide.

REMOVAL OF A STENT WITH A SNARE

Standard Equipment: The GooseNeck Amplatz Microsnare catheter (Microvena Co, White Bear Lake, MN) is a Nitinol retrieval device that includes a transport end-hole catheter and loop snares. The wire, which moves freely in the catheter, extends from the proximal end of the catheter, out the distal end, and then it is folded and re-enters the distal lumen and extends back to the proximal end. Retraction of one or both ends of the wire causes it to retract into the distal tip. The 4Fr catheter tapers to a 2.3Fr tip. The snares are available in 2, 4, and 7 mm diameter. Once emerged from the catheter, the loop is at right angle with the tip, thus facilitating the grasping of target object. The 4Fr transport catheter can easily fit inside a 6Fr guide [3].

Retrieval of a Tubular Stent from the Coronary Artery: Once a stent slips off the delivery balloon, the indwelling wire is advanced as far as possible into the distal vasculature and the balloon

removed. A 4Fr transport catheter with a GooseNeck snare is assembled. The loop of the snare, emerging from the transport catheter is passed over the angioplasty wire, encircles it, and is advanced up to the coronary ostium. The snare is manipulated into the artery to loop around the unexpanded stent under fluoroscopic guidance. An effort should be made to grab the proximal part of the stent. Once the loop is in the right position, the transport catheter is advanced to tighten the loop around the stent. Then the guide, with the stent secured by the snare, is withdrawn to the iliac artery as a unit. If extraction of a stent through the usual 6Fr or 7Fr femoral sheath is difficult or impossible, then the sheath is changed to a larger (9Fr) one through which the embolized stent can be removed. An embolized broken wire segment or any embolized device can be snatched by the snare with the same technique.

TROUBLE-SHOOTING TRICKS

*****Assembling an Improvised Snare From Angioplasty Wire:** If there is no commercial snare available, a snare can be improvised by using regular angioplasty wire available in the cardiac catheterization laboratories. The snare is formed by folding a 300 cm long 0.014" wire and introducing it through a 4Fr transport catheter. Once it arrives near the tip of the catheter, one end of the wire is pulled while the other end is advanced slightly to position the sharp point of the tight fold within the catheter so that it will not injure the vessels or cardiac wall during movement of the snare. By advancing one end of the wire, while holding the other end until a desired diameter is achieved, a workable loop snare emerges from the tip of the catheter (Figure 17-1). The embolized material is trapped by the snare using the usual technique. After the loop is tightened successfully at the distal end, a hemostat is used to fix the wire in position at the proximal end and the entire system is pulled as a unit to the iliac artery.

TECHNIQUE The Art of Loop Snaring [4]: The important difference between the commercial and the improvised snare is the angle of the snare at the tip of the transport catheter. The GooseNeck loop is at a right angle with the catheter while the improvised snare loop is parallel to it. This difference is absolutely vital in positioning the loop and assessing its position in the technique of snaring.

Once a stent slips off the delivery balloon, the wire should be kept indwelling inside the stent so the free movement of the stent is limited to the longitudinal axis of the wire. That position of the wire would tremendously help the rescue effort by giving prompt access to the defective stent. The GooseNeck Microsnare is inserted into the guide with its loop encircling the angioplasty wire. It arrives at the right position as its loop is encircling the proximal end of the stent. Then the loop is tightened by advancing the transport catheter, and the whole stent–snare–wire complex is ready to be pulled out. The improvised snare can achieve the same result but requires more skilful manipulation because the loop is not at a right angle to the catheter. In the

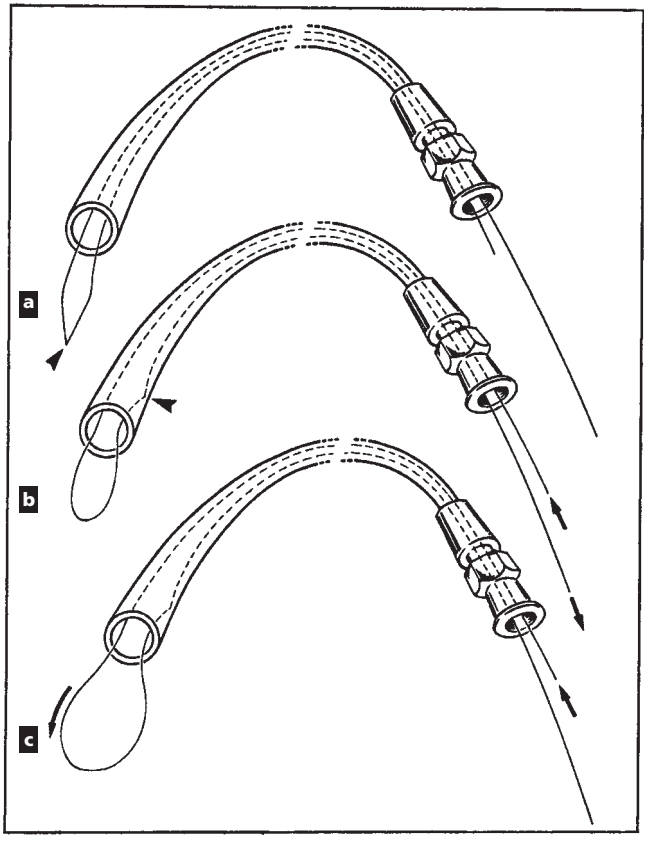

Figure 17-1 Making a snare from angioplasty wire. By advancing one end of the wire, while holding the other end until a desired diameter is achieved, a workable loop snare emerges from the tip of the catheter. (Adapted from Gerlock AJ, Mirfakhraee M [4], with permission.)

case of a broken wire segment or a free stent not on an angioplasty wire, their capture depends on correct alignment of the loop to the free end of these free fragments.

TECHNICAL TIPS

****Which End to Loop [4]?** The loop snare technique is effective if the embolized fragment (wire or stent) has a free end for ensnarement. The patient is positioned under the fluoroscope for locating both ends of the fragment and to identify its free end, which usually pulsates. The operator needs to encircle the free end with the loop.

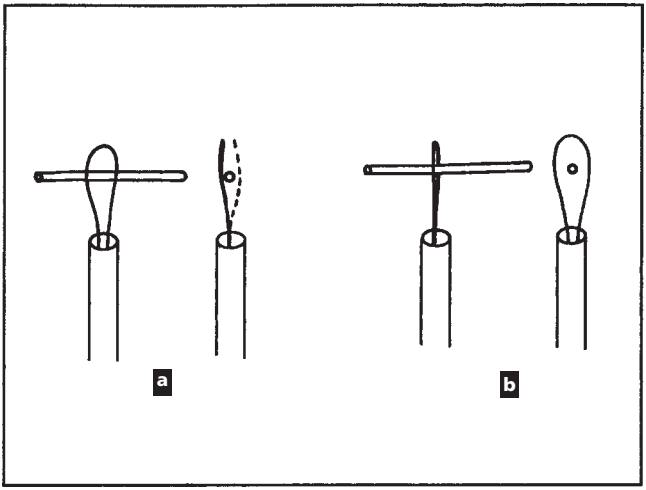

Figure 17-2 (a,b) The significance of the plane of the snare loop in relation to the broken wire or embolized stent. The snare is held in such a way that it is shown under fluoroscopy as a straight line or a closed loop, confirming its vertical plane in relation to the wire or stent fragment. (Adapted from Gerlock AJ, Mirfakhraee M [4], with permission.)

****Critical Positioning of the Snare: Perpendicular to the Object [4]:** The snare is held at right angles to the calculated plane of the embolized fragment. To do this, the patient must be positioned under the fluoroscope in such as way that the wire is seen in its full length. This implies that the wire or stent is vertical to the x-ray beam. Then the snare is held in such a way that it is shown under fluoroscopy as a straight line or a closed loop, confirming its vertical plane in relation to the wire or stent fragment. Then the free end of the wire can be captured. If the snare loop plane is parallel to the plane of the broken wire or stent, ensnarement is impossible (Figure 17-2a,b).

****Securing the Embolized Wire Fragment [4]:** The next important step is to make sure that the snare has encircled the embolized wire or stent. The transport catheter is advanced, causing the broken wire fragment or stent to bend when the snare is engaged. Withdrawing the ends of the wire to capture the embolized wire or stent is not suggested because it can cause disengagement (the stent or the wire fragment can get out of the encircling loop) (Figure 17-3). Remember to tighten the noose by advancing the transport catheter. Keep the wire immobile.

****How to Manipulate SAFELY a Pointed Loop:** If the stiff folded end of the loop cannot be withdrawn in the catheter to make a round loop outside the tip of the catheter, then the pointed loop is kept inside the transport catheter during transit. When the tip of the

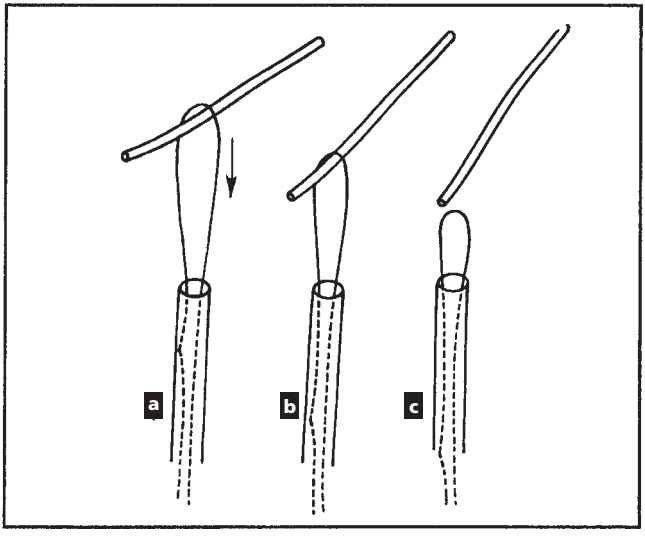

Figure 17-3 Improper technique of ensnarement. Withdrawing the ends of the wire to capture the embolized wire or stent can cause disengagement. (Adapted from Gerlock AJ, Mirfakhraee M [4], with permission.)

catheter arrives near the embolized object, it is positioned with its tip cephalad to the object, and the wire loop, still well inside the catheter, is at the upper level of the object. While the wire loop remains in place, the catheter is withdrawn to expose the loop. This technique is helpful in preventing vascular injury from the stiff, folded end of the pointed loop [4]. (Figure 17-4a–c).

REMOVAL OF A STENT WITH A BALLOON

The technique is to advance a small 1.5 or 2.0 mm balloon, over the wire and through the stent, and inflate the balloon distal to the stent. Retracting it back then will bring the stent back into the guide. If the balloon cannot be advanced all the way through the stent, low-pressure inflation of the balloon when it is at least partially within the stent will suffice. In many cases, the system may be removed without loss of the coronary wire position or removal of the guide. This will be easiest of a 7Fr or 8Fr guide has been used. In some cases, the stent may be contained within the distal tip of the guide, but the inflated balloon cannot be retracted into the guide. In this case, the guide and balloon should be removed as one unit over the wire. An extension wire will allow preservation of coronary access. The removal of an inflated balloon from a coronary artery is not without danger. The balloon should be of very low profile and the artery should be large enough to easily accommodate the movement of an inflated balloon.

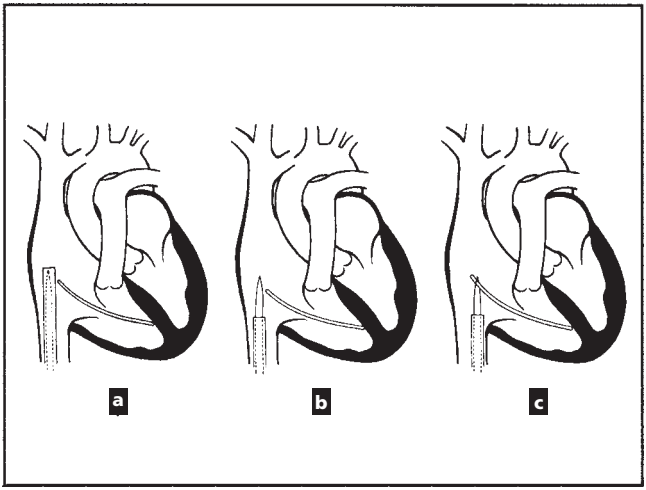

Figure 17-4 (a–c) Technique of ensnarement with a pointed loop. When the tip of the catheter arrives near the embolized object, it is positioned with its tip cephalad to the object and the wire loop, still well inside the catheter, is at the upper level of the object. While the wire loop remains in place, the catheter is withdrawn to expose the loop. (Adapted from Gerlock AJ, Mirfakhraee M [4], with permission.)

REMOVAL OF A STENT WITH TWO WIRES

When a snare is not available to remove the embolized stent, there is a possibility of withdrawing the free stent with a second wire twisting around the stent to immobilize it to the first wire [5,6].

TECHNICAL TIPS

***Manipulation of Wires to Remove an Embolized Stent:** Once a stent slips off the delivery balloon, the wire should be kept indwelling inside the stent so the free movement of the stent is limited to the longitudinal axis of the wire. In order to remove this free-standing stent with wires, a second wire should be advanced and pass through the struts of that unexpanded stent and not through the central lumen. If the stent is half-expanded, then the size of the cell is bigger, to accommodate the tip of a second wire. Once the second wire is advanced as far as possible, then the two wires are twisted proximally with the stent straddling on their stiff segment. The stent is then trapped between the two entangled wires and removed.

In order to be successful in entrapping the stent, both wires should be advanced deeply so the stent is straddling their stiff part. A soft floppy distal tip is not strong enough to entrap a stent when twisted. As the wires are removed slowly, the guide engages deeper into the ostium. This is the sign that the stent has been properly snared. In theory, if the second wire goes through the central lumen of the stent,

both wires can be easily pulled out, leaving the free stent behind. So the second wire should strategically go through the side-struts and not the central lumen. With gentle and persistent pulling, the whole system (guide, stent entwisted between two wires) will be successfully withdrawn [6].

DEPLOYMENT OF AN EMBOLIZED STENT

Proper management of this situation is generally straightforward. The deployment balloon should be advanced back over the wire and fully into the stent. Even if the stent is not advanced completely through the lesion, it should be expanded where it is to its fullest possible dimension using the deployment balloon. If the deployment balloon is unable to be advanced through the stent, a lower profile, flexible tipped balloon catheter should be inserted instead. Use of a very small diameter (1.5–2.0 mm) balloon will facilitate subsequent larger balloon entry, if a nominally-sized balloon will not pass through. It is virtually always possible to advance a balloon at least part way through the stent, and open it partly. The remainder of the stent can be expanded sequentially. Occasionally, a new smaller balloon will be needed to pass through the unopened portion of the lost stent. Pre-dilatation of the target lesion (usually possible with the balloon used to expand the initial stent) will assure success with additional stent implantation efforts.

CRITICAL THINKING

To Deploy or to Remove an Embolized Stent?

It is important to make a decision whether to deploy or to remove an embolized stent right at the beginning, because once a stent is partially deployed, the stent will have to be perfectly deployed with its struts well apposed to the arterial wall (as in any standard stenting procedure). A half deployed stent that obstructs the flow will cause early or late acute vessel occlusion. So either the stent is perfectly deployed or to be removed. It is easier to remove an intact (not-yet-deployed) stent rather to remove one later with its struts sticking out or after being crushed or disfigured. It is also easier to deploy a stent at the time when the patient is still stable rather to recross later an acutely occluded artery due to thrombus obstructing a partially deployed stent in the setting of acute myocardial infarction. If the operator attempts to open the proximal half of a stent, try to open as wide as possible because another balloon will have to be re-inserted at the imperfect opening that has just been created. If the opening of the stent is small or crooked, then the second attempt to re-insert a larger balloon would be difficult. Once the stent is deployed, it will be recrossed by other interventional devices (including a

new stent) to dilate and to stent the distal index lesion. If the first (embolized) stent is not well deployed and the lumen is not large enough, PCI of the distal index lesion would be very hard and almost impossible. Contemplating all these challenges beforehand will help the operator to make a wise decision, whether to remove an embolized stent with a snare or to perfectly deploy it.

REMOVAL OF FRACTURED WIRES

Virtually every coronary angioplasty device is advanced into the coronary system over a wire. The soft, atraumatic tips of coronary wires have been known to fracture off if being manipulated excessively, and embolize in the coronary circulation. This most frequently occurs when the shapeable wire tip becomes lodged in an atherosclerotic plaque and separates from the body of the wire when the wire is retracted. This occurred somewhat more frequently in the past, when nearly all wires were manufactured by bonding a flat forming ribbon to the round end of a wire. Current coronary wires are constructed of a gradually tapering filament that is an extension of the shaft of the wire. So, solder points and other relatively weak junctions are minimized in contemporary wire design. Nonetheless, fracture of wire tips may still occur.

TECHNICAL TIPS

****Removal of Wire Fragment:** Recovery of wire fragments is generally accomplished relatively simply through insertion of two or more additional angioplasty wires into the coronary artery under treatment. By twisting these wires together, the retained fragment could become entrapped in these angioplasty wires, and the entire system can be removed as one block. When this technique fails, a retrieval device such as a snare is needed for removal of these wire fragments.

REMOVAL OF EMBOLIZED MATERIAL FROM THE ILIAC ARTERY

Once the embolized object is brought to the iliac artery, the main problem is to remove it through the vascular sheath without need of arterial cut-down. If the 6Fr or 7Fr sheath is too small the sheath should be changed for a 9Fr sheath. Biliary forceps, alligator forceps, or cardiac bioptome are suitable for retrieving the stent in the common iliac artery or at the tip of the arterial sheath. Coil stents have been successfully retrieved by using the alligator forceps [7], and tubular stents have been successfully retrieved by using the bioptome [8]. The disadvantages of these instruments are: (1) the need to directly grasp the relatively small stent; (2) the likelihood of damaging the stent itself; (3) the possibility of endovascular trauma; and (4) the loss of wire position during stent retrieval. Hence, innovative techniques are developed for stent retrieval using easily available instruments. Most of the stents available today are radiopaque and not difficult to

locate under fluoroscopy. These removal devices are mainly used when the embolized material is brought to below the renal artery level. Familiarity with each removal device can be extremely useful in the rare event of stent misplacement.

BASKET RETRIEVAL DEVICE

The basket retrieval device is designed for capturing biliary stones and other irregularly shaped elements from within tubular biological structures. This device consists of helically arranged loops which can be collapsed or expanded by retracting or advancing a lever on the proximal end of the system. When a stent is dangling from a coronary wire, advancement of a basket retrieval device over the wire will bring it into close proximity to the stent. Retraction of the basket traps the stent and the system can be retrieved safely.

TECHNICAL TIPS

*****Best Use for Basket Retrieval Device:** It can be used to catch a stent from a side and pull it free from a deployment balloon. It works best if the stent has been damaged and misshaped such that a portion of the stents project laterally away from the deployment balloon [2].

BILIARY STONE FORCEPS DEVICE

The biliary stone forceps device is a very effective but potentially hazardous tool. Consisting of a set of curved finger-like projections that extend from the distal tip of a plastic catheter, the system can be used to hook irregularly shaped objects in biological tube structures (Figure 17-5). These systems were originally designed to assist in the removal

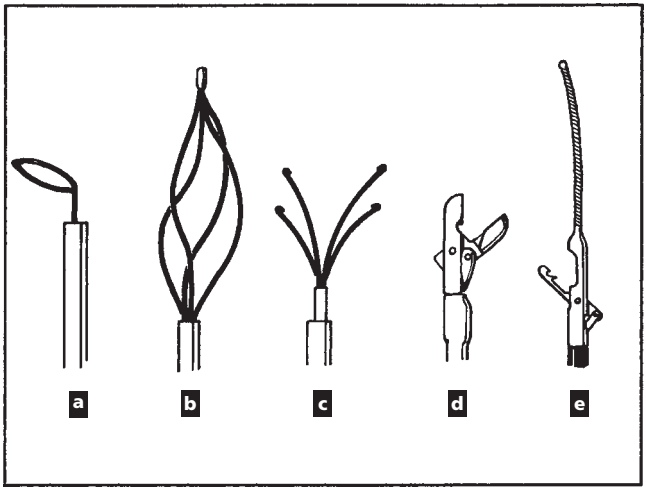

Figure 17-5 Different devices for the removal of foreign bodies.

of stones in the biliary tree. It is difficult to advance this catheter in perfect alignment with a coronary wire. Occasionally it is useful to remove the finger-like components of the system inside the transport catheter and advance the catheter over a separate wire to bring it into proximity with the coronary stent. Then readvance the finger-like component to grasp the embolized material. However, because of its sharp finger-like projections, it is generally not advised to use this within the coronary system or within vein grafts. These catheters are available in lengths of 130–150 cm, with catheter bodies of 4Fr and 5Fr diameter. The retracted device has reasonably good fluoroscopic visibility, but the finger-like projections are quite thin and have poor radiopacity. It can best used to catch a partially deployed stent or in situation a portion of a stent has become separated from the balloon [2].

BIOPSY OR ALLIGATOR FORCEPS

Alligator forceps are familiar to most cardiologists. The design of standard myocardial bioptomes follows the design principles of alligator forceps. This type of forceps device is used widely throughout medicine and surgery. The "biting jaws" action of these devices makes them attractive for capturing embolized material. A variety of these devices are available in most hospital settings, but most are not suitable for use within the vascular tree because the catheter bodies have insufficient length, the shaft diameter is too large, or the devices are too rigid to be advanced safely into the coronary arterial system. Thinner, softer disposable bioptomes are generally immediately available in catheterization laboratories and can be used, but they are still generally too rigid for use beyond the ostium of a vessel. Bioptome jaws are quite sharp, so gripping any device must be attempted with great care to avoid severing thin metallic structures [2].

COOK RETAINED FRAGMENT RETRIEVAL TOOL

Cook Retained Fragment Retrieval Tool (Cook Inc, Bloomington, IN) produces a device that resembles a fixed-wire angioplasty balloon catheter with an articulating arm. The arm is operable from the proximal hub. Activating this arm opens the device in a "trap door" fashion. Advancing this system along side a retained fragment can be very useful for recovery of the lost material, but this device is too bulky and too rigid for safe use within the coronary tree. It is available in lengths of 80 and 145 cm [2].

TECHNICAL TIPS

****Retraction of a Stent into a Guide:** Once the stent is brought to the iliac artery, it is manipulated to be withdrawn into a guide, if there is a favorable alignment between the stent and the guide. In these situations, the guide may be retracted into the arterial

sheath to straighten its tip. If there is no excellent coaxial relationship, the stent can be stripped off the balloon.

***Stent Removal from the Iliac Artery with a Commercial Snare:** Position the snare above the stent and tighten it at the distal end of the stent under fluoroscopic control. The stent can now be pulled into the guide and retrieved. The stent should be snared at the distal end, which is close to the operator. By pulling this end, the operator can manipulate this end to enter the tip of the femoral sheath and be removed from the body. If the stent is snared at the proximal end, it is more difficult to manipulate the stent to enter the guide. If the stent is crushed from the proximal end, the whole stent will collapse and its large mass is difficult to pass through the femoral sheath. If the stent is crushed in the distal end, there is only a small area of damage, and it can still be manipulated to get into the sheath. Changing to a larger size (9Fr) sheath will help to get the stent to enter the sheath.

***How to Upsize a Larger Sheath over two Angioplasty Wires and Catheters:** A stent slipped off the balloon. Advancing the snare and capturing the stent was a straightforward procedure, but the combined stent and snare proved too bulky for retrieval through the 6Fr sheath. Our greatest challenge was ultimately to exchange the 6Fr sheath for a larger 8Fr model over the snare and coronary wire. So the 4Fr snare catheter was removed and the snare noose was tried to be kept in place. The coronary wire had to remain advanced through the stent in the event the stent came loose from the snare. The inner lumen diameter of the 8Fr sheath dilator could not accommodate both the snare shaft and the coronary wire, so we had to resolve to advance a 6Fr dilator over the snare shaft within the 8Fr femoral sheath, while the smaller dilator within the larger sheath allowed the coronary wire to remain between sheath and dilator. After this successful maneuver, the snared stent was retrieved outside the patient's vasculature through the 8F sheath with no damage to the vessel wall [9].

TAKE HOME MESSAGE

Embolization of equipment into the coronary tree is dominated by loss of stents in today's interventional practice. Loss of stents typically occurs because of inadequate pre-dilatation of the target lesion, and/or improper guide alignment with the coronary ostium. Extreme tortuosity and extensive plaque calcification also contribute to the odds of coronary stent loss. The most important consideration in avoiding complications associated with stent embolization is to select appropriate tools and strategies for managing the planned intervention. Use of routine pre-dilatation of a target lesion, careful guide alignment with the ostium of the

target vessel, and appropriately supportive wires will minimize opportunities for stent loss. Specific retrieval techniques to recover lost stents are described. The most consistent device, easiest to use and readily available, are the coronary loop snares, but all of the devices described above may have an important role to play in the event of embolized coronary equipment. Familiarity with, and immediate access to, these devices is important in contemporary practice.

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Chapter 18
Carotid Intervention

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ < 100.00 $US extra; **$$** > 100.00 $US extra
⌛ < 10 minutes extra; ⌛⌛ > 10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

Cerebrovascular strokes account for 160,000 deaths per year in the United States [1]. Half of the patients that survive a stroke are permanently disabled [2]. Several epidemiological studies have shown that ischemic events are four times more frequent than hemorrhagic events. The incidence of ischemic strokes increases with age (33% if age $<$45 years and 80% if age $>$50 years) [2]. Carotid occlusive disease accounts for nearly one third of all ischemic stroke cases. In patients with high-graded and symptomatic carotid stenoses, surgical intervention has been the method of choice.

After the introduction of carotid endarterectomy as a therapy to prevent ischemic strokes due to carotid artery stenosis [3,4] this treatment modality was widely used all over the world with more than 1 million procedures until 1985, although initial trials comparing surgery and medical treatment where negative [5,6]. Only in the early 90's several randomized trials showed superiority of surgery over medical treatment. After obtaining encouraging results with angioplasty and stenting in the coronary [7], renal, and peripheral vascular systems, applications of these new technologies were made to the cerebrovascular system. The first experience with balloon angioplasty of carotid bifurcation lesions was reported in 1979 [8,9]. Elective stenting of carotid bifurcation lesions was performed for the first time in 1989 [10].

ADVANTAGES OF THE PERCUTANEOUS APPROACH

Carotid angioplasty and stenting has been of particular interest since it offers advantages over endarterectomy that makes it attractive for the treatment of carotid disease (Table 18-1).

INDICATIONS AND CONTRA-INDICATIONS

The list below includes the patients who are at high risk of surgical endarterectomy and so will benefit the most from percutaneous carotid stenting. The contraindications are listed in Table 18-2. All these contraindications are of course relative and are less important if the patient needs treatment and has a high surgical risk, and also if the operator has more experience. The Food and Drug Administration approved treatment for patients with symptomatic (>50% stenosis) and asymptomatic (>80% stenosis) extracranial carotid disease who are at high risk for carotid endarterectomy. In the CAVATAS trial [13] it could clearly be shown that the results of stenting are strongly dependent from a steep learning curve. This can be reduced if physicians perform a formal training program as was shown in the CASES-PMS trial. The clinical predictors of complications (death or cerebrovascular accident (CVA)) are identified in Table 18-3. The anatomical factors with high success and low complication rates will help the inexperienced operators to select low-risk patients for the best success at the beginning of his or her learning curve.

1 Patients With Significant Medical Co-morbidities: Since this subgroup of patients was excluded from the endarterectomy trials, the

Table 18-1 Advantages of the Percutaneous Approach in Carotid Interventions

1	It is performed with the patient fully alert, allowing close monitoring for neurological complications during the procedure
2	Since the majority of patients have coexistent coronary artery disease and other comorbidity, avoiding general anesthesia translates into a safer procedure for a population at risk for myocardial infarction during general anesthesia (between 4% and 18%) [11]
3	It is less invasive and traumatic than endarterectomy, thus avoiding local wound problems, medical complications, cranial nerve palsies, and scars
4	Patients can expect to leave the hospital within 24 hours and return to work after 72 hours [12]

Table 18-2 Contraindications for Carotid Interventions

1	Severely tortuous, calcified, and atheromatous aortic arch vessels that make access difficult
2	Pedunculated thrombus at the lesion site
3	Severe renal impairment precluding safe use of contrast agents
4	Patients unable to tolerate appropriate dose of antiplatelet agents

Table 18-3 Patients at higher risk [26–28]

Clinical
Advanced age
Prior stroke (large neurological deficit)
Cerebral atrophy/dementia
Unstable neurological symptoms
Diffuse severe peripheral vascular disease (involving aortic arch vessel)
Anatomical
Severe tortuous, calcified and atherosclerotic arch vessels
Coexistent proximal common carotid lesions
Angiographic evidence of large amount of thrombi
Long, complex lesions extending into the distal internal carotid
Severe tortuosity just distal to the bifurcation

indications and results of surgery are not well established. Myocardial infarction was the leading long-term cause of death after endarterectomy in patients with concomitant clinically important coronary artery disease [14]. Patients with significant carotid disease undergoing coronary artery bypass grafting (CABG) have a risk for stroke from hypotension during general anesthesia [15]. Published reports on combined endarterectomy and CABG suggest that the risk of stroke or death ranges from 7.4% to 9.4%, 1.5–2.0 times the risk of each operation alone [16].

2 Carotid Restenosis: This is technically challenging because of scar tissue surrounding the carotid bifurcation. The complication rate of redo-endarterectomy approximates at 10% [17–19]. Early results indicate that carotid angioplasty and stenting can be safely achieved and represent a valid alternative to carotid reexploration in this high-risk group [20–23].

3 High-grade Carotid Stenosis With Contralateral Occlusion: The perioperative risk of stroke or death in the presence of a contralateral carotid occlusion was 14.3% in NASCET [24]. There is no evidence that carotid shunting reduces the perioperative risk of stroke. Carotid angioplasty and stenting obviate the need for carotid occlusion in the presence of reduced cerebrovascular reserve.

4 Radiation-induced Carotid Stenosis: This is a surgical challenge because of both involvement of the distal common carotid artery, and extensive scarring and fibrosis [25]. Infections and wound problems are increased by previous radiation.

5 High Cervical or Intracranial Stenosis: High lesions in patients with short or thick necks or intracranial stenosis are difficult or impossible to expose surgically.

STANDARD PRE- AND POSTOPERATIVE PROCEDURES

These are listed in Table 18-4. Especially for a center starting a carotid program, it is recommended that all these evaluations should be

Table 18-4 Pre- and Postprocedural Checklist for Carotid Procedures

1	Adequate medical and neurological evaluation
2	CT scan or MRI of the brain to document preprocedural anatomic deficits
3	A formal neurological assessment and completion of a National Institutes of Health (NIH) stroke scale pre- and post-procedure
4	Some operators still recommend a complete cerebral angiography in a separate examination or immediately before carotid angioplasty and stenting. However, in the days of MRI angiography this seems to be less important. In our center a four-vessel angiography is rarely performed
5	Duplex ultrasound pre- and post-stenting to exclude fresh thrombus and as a baseline for follow-up
6	Aspirin 300 or 325 mg and clopidogrel (Plavix) 75 mg once a day. Different from coronary interventions the target is not so much to avoid thrombus formation after stent implantation but to avoid fresh thrombus prior to stent implantation. This fresh thrombus may embolize during the procedure. Therefore, the treatment with aspirin and clopidogrel should start at least one week before

performed before doing the procedure. Of course it is essential that the diagnostic tests listed in Table 18-4 are repeated after the procedure.

CAROTID STENTING STEP BY STEP

Carotid intervention is a dynamic procedure that changes as progress is made [29,30]. The use and manipulation of guides, wires, sheaths, balloons, or stents evolves according to the experience of the operators. A generic step-by-step procedural approach for carotid intervention is suggested below.

Step 1 Vascular Access: The femoral access approach is the most commonly used. Femoral puncture is done with insertion of a guide (e.g. Fr 4). In complex anatomy or when it is planned to choose a proximal cerebral protection system, a standard 5Fr- 9Fr 12-cm arterial sheath is used. In case of diseased iliac arteries a 23 cm sheath and in case of an abdominal aortic aneurysm a 40 cm sheath may be preferred. The patient is given heparin to obtain an activated clotting time of 200–250 sec.

Step 2 Access With a Guide: An 8Fr guide (usually a right coronary guide) is advanced into the ascending aorta over a hydrophilic 0.0035" wire. For a difficult or anomalous anatomy, an aortogram of the arch vessels can be done and used as a guide to selective cannulation. After angiography of the aortic arch and recognition of the anatomy, the guide is advanced into the common carotid artery (CCA). Prior to cannulation of the common carotid, a careful aspiration and flush of saline should be performed to clear any debris or thrombus.

Access With a Sheath: Cannulate the common carotid artery with a 5Fr diagnostic catheter, usually with a right coronary or a headhunter catheter. Access the external carotid artery (ECA) with an angled

hydrophilic guide wire and advance the diagnostic catheter into the external carotid artery. Some investigators recommend road mapping to display the origin of the external carotid artery. We prefer to use bony landmarks. Replace the current wire with an exchange length 0.035" wire. Generally, a stiff Amplatz-type wire should be used. The diagnostic guide is then exchanged over the wire for a 6Fr 90 cm sheath that is then advanced into the common carotid artery below the bifurcation. Gently manipulate the sheath during engagement because it can cause a tear at the ostium of the common carotid artery or dislodge atherosclerotic debris. Aspirate and flush meticulously so there is no air inside.

Step 3 Angiography of Intracranial Vessels: Perform angiography of intracranial vessels in two projections, lateral and ap-30° cranial. This angiogram will be very important for comparison and further intracranial rescue procedures in case embolisation occurs during the intervention.

Step 4 Landmark Identification: Remove the 0.035" wire for pressure measurement. "Back bleed" and flush carefully and take a "guiding" image lesion by injecting through sheath. An arteriography is performed to maximize the opening of the bifurcation and pinpoint the severity of the stenosis. Then, during the intervention, the most useful projection is not only the one showing the maximum stenosis, but also those separating the internal and external carotid arteries and those showing the bony landmarks.

Step 5 Cerebral Protection: Distal embolisation is the major cause of complications during carotid stenting. Therefore, we use cerebral protection devices in all patients. They are described in more detail later in this chapter. Generally, the device has to be introduced and placed distal (filter or occlusion balloon) or proximal (occlusion balloon) of the lesion.

Step 6 Atropine: Give atropine (1 mg intravenously) to prevent bradycardia 2 to 3 min before balloon inflation.

Step 7 Predilation: This is in order to facilitate introduction of the stent delivery system. We use it only occasionally (for example if we failed with an attempt to introduce the stent without predilatation) but some other operators recommend predilatation in all cases. A 3 or 4 mm monorail or coaxial angioplasty balloon is advanced to the lesion over the 0.014" wire which is attached to the filter or distal occlusion balloon or over a separate 0.014" wire in case a proximal occlusion balloon is used for cerebral protection. Usually stiff wires are preferred. For predilatation the angioplasty balloon is inflated at low pressures 1 atm above the disappearance of the waist. In very rare occasions (very tight and calcified lesions) predilatation has to be performed even before introduction of a cerebral protection device.

Step 8 Stent Deployment: Exchange the balloon system for a stent system. The diameter of the stent should be 1–2 mm larger than the largest segment to be covered. Most often stents with a diameter between 6 (if the stent is implanted into the internal carotid artery

only) or 8 and 10 mm are used. Today only self-expanding stents are used. Although the internal carotid artery is 2–3 mm smaller than the common carotid artery, oversizing the stent in the internal carotid artery does not cause problems. Covering the external carotid artery is safe and rarely causes occlusion of the external carotid artery. The stent should be long enough to cover the lesion completely. Most often we use stents 3 cm long.

Step 9 Post-stenting Management: Post-stent deployment a balloon dilatation should be done at nominal pressure. The balloon diameter should equal the diameter of the internal carotid artery distal of the stent. Post stent dilatation of the stent segment in the common carotid artery is not necessary and not recommended. If the external carotid artery becomes significantly stenosed or occluded, this does not cause symptoms and does not need treatment. Perform angiography to identify further lesions, dissections, and embolic complications.

Step 10 Removal of the Cerebral Protection Device: Almost all currently available filter devices are removed with a retrieval catheter. In case of an occlusion balloon, the debris in the internal carotid artery has to be aspirated before occlusion balloon deflation and retrieval. Perform carotid angiography including intracranial branches to document the final result and to exclude distal embolisation.

General Measures: Continuous monitoring of the heart rate, blood pressure, and neurological status throughout and post intervention is mandatory. Good hydration and maintenance of an appropriate blood pressure are important in the recovery period. At the time of balloon inflation the blood pressure always goes down. So there is no need to lower the blood pressure before, even if the patient is severe hypertensive. After the procedure the systolic blood pressure should be below 140 mmHg. A lower pressure is preferable, especially in case of a very tight lesion before stenting and/or in case of a contralateral occlusion because these patients have a higher risk of intracranial bleeding. The sheath is removed when the activated clotting time (ACT) is $<$180 sec. The patient usually can be discharged after 6–8 hours if no complications are encountered and if blood pressure and heart rate are stable.

CEREBRAL PROTECTION DEVICES

A major limitation of carotid angioplasty is distal embolization during the procedure. Balloon dilatation, stent implantation and manipulation of the vessels through catheters and wires release embolic debris, which can cause severe damage [28,29].

Kastrup *et al.* [30] compared the frequency, number and size of new diffusion-weighted imaging (DWI) lesions after unprotected and protected angioplasty in 206 patients. They concluded that the use of cerebral protection devices significantly reduces the incidence of new DWI lesions after carotid stenting. The lesions correlate closely with the clinical outcome.

Subsequently, in almost all centers cerebral protection devices are applied routinely despite the lack of randomized trials, because many series and registries revealed a benefit of protection devices. The World Registry [31] described a perioperative stroke and death rate of 5.29% in patients without, and of 2.23% in procedures with, protection. Kastrup *et al.* [32] reviewed trials from 1990 to 2002 with a total of 2537 procedures performed without protection devices, and 896 stenting procedures performed with a cerebral protection device. The combined stroke and death rate at 30 days in patients treated with cerebral protection was 1.8% compared with 5.5% in patients treated without cerebral protection devices.

FILTER DEVICES

Angioguard XP™/Angioguard RX™ (Cordis): The Angioguard filter (Figure 18-1) was the first filter which became available. It consists of the parachute type filter itself, which is mounted on a 300 or 180 cm long 0.014" wire, a delivery catheter and a retrieval catheter. The monorail version using the short wire is preferred by most interventionalists. The filter comes in diameters between 4 and 8 mm and is compatible to vessels between 3.5 and 7.5 mm. The filter membrane is made of polyurethane. The pores in the filter have a diameter of 100 μm. The filter has 8 nitinol struts. Four of these struts have a radiopaque marker. The deployment sheath is a rapid exchange system and has a crossing profile of 3.2 to 3.9Fr.

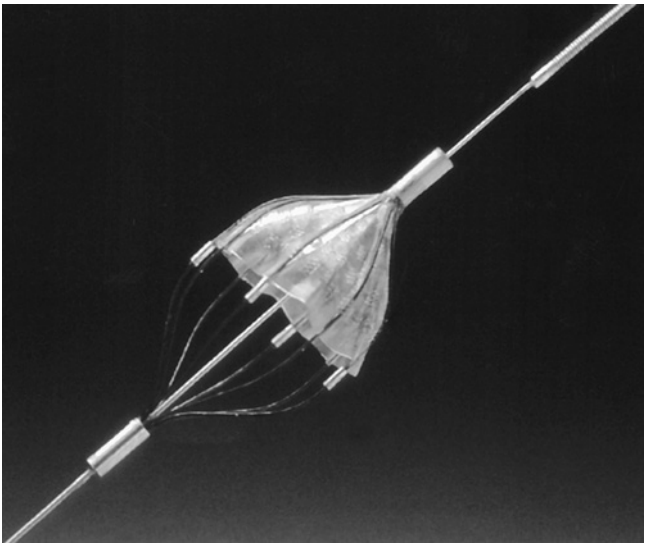

Figure 18-1 Angioguard embolic protection device (Cordis Corporation, Miami, FL, USA).

FilterWire EX/EZ™ (Boston Scientific): This filter (Figure 18-2) is mounted on a 0.014″ wire by means of an eccentric nitinol wire loop. Due to this design, the entry of the particles into the filter is not impeded by filter struts. The new version of this device (FilterWire EZ) has a better vessel wall apposition than the former device. The membrane of the filter is made of polyurethane and has pores with a diameter of 110 μm. The delivery catheter has an outer diameter of 3.2Fr. The filter comes in one size and adapts to vessel diameters between 3.5 and 5.5 Ch18.wpsmm in diameter. It can be withdrawn with a retrieval catheter of 4.3Fr or with any 0.018″ compatible balloon catheter.

Emboshield RX Vascular Protection System (Abbott Mednova): This system consists of a guide wire, a delivery catheter (3.7–3.9Fr), a filter basket (3–6 mm) and a retrieval sheath (5.5Fr). Initially the lesion is crossed with the guide wire alone. Thereafter, the filter is loaded into the delivery catheter, which is advanced over the wire distal to the lesion. After angioplasty and stenting the retrieval catheter is advanced over the wire and the filter. The retrieval catheter has an expansile distal section which expands upon filter retrieval and allows full recapture of the filter.

SpideRX (ev3): The newest version of this filter is the SpideRX Vascular Filtration System (Figure 18-3). It consists of a windsock-type

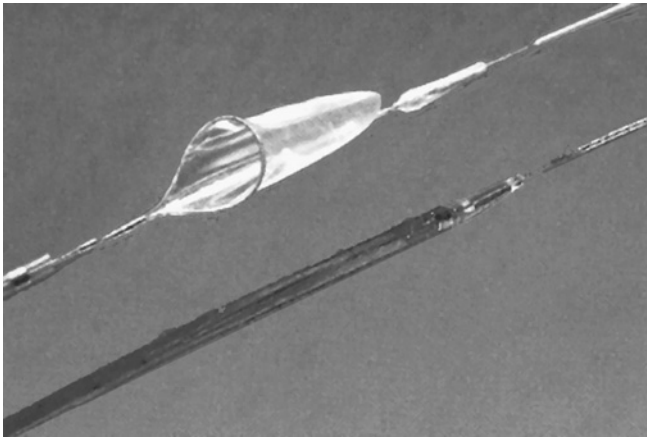

Figure 18-2 FilterWire (Boston Scientific).

Figure 18-3 Microvena Spider vascular filtration system.

filter basket made of a nitinol wire mesh. The design of this filter has some similarities with the E.P.I. filter. However, it comes in different sizes between 3 and 7 mm. At the entrance of this filter there is a clasp to ensure a better vessel wall apposition of the opening of the filter. After crossing the lesion with the wire the delivery catheter of the system is introduced. The crossing profile of the delivery catheter is 3.2Fr. The wire is removed and the filter is advanced through the delivery catheter and placed distal of the stenosis.

Rubicon-Filter (Boston Scientific): This system is a filter mounted on a 0.014" wire. Due to its design and small crossing profile it allows one-step insertion and deployment of both filter and stent. The filter basket is supported by floating Nitinol™ struts for superior apposition in vessels. The pore size is 100 μm.

Interceptor Plus (Medtronic): The Interceptor filter consists of a Nitinol basket attached to a 0.014" steerable guide wire. The basket diameter ranges between 5.5 and 6.5 mm. The size of the pores is 100 μm. The filter is deployed with a delivery catheter with a very low profile of 2.7Fr.

RX AccuNet™ Embolic Protection System (Guidant): This is a polyurethane filter with a diameter ranging from 4.5–7.5 mm. The system is 6F sheath/8F guide catheter compatible. The 0.014" wire can be torqued independent from the filter basket and utilizes rapid exchange technology. The basket is secured with all cage strut openings enclosed during withdrawal.

OCCLUSION DEVICES

Guardwire® Temporary Occlusion and Aspiration System (Medtronic Vascular): This was the first occlusion device which was available commercially. An elastomeric balloon is mounted at the tip of a 0.014" or a 0.018" wire. The balloon can be filled through a lumen inside of the wire up to a diameter between 3 and 6 mm. This is done via a so called MicroSeal™ adapter. With this adapter a MicroSeal in the wire can be opened and closed. When the MicroSeal is closed, the adapter can be removed from the wire without deflation of the balloon. After crossing the lesion with the GuardWire, the distal balloon is inflated distal of the stenosis to occlude the internal carotid artery. The MicroSeal adapter is removed and the angioplasty balloon and the stent are introduced over the wire. After stent implantation an aspiration catheter is advanced over the wire into the internal carotid artery. The debris, which may have been dislodged from the lesion is aspirated and removed. Thereafter, the distal occlusion balloon is deflated.

The advantage of this technique compared to filter techniques is that even very small particles can be captured. Furthermore, the crossing profile of the device is very low. However, some patients do not tolerate the balloon occlusion of the internal carotid artery [33]. It is possible to do the procedure stepwise with intermittent deflation of

the balloon. Obviously this makes the procedure a little bit cumbersome. Another disadvantage is that angiography during the procedure is not possible.

TriActiv® ProGuard™ Embolic Protection System: The TriActiv®ProGuard™ System is an embolic protection system designed to handle branched vessel anatomy with a novel localized flush and extraction technology (LFX™Catheter). The 2.5–5.0 mm balloon is integrated in a 0.014" guidewire and is filled with CO_2.

Gore Neuro Protection System (Formerly ArteriA Medical Science): This device prevents distal embolisation by establishing a retrograde flow in the internal carotid artery (Figure 18-4). It consists of a 9Fr guide with a balloon at its distal tip. This balloon is inflated in

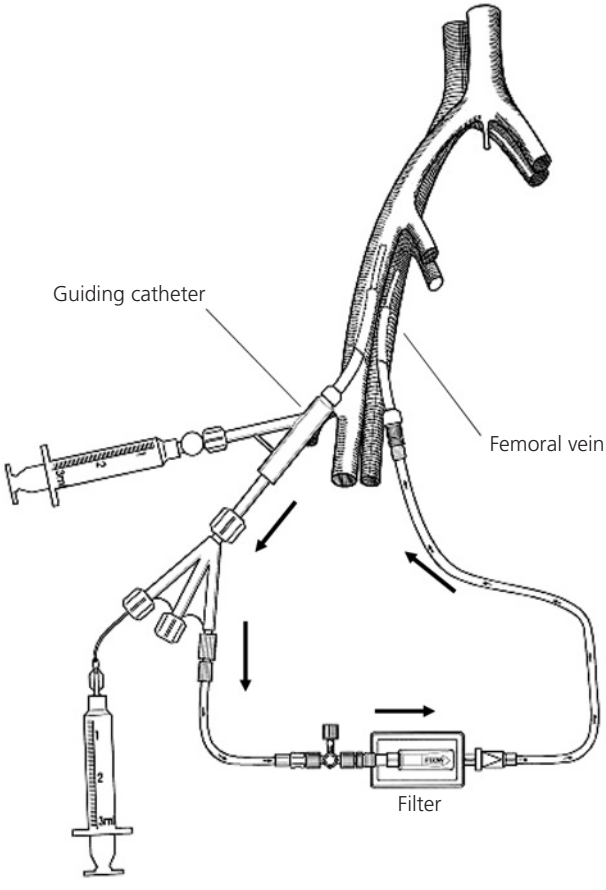

Figure 18-4 Gore Neuro Protection System.

the common carotid artery. To avoid blood flow from the external to the internal carotid artery the external carotid artery is occluded with a separate balloon mounted on a wire which is introduced through the lumen of the guide. The proximal hub of the guide is connected with a venous sheath. Due to the pressure difference between the distal internal carotid artery and the venous system a retrograde blood flow is established. A filter located in the arteriovenous shunt prevents embolisation of the debris into the venous system.

The major advantage of this technique is that during the procedure emboli can not move towards the brain. This protection starts already before crossing the lesion. This is of special importance in lesions which contain fresh thrombus (Figure 18-5).

The operator may use the wire of his choice which helps to cross difficult lesions. There is no risk of distal problems in elongated vessels (Figure 18-6).

Disadvantages of this technique are the need for a 9Fr sheath and intolerance of balloon occlusion in some patients. In contrast to the distal balloon occlusion technique (Percusurge) angiography during the

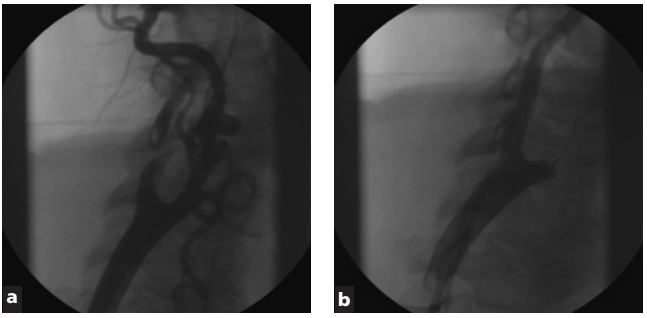

Figure 18-5 Angioplasty of a lesion with a fresh thrombus. In this situation a proximal occlusion system as embolic protection device should be used.

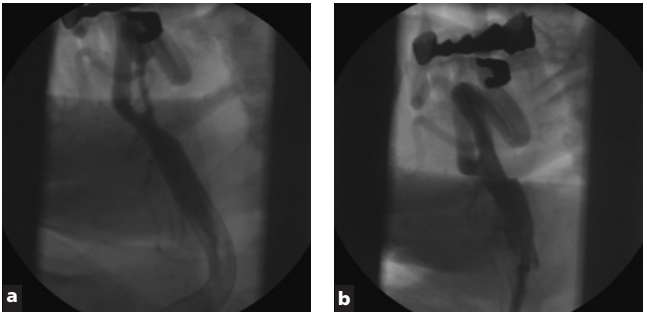

Figure 18-6 Angiogram of severely elongated vessel. As it might be difficult to place a distal protection system in the internal carotid artery we would suggest the use of a proximal occlusion system.

procedure is possible. To perform the procedure in a stepwise fashion is easier and faster than with the Percusurge technique, because there is no need for an aspiration catheter before deflating the balloon.

MO.MA (Invatec): This device has some similarities with the Gore Neuro Protection System. The occlusion balloon for the external carotid artery is fixed to the guide (9Fr) which allows faster and more reliable placement. This implies that the fixed distance between the balloon at the tip of the guide and the external occlusion balloon is suitable for the individual anatomy of the patient which is the case in the majority. The distal balloon is capable to occlude vessels up to 6 mm (external carotid artery) and the proximal up to 13 mm (common carotid artery). Instead of a continuous retrograde flow to the venous system an aspiration with a syringe is used to remove the debris between the different steps of the procedure or at the end of the procedure. As with the Gore Neuro Protection System device angiography during the procedure is possible. The operator may use any kind of wire to cross the lesion and in case of intolerance the procedure can be performed stepwise.

SELF-EXPANDABLE STENTS

Within the last few years the number of available carotid stents increased considerable as well as major improvements have been made to meet the specific requirements of carotid stenting.

During the first years of carotid stent implantation mainly balloon-expandable stents were used. This technique had to be abandoned due to stent crushing which lead to cerebral flow impairment. Since then the interventionalists can choose between self-expanding nitinol stents and stainless steel stents. Which stent to choose, depends on the arterial anatomy and the specific characteristics of the lesion.

With exception of the NexStent (Endotex), which is laser-cut from a nitinol sheet and coiled into a tube-like form, all nitinol stents are constructed from a nitinol tube that is laser-cut during the production. Depending on the number of the bridges between the different rings, the nitinol stents can be classified into stents with a closed-cell or an open-cell configuration.

In very tortuous access vessels a low crossing profile and high flexibility of the delivery catheter is important. At present the crossing profile does not differ significantly between the different stents. However, the Wallstent delivery system has currently the highest flexibility.

In tortuous lesions a flexible stent is required to avoid straightening of the vessel and kinking of the artery at the end of the stent. Stents with a high flexibility are open-cell nitinol carotid stents.

To treat lesions in carotid bifurcations with a significant difference in diameter between the common and internal carotid artery, the stent needs to adjust its diameter to the width of the vessel lumen. All stents available provide these features.

To accomplish an even smoother transition between the internal and common carotid artery tapered stents have been developed,

which are characterized by a smaller stent diameter at the distal end of the stent.

To treat severely calcified lesions a stent with a high radial force is recommended. In general, closed-cell design stents (Xact stent, Abbott) have a higher radial force.

Closed-cell stents provide better scaffolding to treat lesions with high emboligenic potential. However, the clinical impact of open cell design versus closed cell design or probably more important of cell size and pore size is still unclear.

Carotid Wallstent (Boston Scientific): The Carotid Wallstent comes in a diameter of 6 to 10 mm and a length of 22 to 37 mm when fully opened. If the stent is implanted in a vessel, the length varies depending upon the degree of compression. This also means that the stent is much longer as long as it is fixed in the delivery system. During deployment, it foreshortens according to the diameter of the vessel. For example, a 31 mm long stent with a diameter of 10 mm (fully open) is approximately 60 to 70 mm long as long as it is compressed in the delivery system. Implanted in a 9 mm vessel it has a length of 40 mm. If it is implanted in an 8 mm vessel it is 49 mm long.

It is a rapid exchange system and 0.014" wire compatible. The outer diameter of the delivery system is 5 or 5.9Fr depending upon the diameter of the stent. The stent mesh design provides high plaque coverage. A disadvantage is that the stent straightens the vessel more than open cell Nitinol stents do.

Exponent RX (Medtronic): The carotid nitinol open cell design stent by Medtronic has a monorail system and is 0.014" wire compatible. The outer diameter of the system is compatible to 6 or 7Fr sheaths. It is available in a diameter of 6 to 10 mm and has a length of 20 to 40 mm. With the Exponent stent foreshortening of less than 10% can be observed. The maximum crossing profile ranges from 1.83 to 2.08 mm.

Xact Carotid Stent (Abbott Vascular): The Xact stent is a nitinol stent that comes in two different shapes. The straight stent is available in a diameter of 7 to 10 mm and a length of 20 to 30 mm and the tapered stent in a diameter of 6–8 mm, 7–9 mm and 8–10 mm and a length of 30 and 40 mm. It has a crossing profile of 5.7Fr and a rapid exchange delivery system. The Xact stent has a closed cell design and therefore it is stiffer than other nitinol stents. It should be deployed only in straight vessel segment.

RX Acculink (Abbott Vascular): This open cell Nitinol stent is also available in two different shapes. The straight stent comes in a diameter of 5 to 10 mm and has a length of 20 to 40 mm and the tapered stent has a diameter of 6–8 mm and 7–10 mm (length of 30 and 40 mm). The rapid exchange system is compatible to a 0.014" wire and a 6Fr sheath.

Sinus-Carotid-RX/Conical RX (Optimed): This Nitinol stent is another stent which is available in two configurations. The straight

stent has a diameter of 6 to 9 mm and a length of 20 to 40 mm. The tapered stent comes in a diameter of 6–9 and 7–10 mm and has a length of 30 or 40 mm. It has an open cell design. The distal ends of the stent have higher radial force than in the middle. A special feature is that the stent is attached to the 5Fr delivery system until the very end of the release process which prevents the stent from "jumping distally" during deployment.

NexStent (Boston Scientific): This stent is available in just one size which fits into vessels with a diameter of 4 to 9 mm and has a length of 30 mm. It has a closed cell design which prevents entrapment of balloons and filters. It is much more flexible because unlike other closed cell design stents it is made of a rolled Nitinol sheet. The outer diameter of the delivery system is 5Fr. It is 0.014" wire compatible and has a tip which is formed by the delivery sheath instead of the delivery shaft. This facilitates removal of the delivery system after stent implantation.

Precise RX Nitinol Stent System (Cordis): The Precise stent has a rapid exchange system with an outer diameter of 5 to 6Fr. It is 0.014" and 0.018" wire compatible. The open cell nitinol stent has a diameter of 5 to 10 mm and a length of 20 to 40 mm. This stent has a high conturability and flexibility.

Zilver Stent (Cook): The Zilver stent has a diameter of 5 to 10 mm and a length of 20 to 60 mm depending on the size of the diameter of the stent. It is made of Nitinol and has an open cell design. Due to the larger open-cell area and fewer connectors this stent is highly flexible. The system has an outer diameter of just 5Fr and is 0.014" and 0.018" wire compatible. Because of the flexibility of the delivery catheter, this system can be used in tortuous vessels. There is no shortening of the stent during releasing, so that an exact placement can be performed. It is available only in an over the wire version.

Protégé RX (ev3): This nitinol stent is available in a straight and a tapered design. The straight stent has a diameter of 6 to 10 mm and a length of 20 to 60 mm. The tapered stent has a diameter of 8–6 mm or 10–7 mm and a length of 30 or 40 mm. The stent does not shorten during the implantation. The Exact Placement Release Technology avoids premature stent deployment.

ViVEXX® Carotid Stent and Conformexx® Carotid Stent (C. R. Bard): The rapid exchange delivery system of the ViVEXX® carotid stent has an outer diameter of 5Fr and is compatible with a guide wire of 0.014". The stent can be chosen in two design versions: straight and tapered. The straight stent is available in diameters of 5 to 12 mm and lengths of 20, 30 and 40 mm and the tapered stent in 8–12, 7–10 or 6–8 mm with a length of 30 or 40 mm. The tip of the delivery system is formed from the outer sheath which means that the delivery system can always be removed without any resistance.

The Over-The-Wire delivery system of the Conformexx® carotid stent has an outer diameter of 6Fr and is compatible with guide wires of 0.014" to 0.018". It is available in diameters of 6 to 12 mm and lengths of 20, 30 and 40 mm. The tip of the delivery system is formed from the outer sheath. The advantage of this design is that after stent delivery removal of the delivery system is easier.

Cristallo Ideale (Invatec): This is a 5Fr rapid exchange system. The straight stent has a diameter of 7, 9 or 11 mm and a length of 20 to 40 mm. The tapered stent comes in sizes of 7–10 and 6–9 mm with a length of 30 or 40 mm. It has a small cell size in the middle and a larger cell size (with more flexibility) at the proximal and distal end.

TECHNICAL TIPS

***Cannulation of the Brachiocephalic Arteries:** We usually start with a right Judkins catheter. Catheters with a similar shape are the head hunter H1 and the Bentson/Hannafee JB1 catheter. This type of catheter is advanced over the aortic arch by keeping the tip pointed inferiorly or over a guide wire. This avoids trauma to the intima of the aortic arch and prevents the catheter tip from becoming trapped by vessel ostia. In the ascending aorta the catheter is turned around 180° which places the tip in a vertical upright position. Thereafter, the catheter is gently pulled back. Usually this motion will bring the catheter tip into the brachiocephalic artery. If the left common carotid artery is the target, the catheter should be pulled further distally very slowly. During this period, the catheter should be turned 20° counter-clockwise to make the tip point slightly anteriorly. This helps to engage the left common carotid artery. To stabilize the catheter in the left common carotid artery, it is necessary to rotate the catheter 20° clockwise to make the tip of the catheter point vertically or slightly posteriorly again.

If we are not successful with one of these catheters, we usually switch to a Simmons/Sidewinder-catheter. This catheter forms a loop in the ascending aorta. By pulling back this type of catheter, the tip engages the vessels of the aortic arch (brachiocephalic trunk first). In contrast, with the Vitek catheter a loop is formed in the descending aorta. A catheter with a similar shape is the Mani catheter. By pushing the catheter towards the ascending aorta, the tip engages the left subclavian artery, the left common carotid artery and finally the brachiocephalic trunc.

***Injection of Contrast Media:** After the guide enters into the artery, slow hand injection is done to confirm the position of the guide, to make sure that good blood flow is maintained and that there is no subintimal entry of the contrast agent. Injections of contrast agent into all brachiocephalic arteries should be done with small amounts of contrast (no more then 6 cc per injection, hand injection or 3–6 cc per second). Larger volumes create a mixture of arterial, intermediate, and venous phases, thus obscuring early filling veins and other pathologies.

****The Need for Diagnostic Angiography of the Brachiocephalic Arteries:** Some operators always perform a 4-vessel angiography to check collaterals for interventions and rescue when needed. We usually do not do this in order to avoid the additional risk, especially if we have an angio MRI. Of course, the angio MRI does not provide information about the functional capacity of the intracranial collaterals. If the internal carotid artery provides collaterals to the contralateral system, balloon inflation with transient occlusion of the internal carotid artery can cause seizure. On the other hand, stenting would still be possible.

****Technique of Engagement of the Left Carotid Artery:** If catheterization of the internal carotid artery is desired, the head is again turned to the contralateral side; however, the neck is flexed to align the internal carotid artery with the common carotid artery. The tip of the catheter is now pointed posteriorly and the guidewire is inserted. The catheter is advanced over the guidewire, and its tip is placed at the level of C2. It is important not to advance the catheter farther, since this may cause spasm. In cases of a cervical carotid loop, the catheter is placed below the loop (Figure 18-7) [25].

****The Art of Advancing the Catheter:** The clue is to advance the guide slowly over the wire while maintaining the wire deep inside the carotid artery. The left wall of the upper thoracic aorta can be used to support advancement of the guide into the carotid artery. One has to recognize "bad" and "good" curves of the guide within the aortic arch while advancing the guide. The guide is pushed over the wire slowly, also taking advantage of the pulsating blood flow. This maneuver, advancement of the guide, and withdrawal of the wire is made several times until the guide is securely placed in the artery. If there is a tortuosity of the proximal segment, the artery can be straightened with a wire. The guide can also be rotated while being advanced, clockwise or counter-clockwise, depending on the curve formation in the aortic arch. Asking the patient to take a deep breath would help to elongate and thus straighten the great vessels. During that short window of opportunity, the guide is moved farther. Another important aspect is to gently "ease back" on the guide curve in the arch as successive wires are advanced. This reduces the curve in the arch and prevents the successively stiffer wires from prolapsing the guide down into the ascending aorta [26]. Excessive manipulation of the guide in the arch may predispose to distal embolization.

****Difficult Carotid Access:** With experience, 95% of bifurcations can be accessed using the current guide and wire systems. However, it may be difficult to access severely tortuous, calcified arteries using the standard approach. In those arteries in which the wire will not advance without "kicking" the guide back, alternative strategies have to be considered. If the common carotid artery is very atherosclerotic along its course, it may be prudent to refer the patient to surgery since the additional manipulation may produce embolic complications [26].

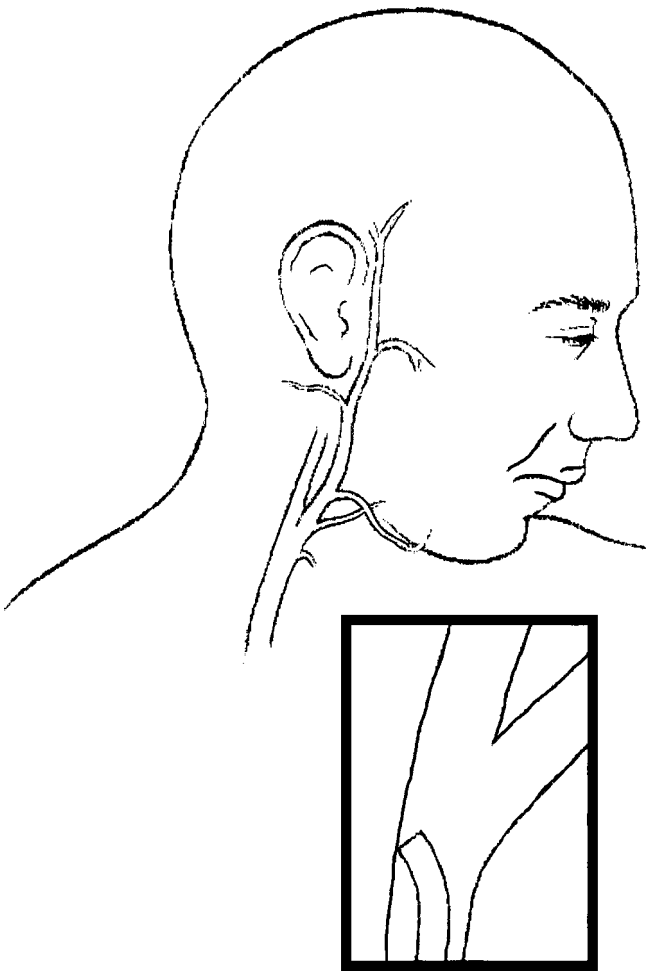

Figure 18-7 Catheterization of the internal carotid artery. The neck is flexed. This maneuver brings the internal carotid artery in line with the CCA. Note the position of the tip of the catheter, which is pointed posteriorly (inset). Adapted from [25] with permission.

*****Alternative Carotid Access:** Access to the common carotid artery is possible via the brachial or radial artery. Further more, direct puncture of the common carotid artery is feasible.

Transbrachial and Radial Approach: Puncture (radial artery and brachial artery) or cut down (brachial artery) is performed according to standard techniques. We prefer the right arm for both carotid arteries (right and left side). A 5 or 6F sheath is introduced. Cannulation

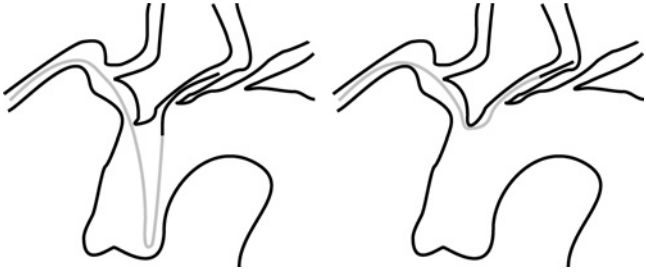

Figure 18-8 Entering the left common carotid artery from the right brachial approach: the left common carotid artery can be entered via the right brachial artery using a Sidewinder catheter.

of the common carotid artery is usually possible with a right Judkins catheter or with a Mammaria catheter. If this turns out to be difficult, a Sidewinder catheter can be used. After entering the ostium of the right or left common carotid artery with the catheter, a guide wire with hydrophilic coating (Terumo) is advanced. Over this wire, a 6Fr long sheath is introduced.

In a patient without elongation of the aortic arch the angle between the right brachial artery and the right common carotid artery is not suitable for the transbrachial approach, whereas in a patient with aortic arch elongation the angle is often more favourable. Figure 18-8 shows how to enter the left common carotid artery via the right brachial artery with a Sidewinder catheter together with a Terumo wire.

In our experience, in most patients both common carotid arteries can be more easily cannulated from the right arm. This is especially true in patients with an elongated aortic arch. Figure 18-9 shows an example of a transradial stent implantation of the right internal carotid artery [34].

*****Cervical Approach Via Direct Puncture:** The patient is placed on the table with a cushion under the shoulders. The head is turned away from the side which has to be punctured. Duplex ultrasound should be used to locate and mark the carotid bifurcation.

The best puncture side is located approximately 1.5 to 2 cm above the clavicle. The position of the needle tip in relation to the carotid bifurcation has to be checked with a contrast injection before any further steps are taken. If the position is correct, the needle is exchanged for a 5 or 6F sheath over a 0.035" guide wire. After the procedure the sheath is withdrawn and gentle pressure has to be applied for 10 to 15 minutes. Protamine should not be given.

****Why a Guide Fails to Advance Through Tortuous Artery:** One major problem is the failure of advancing the catheter into the carotid artery. Persistent forward movement of the catheter will cause a loop to form in the aorta or the catheter tip flips back

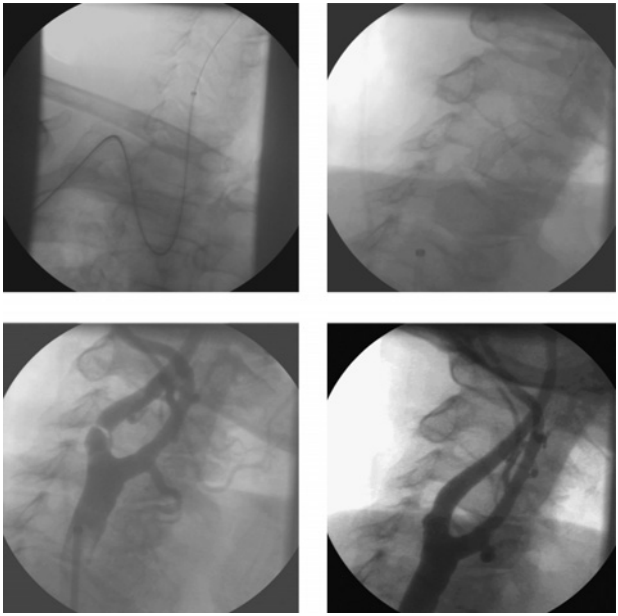

Figure 18-9 Transradial stenting of the right internal carotid artery: transradial approach via the right arm for angioplasty and stenting of the right internal carotid artery.

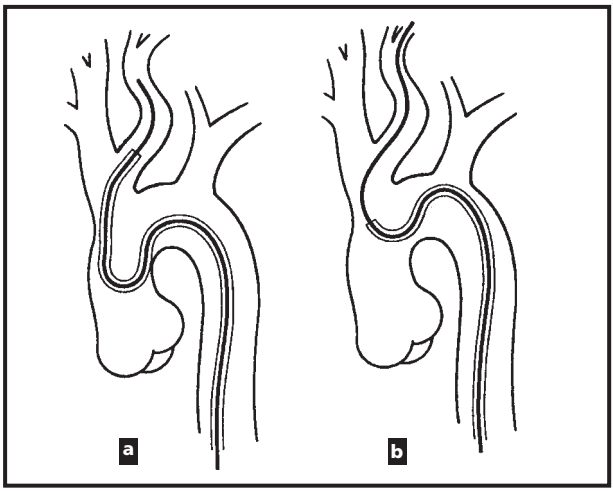

Figure 18-10 Difficulty in advancing the catheter over the guidewire. (a) The catheter forms a loop in the aorta. (b) The tip of the catheter flips back into the aorta. Adapted from [25] with permission.

into the aorta (Figure 18-10). The physical and mechanical mechanisms of the above problems are discussed below (Table 18-5). These mechanisms and their solutions can be applied universally during instrumentation of any vascular bed including the coronary, carotid, or renal arteries or their anomalies. The exampled illustrations are based on the anomaly of the left common carotid artery.

To solve the problem of a weak platform created by a floppy wire (mechanism 1), the wire has to be advanced further so the stiff segment is in the proper area. If the wire is not strong enough, it has to be exchanged to a stiffer one [35].

To solve the problem of acute angle at the origin of the artery (mechanism 2), a stiff wire will straighten out the angle and help to advance the wire. To solve the problem created by mechanism 3 due to excessive friction between the wire and the internal surface of the catheter, the catheter should be advanced and the guidewire withdrawn simultaneously (Figure 18-11). This maneuver reduces significantly the friction between

Table 18-5 Mechanisms of Failure of Advancing the Catheter

1	The wire is not strong enough to support the catheter
2	The angle of the origin of the carotid artery is too acute
3	Too much friction exists between the guidewire and the internal surface of the catheter
4	The curve of the distal end of the catheter prevents further advancement

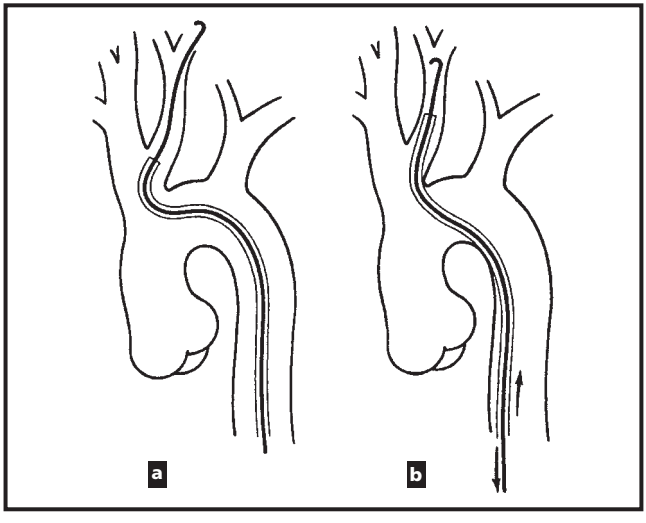

Figure 18-11 Diagram showing how to reduce the friction between the wire and the internal surface of the catheter. (a) The catheter tip is at the orifice of the left common carotid artery and the tip of the wire is in the left internal carotid artery. (b) The catheter is advanced while the wire is withdrawn. Adapted from [25] with permission.

the wire and the internal surface of the catheter. Another way is to change the size of the wire to a smaller one, although this wire would not provide the same support as the previous wire; however, it would help to advance the catheter if the problem is related primarily to friction rather than support [36].

To solve the problem of mechanism 4 (a sharp angle at the end of the catheter), while the wire is fixed, the catheter is advanced over it while rotating the catheter gently. The goal is to straighten the distal segment of the catheter by the wall of the artery so the catheter can adopt itself more to the angle and be advanced further (Figure 18-12). In difficult situations, two or three of the above-mentioned maneuvers may be required before the tip of the catheter can be advanced to the desired level.

*****Carotid Access in Presence of Occluded ECA, CCA Lesion Below Bifurcation, or Ostial CCA Lesion:** Where there is occluded external carotid artery, common carotid artery lesion below bifurcation, or ostial common carotid artery lesion: placing the 7Fr 90 cm access sheath into the common carotid artery may present special challenges when the external carotid artery is occluded, a critical lesion is situated below the bifurcation, or there is a critical ostial common carotid lesion. If possible, avoid crossing the lesion with a stiff 0.038" wire since this is more likely to disrupt the necrotic plaque material and cause distal embolization. When possible, advance the

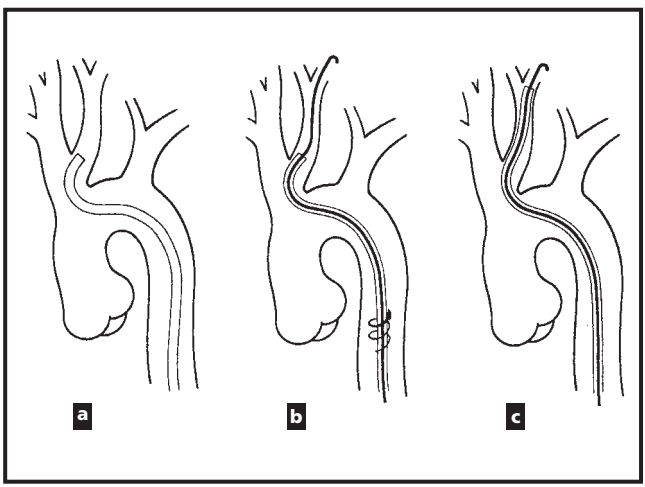

Figure 18-12 Straightening of the tip of the catheter by the wall of the artery. (a) The tip of the catheter is at the orifice of the left CCA. (b) While the wire is fixed, the catheter is advanced over it using rotating forward movement. (c) The catheter has advanced over the wire into the vessel. Adapted from [25] with permission.

5Fr diagnostic guide over the 0.038" glidewire to be placed more distally. In this situation, the glidewire and 5Fr guide are first advanced through the lesion. This maneuver should be done only in patients considered at high risk from carotid surgery if the risk-benefit ratio still favors stenting.

In the presence of a carotid ostial lesion, the origin of the common carotid artery should be first dilated to allow sheath access. The bifurcation should be stented first, and the ostium stented with a Palmaz stent on the "way out".

*****Choice of Balloon Expandable or Self-expandable Stents:** The use of balloon expandable stents was abandoned with three exceptions listed in Table 18-6. Forcing the current high-profile delivery systems may break off plaque and cause distal embolisation. In this situation, a short balloon expandable stent may be placed to hold the lesion open before passing a definitive self-expanding stent.

****Post Dilatation:** It is safer to underdilate than overdilate the oversized self-expanding stents. Overdilatation squeezes the atherosclerotic material through the stent mesh, causing emboli. A 10–15% remaining stenosis does not cause clinical problems. Importantly, it is not necessary to dilate the stent to obliterate segments of contrast-filled ulcerations external to the stent. This angiographic appearance is of no prognostic significance and follow-up angiography has documented complete fibrotic healing of these lesions over time. Importantly, it is not necessary to overexpand the stent to produce a 0% residual diameter narrowing. Covering the external carotid artery with a stent does not cause problems. Our follow-up arteriograms showed the external carotid artery to be patent with rare exceptions. If the external carotid artery becomes significantly stenosed with <TIMI-3Flow or occlude after post dilation of the stent, this vessel can be approached through the stent mesh, and reopened using coronary balloon techniques. A 0.014" wire is used to enter the external carotid artery, a 2 mm balloon to predilate, and a 4 mm balloon for final dilation. However there is almost never a clinical indication to do this.

Table 18-6 Indications for Use of Balloon Expandable Stents

1	When the ostium of the common carotid artery is treated and the proximal end of the stent has to be placed with precision
2	When the most distal segment of the internal carotid artery is treated (present delivery systems for a self-expanding stent cause dissections in the petrous portion of the internal carotid artery)
3	When the self-expanding stent delivery system will not pass through a calcified, recoiling lesion

COMPLICATIONS

Although major complications can be encountered during the learning curve of carotid angioplasty and stenting [37], they are minimized by the use of meticulous techniques.

Thrombotic and Embolic Complications: Advantages of the endovascular approach over endarterectomy include the ability to immediately diagnose and treat these complications, and the patient can be awake, allowing close neurological monitoring. For acute thrombosis, local intra-arterial thrombolysis can be carried out using mechanical as well as chemical disruption of the clot. Extreme care must be exercised to avoid vessel perforation. Only very flexible microcatheters and soft wires may be used in the intracerebral circulation.

To prevent thrombotic complications, investigators have advocated the use of glycoprotein IIb–IIIa platelet inhibition [38]. However, this encounters the risk of cerebral bleeding and therefore it should not be used routinely. Today cerebral protection devices are widely used although randomized trials could not prove a benefit of protection [38–40]. Several registries and a review article [38] demonstrated possible efficacy of protection devices. Atherosclerotic debris can be found in the filter in the majority of cases. Therefore, most investigators consider it to be unethical to conduct such a trial.

Carotid Artery Spasm: Guidewire-induced phenomena are minimized by the use of 0.014–0.018" wires. Carotid artery spasm can be successfully treated with palavering [41] or nitroglycerin. Often they disappear spontaneously.

Transient Bradyarrhythmias and Hypotension: Mediated by stretch of the carotid baroreceptors. This usually can be avoided by atropine given at least 2–3 minutes before balloon inflation. Asystole is very rare, but if it occurs, it is transient and resolves with balloon deflation. A routine pacemaker is not necessary.

Post-stenting Hypotension: Mediated by stretch of the carotid baroreceptors. Treat aggressively if the patient has severe distal or contralateral disease. Puncture site complications should be ruled out [42].

External Carotid Artery Occlusion: Acute occlusion of the external carotid artery is well tolerated. In the absence of collateral circulation, patients may experience jaw muscle angina which is usually transient.

Stent Restenosis: The restenosis rate for carotid stenting is less than 10%. It is treated with balloon dilatation [41,43] A new stenosis may occur at the distal end of a stiff stent. This may require an additional stent.

Carotid Perforation: This can be seen after excessive balloon sizing prior to or after stent placement. If encountered, try to seal it with prolonged balloon inflation. Covered stents can be used if there is no compromise of major side branches.

Carotid Dissection: This is seen mainly in areas of vessel tortuosity or calcification. Stented segments should not be overdilated in comparison to the reference vessel. Further stenting may be necessary to avoid flow disruption in the area of dissection. Figure 18-13 shows contrast dye outside of the carotid artery after stent implantation.

Cerebral Hemorrhage: Associated with a combination of excessive anticoagulation, uncontrolled hypertension, intracranial vessel manipulation, and stenting after a recent stroke (<3 weeks). Terminate the procedure, reverse the anticoagulation and control the hypertension. An emergency brain CT scan should be performed. Operators should be familiar with the angiographic features of an intracranial mass effect. Sudden loss of consciousness preceded by a severe headache in the absence of intracranial vessel occlusion should alert the operator to this devastating event. Fortunately, with careful patient selection and compulsive attention to the above technical and anticoagulation issues, cerebral hemorrhage should be a very rare occurrence.

Jaw Claudication: After carotid stenting, some patients complain of pain when masticating, especially if the external carotid artery is jailed. Jaw claudication should slowly disappear in 1–2 weeks.

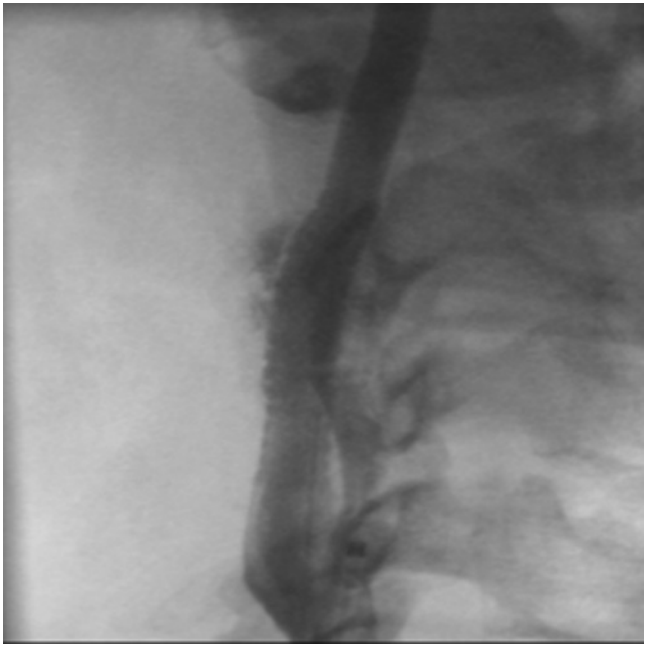

Figure 18-13 Carotid dissection with contrast dye outside of the carotid artery after stent implantation.

Problems and Complications With Cerebral Protection Devices

Cerebral protection devices may also cause problems. All devices placed distally in the internal carotid artery may cause spasm or dissection. Rarely additional balloon inflations and/or stent implantations have been necessary to solve the problem. It may be difficult to retrieve these devices through the implanted stent. It may occur that the filter is not fully apposed to the vessel wall. In contrast, the major disadvantage of the occlusion devices is intolerance in patients with occlusion or high-grade stenosis of the contra-lateral internal carotid artery or patients with poorly developed intracranial collaterals. A specific disadvantage of the MO.MA and the Gore NPS device is the need for a larger sheath which may cause vascular access problems.

FUTURE DIRECTIONS

Future developments in the field of carotid percutaneous intervention will include new stents with higher flexibility which can be introduced through smaller sheaths. We will have improved cerebral protection devices with better wall apposition and without need for a retrieval catheter. All these new developments will help carotid stenting to become the new gold standard for treatment of carotid atherosclerotic disease within the next few years.

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Chapter 19
Subclavian Artery Interventions

Gianluca Rigatelli, Paolo Cardaioli

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complication

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GENERAL OVERVIEW

In recent years, percutaneous transluminal angioplasty (PTA) of the supraaortic vessels, especially the subclavian and innominate arteries has become the treatment of choice for the majority of patients with outcomes equal or superior to surgery [1,2]. Because the subclavian and innominate arteries supply the blood to the brain and arms, in case of proximal obstruction, both territories compete for blood flow and the clinical situations one can encounter may be very diverse [3]. The subclavian steal syndrome arises if there is flow reversal in the vertebral artery shunting blood away from the brain and results in the symptoms of vertebrobasilar insufficiency, including dizziness, vertigo, ataxia, diplopia, nausea, vomiting, and syncope. Ipsilateral upper extremity ischemic symptoms, including arm claudication, paresis, and atheroembolic digital ischemia. In patients with internal mammary artery (IMA) graft to the coronary bed, ipsilateral critical subclavian artery stenosis might cause myocardial ischemia to the territory it supplies and this is clinically known as coronary-subclavian steal. The various clinical presentations are discussed below.

1 The subclavian-steal syndrome when blood is diverted from the vertebral arteries with symptoms involving the posterior cerebral circulation.
2 Acute ischemia or chronic ischemia of the upper extremity when a subclavian artery stenosis is the cause of thromboembolism or when is obstructed with disabling extertional discomfort.
3 Coronary-steal syndrome when the blood is diverted from an arterial mammary graft which supplies the left coronary system in favour of the left upper extremity, in case of subclavian proximal stenosis.

Atherosclerosis is the main cause of subclavian stenosis but other etiologies include fibromuscular dysplasia, neurofibromatosis, arteritis, inflammation secondary to radiation or compression syndromes. Traumatic injuries of the subclavian artery may lead to acute upper limb ischemia and may be caused by shoulder dislocation, shoulder fracture and injury. Dissection of the subclavian artery is very rare but it can occur after car accident or associated to the dissection from the thoracic aorta. Pseudo-aneurysm of the subclavian artery could be

formed as complication of venous line placement or late complication of a chest blunt trauma [4].

NON-INVASIVE EVALUATION

Clinical evaluation of a suspected subclavian stenosis is very simple by measuring the blood pressure of both arms: a difference of more of 20 mmHg is highly suggestive. The standard work-up of a subclavian artery stenosis includes ultrasound, computed tomography and magnetic resonance imaging.

Ultrasound: Ultrasound is more effective in detecting distal subclavian stenosis than proximal stenosis that are most often unrecognized. However, ultrasound is important in assessing the subclavian steal syndrome by detecting the flow inversion in the vertebral artery and in assessing the coronary subclavian steal syndrome by evaluating the eventual inversion of blood flow in the internal mammary artery. Ultrasound is also useful in analyzing the plaque composition as in carotid artery PTA, soft plaque prone to embolization and should be managed with distal protection device.

Computed Tomography and Magnetic Resonance Imaging: CT scan of subclavian arteries is highly effective in detecting the subclavian artery stenosis. Multidetector scan may be useful in planning endovascular treatment of subclavian stenosis, especially in complex case, when the vertebral artery origin is not readily shown by ultrasound. CT scan is indicated in particular in cases of subclavian dissection, thoracic aorta dissection involving the subclavian artery and subclavian artery pseudoaneurysm [5]. MRI is helpful in patients with impaired renal function but its utility is limited in cases of suspected occlusion of the subclavian artery.

INVASIVE EVALUATION

Angiography: Digital angiography of the subclavian artery remains the gold standard for assessing any significant stenosis. In patients undergoing angiography for coronary arteries, venous and arterial graft, subclavian artery angiography, even in the absence of clinical symptoms, should be always performed in order to detect possible subclavian artery stenosis [6]. The technique through the femoral approach includes the following. (1) Study of the thoracic aorta and subclavian artery takeoff by contrast injection through a pigtail catheter positioned in the ascending thoracic aorta in antero-posterior projection: 5Fr catheter is preferable over 4Fr catheter due to the larger amount of injectable contrast (25 cc is usually enough). (2) Study of the subclavian artery itself by a 5Fr Judkins Right or Multipurpose diagnostic catheter placed at the ostium of the subclavian artery (10 cc is usually enough). (3) Use of multiple projections: first, the antero-posterior and then ipsilateral oblique projection to exactly assess the origin of the artery and its correlations with the vertebral and internal mammary artery. Manual pullback with a diagnostic 5Fr catheter from distal to

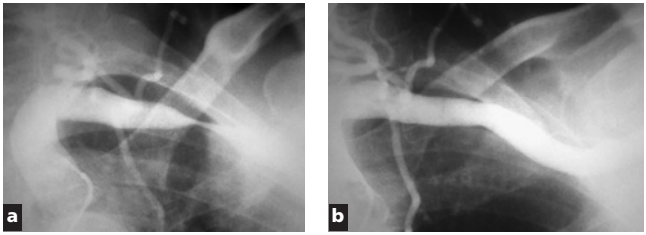

Figure 19-1 Thoracic outlet syndrome angiographic appearance: subclavian stenosis during subclavian angioplasty in patient scheduled for internal mammary graft for triple coronary vessels disease (a). After having asked to moving the arms behind the head, a complete relief of the stenosis (b).

proximal subclavian artery to detect pressure gradient: 25–30 mmHg of gradient are usually recorded for significant stenosis. When a stenosis of the body of the subclavian artery is detected, be sure that it is not a case of thoracic outlet syndrome by asking the patient to move the arm up behind the head and then check the flow (Figure 19-1).

Intravascular Ultrasound: When the length and severity of the stenosis could not be assessed accurately by angiography, intravascular ultrasound may help to define the lesion severity, plaque composition and lesion length in order to select the correct balloon-stent system.

STENTING

Access: Either the femoral or brachial access may be used. 6 or 7Fr sheath should be placed according to the location of the lesion in either the right or left subclavian artery. Brachial access may be preferable in case of total occlusion or in patients with coronary subclavian steal syndrome.

The brachial or radial approach is mandatory to cross the occlusion segment at the ostial or proximal part of the subclavian artery, since guides and wires engaged in the aortic arch are usually unable to provide enough back up and penetrating force. If the takeoff of the subclavian or innominate artery is at such a steep angle to the aorta or when severe aorto-iliac disease is present then the brachial access is preferred. The low brachial approach near the olecranon fossa is better because of the difficulty in holding pressure to the brachial artery in the upper arm [7]. The axillary approach is not used because of possible brachial plexus injury from a hematoma: the main cause for recanalization failure is the creation of the subintimal false lumen with a wire. IVUS (intravascular ultrasound) can play an important role in controlling the wire position during recanalization [8].

Guide: Usually a 5, 6 or 7Fr Judkins Right or Multipurpose guide offers a good support and is quite atraumatic. Sometimes, in case of angled take-off, a Sidewinder or a Vitek guide is very helpful. Catheterize the subclavian artery by positioning the guide in the aortic arch.

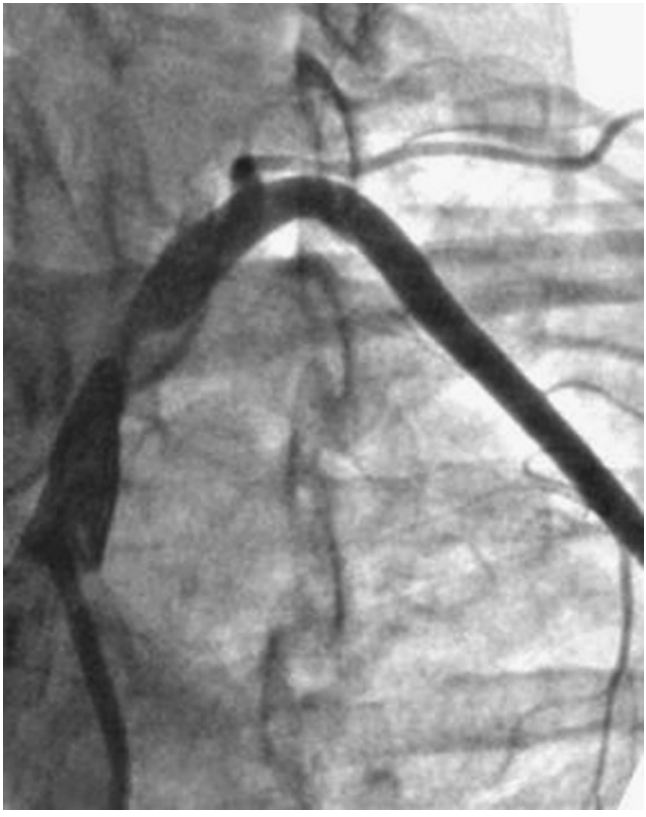

Figure 19-2 Catheterization of the left subclavian artery. Through a standard multipurpose guide.

Slowly rotate clockwise the guide in order to lift its tip upwards and to engage the subclavian ostium. In case the tip engages the right subclavian artery, do not pull the guide from the right subclavian to the left common because it may lead to dissection or embolization from the plaque (Figure 19-2). Just remove it from the right subclavian artery first, then re-orient the guide in order to engage the left subclavian. Once in place, check the position with a 5–7 cc contrast injection and take a reference pictures. Roadmapping technique is quite useful to avoid excessive contrast injection and to ensure correct wire placement.

Wires: A 0.035″ soft-tip performable wire such as the Storque (Johnson & Johnson) can be used in case of non-subocclusive stenosis; a 0.014″ high support hydrophilic coronary wire may be selected in case of subocclusive disease, when predilation is needed or when protection

filter is to be placed [9,10]. Place the wire in a safe position, distal to the cervical and mammary artery: check the position with a small contrast injection or in roadmapping technique. Protect the vertebral artery when the subclavian stenosis is closed to the origin of the vertebral artery: a non hydrophilic .014″ wire should be advanced into the vertebral artery.

TECHNICAL TIPS

***Crossing the Lesion:** Pre-form the wire tip as a smooth J curve: avoid a too-angled wire shape that may lead to ostial dissection. Be gentle with wire manipulations to avoid causing the wire to dissect across the ostium. If a dissection occurs, stop and have the patient return at a later date [7].

****Sheath or Guide:** Whether with a guide or a sheath, it is important never to compromise the ability to inject contrast material to visualize the lesion in relation to the balloon or stent catheter. Likewise, it is essential to obtain the best angle to see the takeoff of the vessel in relation to the aorta. It is also crucial to have the best angle to visualize the takeoff of key vessels (vertebral, common carotid, or internal mammary arteries). Because of respiration, roadmap images are not often helpful [7].

****Telescoping the Guide or the Sheath:** With the guidewire past the lesion, remove the diagnostic catheter and advance the long sheath (6Fr to 7Fr) or the guide (7Fr to 8Fr) just proximal to the lesion. If the diagnostic catheter is extra long (125–135-cm), telescope the sheath or guide over the diagnostic catheter and save a step. Never advance the sheath over and past the lesion [7].

Distal Protection: When a filter is needed, as in case of previous embolic episodes or in presence of a soft, ulcerated plaque (Figure 19-3a), an eccentric filter is preferred, at least when the subclavian artery diameter is about 5–7 mm. Select a concentric filter when the

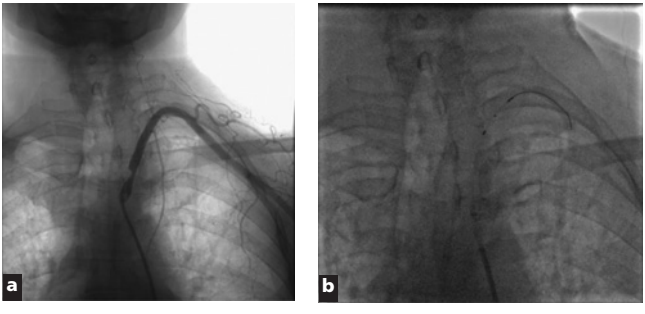

Figure 19-3 A soft embolizing plaque is detected on subclavian artery angiography (a). Filter wire has been selected and placed distally to the internal mammary artery (b).

diameter of the vessel is about 7–9 mm. The indication for neuroprotected PTA is a tight stenosis (especially a soft plaque) involving the origin of the vertebral or right common carotid artery, with residual antegrade flow in the vertebral or right common carotid artery [8].

Patients with antegrade vertebral flow may be particularly vulnerable to cerebral embolization during vertebral and carotid angioplasty, indicating that retrograde flow in vertebral artery plays a protective role against brain embolization. Subjects with a large soft plaque involving origin of the vertebral artery and no subclavian steal are to be at particularly high risk of brain embolization. In such patients the double balloon kissing technique using (preferably proximal) neuroprotection system should be considered [8].

Place the filter proximally to the origin of the vertebral artery if there is enough space or distally to the internal mammary artery if there is no bypass graft with the left internal mammary artery (Figure 19-3b). Placing double filter for vertebral and distal subclavian artery is very challenging and difficult. Most of the time, protect the vertebral artery only with a nonhydrophilic wire and the distal subclavian artery with a filter. However, in lesions involving the vertebral origin the need to remove the protection device before subclavian stenting limits its application [8].

Balloons: Predilation may be needed when dealing with subocclusive disease so the stent is not stripped during tight passage. Inflate the balloon slowly (1 atm/2–3 sec) and watch as the lesion is being modified. Avoid high pressure, especially in case of highly calcified lesions: 6–9 atm are usually enough. Unlike other major arteries, the origin of the subclavian artery is somewhat fragile, so always be cautious not to overdilate this vessel for fear of rupture, which can have catastrophic results [7].

Stents: Stent use has become the standard of care in subclavian stenosis endovascular repair, because simple angioplasty resulted too much in restenosis [10]. Stent types and sizes differ accordingly to lesion diameter, length and morphology. Focal, calcified ostial stenosis can be simply managed with stainless steel balloon expandable stent: they can be simple expanded into the ostium and their placement is easy without excessive contrast injection, thanks to their excellent radiopacity and radial force. Long or soft lesions involving the ostium can be treated with nitinol self-expandable stent: their placement is more difficult because of the risk of missing complete coverage of the ostium, but the close design of the strut minimizes the risk of plaque shift and embolization.

TECHNICAL TIPS

****Selection of Stent:** For lesions involving the proximal segments of the left subclavian, left common carotid, and especially the innominate artery, we always use a balloon-mounted stent. The chance of compression and deformation of the stent is low. Self-expandable

stents are not chosen because of the inability to be exactly precise in a region where millimeters count. Furthermore, there is the possibility of stent migration with the self-expandable stents [7].

****Stent Positioning:** Another feature to be aware of is dramatic aortic pulsations when deploying a stent. If there is a large gap in diastolic and systolic blood pressures, there can be an excess of pulsations of the vessels. These pulsations can cause large motions (1 cm or more) in the position of the lesion relative to the balloon catheter or self-expanding stent when trying to deploy. Blood pressure control is essential in these patients, as well as the need for slightly longer stents [7].

****Perfect Stenting:** For a stainless steel stent, be sure to maintain the stent 2–3 mm out of the ostium into the aorta (Figure 19-4). Be sure that important vessels such as the internal mammary and the vertebral arteries are not compromised. When using balloon-expandable stent, it is sufficient to have a diameter sightly inferior to the vessel size: The majority of the balloon-exapndable stent can be expanded to the correct size with postdilation balloon and minimal shortening. This ensures a safe procedure and minimizes the risk of vessel dissection or rupture. When using a self-expandable stent, select a stent with a diameter equal or slightly superior to that of the vessel. Maintain the stent 3–4 mm out to the ostium in the aorta to assure full coverage of the ostium. Check the position repeatedly with 4–5 cc of contrast; avoid roadmap technique during self-expandable stent deployment. If you have protected the vertebral artery, inflate the stent to nominal pressure then place the subclavian guide wire into the vertebral artery and pass the vertebral wire into the subclavian artery. Then, hold the balloon tightly because of movement from the aortic arch pulsations

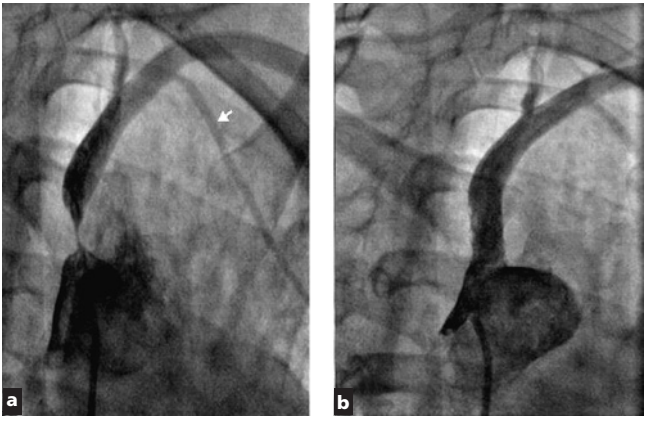

Figure 19-4 Stenting of an ostial subclavian artery stenosis: baseline (a) and final result (b) using a balloon expandable stent.

and then deploy the stent quickly to approximately 8 atm [7]. Repeat angiogram to assess the stent apposition to the vessel diameter. Perform a kissing balloon if needed (3.5 mm in diameter balloon is usually sufficient for vertebral artery).

Postdilation: Postdilation is usually required especially in case of ostial stenosis. Mono-rail peripheral balloons 7 to 9 mm in diameter help to achieve good results. If a .035″ system has been used, be sure that the length of the balloon catheter is at least 120 cm, because normally, peripheral balloon catheters are 80 cm in length.

TECHNICAL TIPS

****Postdilation:** First inflate the stent, deflate and then withdraw the balloon at least for an half outside of the proximal end of the stent and inflate there to flare the ostial portion of the stent. Don't use high pressure: 8–10 atm are usually effective. Higher pressure can make the balloon to slip out of the stent and damage the distal subclavian artery. Be happy of the results if the gradient across the lesion falls to near 0, even if the angiographic result doesn't look satiafactory. Restenosis is unlikely for stent of such diameter of >6–7 mm.

Distal Subclavian Axillary Lesions: When there is indication for intervention, angioplasty is generally preferred at the crucial areas, such as between the first rib and clavicle, as well as at the subclavian/axillary junction where there is bending and compression. When the lesion does not respond to angioplasty, then self-expanding stents, such as Wallstents (Boston Scientific Corporation, Natick, MA) and nitinol stents should be used. The stent should be oversized by 1–2 mm greater than the vessel diameter and delivered and deployed through a long 7Fr to 8Fr sheath. Interestingly, there is a lot of slack that must be removed when deploying self-expandable nitinol stents. Furthermore, care must be taken to watch the proximal end of the stent, which tends to jump or shrink farther distally than planned [7].

Inominate Artery Lesions: The technique for innominate artery lesions is similar to stenting the left subclavian and the left common carotid arteries. Attention must be given to the bifurcation of the right common carotid and the right subclavian arteries. For disease that exists at the origin of the vessels, kissing stents may then be required. There has been some debate regarding the use of distal embolic protection in treating right subclavian artery disease, especially if the disease is close to the ostium of the subclavian artery. Distal protection should be used with a filter placed in either the internal carotid artery or the common carotid, depending upon the carotid diameter and filter size available. The 7.5-mm Guidant Accunet (Guidant Co, Indianapolis, IN) is often large enough to protect patients with small common carotid arteries [7].

Associated Vertebral Disease: Associated vertebral artery can be treated concurrently with subclavian artery stenosis using kissing

balloon technique and T-stent technique if needed: however, it is very rarely required to treat vertebral and subclavian artery together. The double balloon or stent procedure can be useful only in case of very closed proximity of a diseased vertebral artery and in presence of clear clinical indications (e.g. controlateral vertebral occlusion) [9].

Subclavian Total Occlusion: Subclavian artery recanalization remains a debated issue: complication rate is higher than for subclavian artery stenosis and results are somewhat inferior even in the stent era. In case of chronic occlusion, the rules to be followed are the same in regards to catheter, balloon, and stent selection whereas approach and guide selection differ substantially.

TECHNICAL TIPS

****Preferred Vascular Access:** The presence of a nipple favors the femoral approach whereas the absence of a nipple makes brachial approach the preferred one. The ideal wire is the hydrophilic .035″ wire in almost all cases. Therefore, high support hydrophilic .014″ coronary wires may be selected in specific cases. In very difficult cases, a long wire between the brachial and femoral artery, therefore creating a strong arterial loop, may help to advance the balloon-stent system through heavily calcified lesions.

Be sure that the patients really need such procedure. Better if you have a vascular surgical second opinion and stand-by. Be careful when advancing the wire: check the position in two orthogonal planes. If the wire position is not sure, exchange the wire with a 4 or 5Fr hydrophilic exchange catheter (Glidecath, Terumo Co, Tokyo, Japan) and inject from the catheter itself. Use of rheolytic thrombectomy catheter or manual aspiration catheter through a 7–8Fr large lumen guide may be useful in recanalizing acute or subacute occlusion, a really very rare occurrence. Stent implantation is usually performed after recanalization to stabilize the plaque.

CASE REPORT Coronary-Subclavian Steal Syndrome: A patient underwent coronary bypass grafting with the left internal mammary artery (LIMA) may develop a coronary subclavian steal syndrome because of a left subclavian artery (LSA) stenosis. Usually stenting of LSA is performed by the femoral route with a guide technique. This technique has clear drawback in case of coronary subclavian steal due to the poor opacification of the LIMA ostium, and a difficult access to the LIMA in case of plaque shifting, especially when the vertebral artery and the LIMA ostia are very close to the LSA stenosis. In this case, subclavian angioplasty and stenting should be performed from the left brachial artery access [11,12].

TECHNICAL TIPS

*****Monitoring the Intervention with Contrast Injection through the Sheath:** Use as a 6Fr or 7Fr guide with a 45 cm-long

valved anti-kinking sheath as the Super Arrow Flex sheath (Arrow International) or the Cook Shuttle Check Flow (Cook Co, Bloomingotn IN): insertion of the long sheath should be monitored under fluoroscopy during the advancement to the subclavian artery ostium. It is preferable to use an hydrophilic anti-kinking sheath as the Arrow or Cook ones to minimize the arterial damage (Figure 19-5a). Choose the diameter of the sheath in order to allow for injecting sufficient contrast volume through the sheath itself even when the balloon catheter is inside during the deployment of the stent. Place the sheath just before the LIMA graft ostium and engage the lesion with a.035" 260 cm-long Storq guidewire (Cordis Inc, Johnson & Johnson, Warren, NJ) and leave it to the descending aorta (Figure 19-5b). A balloon-expandable endovascular stent or a self-expandable stent can be implanted. It is important to check the correct position by direct contrast injection through the long sheath (Figure 19-5c).

This technique may be considered the optimal route to treat coronary subclavian steal syndrome because of clear advantages: no manipulation of catheter to cannulate the artery, perfect coaxial position of the catheter at the site of LSA stenosis, clear visualization of the LIMA and vertebral ostia, easy access to these vessel in case of plaque shifting or embolic protection device deployment. Moreover, the procedure

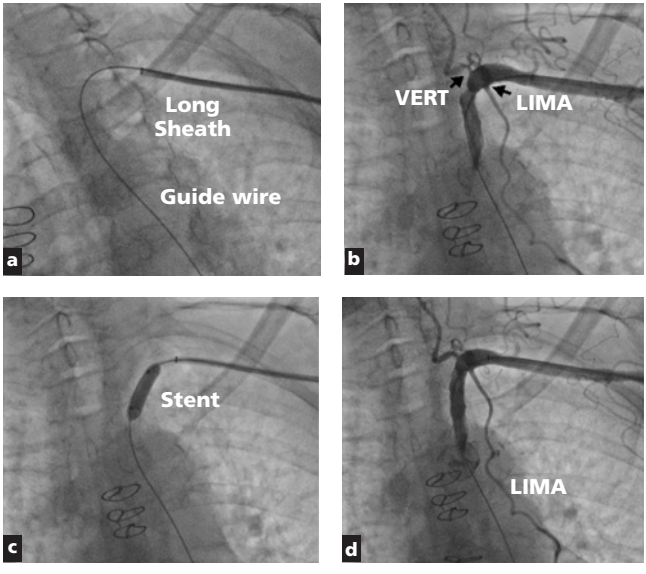

Figure 19-5 Subclavian artery tight stenosis in a 70-year old female patient. (a) Advancement of the long sheath to the lesion site. (b) Baseline angiography: the stenosis is very closed to the LIMA and vertebral artery origin (arrow). (c) Genesis stent deployment. (d) Final result after angioplasty and stenting: the LIMA and vertebral artery are perfectly patent.

can be accomplished with a very little amount of contrast and probably similar stenosis visualization than the standard femoral route.

Injuries and Pseudoaneurysm: Subclavian pseudoaneurysm can be approached by femoral route through an 8Fr guide due the bigger size of the guide needed to advance the covered stent. The procedure can be performed easily as described above for standard subclavian artery stenosis. Rupture of the subclavian artery constitutes a life-threatening condition in which sometimes interventional techniques are useful in minimize operative stress and operation time particularly in case of complex multi-trauma settings.

TECHNICAL TIPS

***Stenting of Subclavian Traumatic Injury:** Use femoral route because the brachial route may be inaccessible due to pulse loss. Be sure to place the wire in the true lumen of the vessel by placing it in the descending aorta. Select stents of overestimated length to be sure to cover the lesion. Obtain a venous access and then check the status of the subclavian vein: often the rupture involves also the vein.

CASE REPORT Stenting for Injury-related Subclavian-Arterial Venous Fistula: A young man sustains a 1 cm stab wound in the supraclavicular fossa. Emergent aortic arch angiogram confirmed a large arterial venous fistula, but dilution of contrast made accurate definition of the anatomy very difficult. A catheter was then navigated into the innominate artery, and high-volume contrast angiograms were performed further defining the fistula. Selective angiography of the thyrocervical trunk and ipsilateral internal mammary artery was performed without difficulty. This confirmed that the arterial venous fistula was distal to the origin of these vessels. Further manipulation of the catheter allowed advancement through the arterial venous fistula into the superior vena cava. The catheter was then gently retracted while injecting and ultimately returned into the subclavian artery.

Over a 0.035-inch exchange length anchor wire, a 10 mm balloon was advanced into the origin of the subclavian artery based on anatomic landmarks where it was inflated at a very low pressure. The patient hemodynamically stabilized within 2–3 min of balloon inflation. The stent graft was prepared, and immediately following balloon deflation via 9Fr sheath, a 9 mm self-expanding Wallgraft (Boston Scientific, Natick MA) was then positioned in the left subclavian artery where it was deployed using meticulous fluoroscopic guidance and anatomic landmarks without difficulty. Finally, the 10 mm balloon was again inflated at low pressure in the stent-grafted segment and final angiography showed no evidence of endoleak [13].

Complications: Complication may occur at different levels: brain complication such as transient ischemic attacks or even strokes; acute upper extremities ischemia of which the main causes are distal embolization, arterial dissection or rupture, subacute stent thrombosis.

Prevention of such complications can be obtained following clear rules, being careful at every procedural steps, accurately planning the procedure, and being aware of any potential complications.

EXOTIC COMPLEX INTERVENTIONS FOR THE URBAN WEEK-END WARRIORS

1 Occlusion of a Subclavian Artery Causing Acute Myocardial Infarction: A 64-year-old man with history of coronary artery bypass graft (CABG) including a LIMA to the left anterior descending artery (LAD) presented with severe chest pain. The initial electrocardiography demonstrated an acute anterior ST myocardial infarction. Angiography of the coronary vessels and aortic arch revealed an occlusion at the ostium of the left subclavian artery with no flow into the LIMA and LAD. There was normal flow identified in the left common carotid artery and innominate artery with no retrograde filling identified in the left vertebral artery. An 8Fr multipurpose guide was inserted with a 035" glide wire used to cross the thrombotic occlusion of the ostium of the left subclavian artery. A balloon catheter was used to dilate the lesion. A stent was deployed at the occlusion site. The angiographic result at the end of the procedure was excellent, reestablishing antegrade flow into the LIMA and LAD. Upon reperfusion the patient became pain free with significant resolution of his electrocardiographic changes [14].

2 Aneurysmal Exclusion of Subclavian Artery: A 72-year-old male presented to the emergency department with a possible mediastinal mass on chest x-ray. Computed tomography (CT) of the chest revealed a large right-sided subclavian artery aneurysm measuring 5.3 cm in diameter, arising just distal to the origin of the common carotid artery. The aneurysm was noted to encroach on the trachea and esophagus with significant associated tracheal compression. Selective angiography of the right subclavian artery confirmed the aneurysm had a short proximal neck just distal to the takeoff of the right common carotid artery. The ipsilateral vertebral artery was occluded and selective angiography of the dominant left vertebral showed excellent crossover support to the right posterior cerebral circulation. The right femoral artery was cannulated and an exchange-length glidewire (Boston Scientific, Nattick MA) was advanced into the aneurysm through a diagnostic catheter and with some difficulty navigated into the distal portion of the aneurysm. A 12Fr sheath was inserted into the right brachial artery and a 10 mm snare was introduced retrograde into the aneurysm to facilitate capture of the aforementioned glidewire. The distal end of the wire was cautiously extricated to avoid injuring the subclavian artery and finally the guidewire was extending from the femoral artery to the brachial artery. A retrograde approach for insertion of the stent graft was used due to the short proximal neck of the aneurysm and concern about accuracy of placement including the potential risk of occluding the ipsilateral carotid artery. Embolization of the vertebral artery to avoid

retrograde collateral endoleak was not necessary since the vessel was occluded at baseline. When position was confirmed by fluoroscopy, the Wallgraft was partially deployed at the origin of the right subclavian artery. A soft-tip wire was then inserted in the proximal portion of the stent graft via the femoral approach and a 9 mm balloon was used to anchor the proximal portion of the device in place. After proximal fixation was assured, the stent was deployed completely. A bare metal stent was then placed in the proximal neck in order to prevent migration of the stent graft during subsequent manipulation. Final arteriogram was performed and showed no evidence of endoleak [15].

3 Coronary Steal Syndrome Aggravated by Arteriovenous (AV) Fistula: A patient with chronic renal failure treated with dialysis underwent CABG with a LIMA to the LAD. He was well and free of any angina until this presentation. Physical examination showed a systolic blood pressure difference between the right (150/70 mmHg) and left (90/60 mmHg) arms of 60 mmHg. Doppler study showed reversed flow in the left vertebral artery and normal cephalic flow in the right vertebral artery suggestive of significant left subclavian artery stenosis. Since the reverse flow in the left vertebral artery might be secondary to the high flow in the ipsilateral AVF, magnetic resonance angiography (MRA) was arranged to delineate the supra-aortic arteries anatomy. MRA documented severe stenosis at origin of left subclavian artery with subclavian steal syndrome.

The critical left subclavian artery ostial stenosis resulted in significant pressure drop in the proximal part of the artery. This resulted in flow reversal in the ipsilateral vertebral artery as documented by Doppler, MRA, and contrast angiography. While hemodialysis was performed via an AVF in the ipsilateral forearm, blood was withdrawn from the left upper arm, which would cause reduction in flow in the LIMA and flow reversal in the left vertebral artery. This was clinically manifested as angina and dizziness during hemodialysis. Percutaneous recanalization and stenting of the left subclavian artery abolished the pressure drop in the proximal subclavian artery and hence resulted in antegrade flow down both the left vertebral artery and the LIMA [16].

4 Open the Total Occlusion Subclavian Artery for Central PCI Access: A patient with severe coronary artery disease (CAD) was seen and angiogram showed total occlusion of both iliac arteries and both subclavian and inominate arteries. Given the length of the right axillary artery occlusion and the unknown length of both iliac occlusions, the chance of short-term success in these regions was felt to be lower than that of the left subclavian artery which showed a shortest occlusion length. PTA of the left subclavian artery was performed via left brachial arterial access. With the support of a guide, a 0.035" angled Glidewire (Terumo, Tokyo, Japan) was successfully used to cross the majority of the length of the totally occluded left subclavian artery. Despite multiple attempts, the angled tip of the Terumo wire failed to cross the final few millimeters of the occlusion.

After confirming proper angulation of the headhunter catheter in multiple orthogonal views, final access into the aorta was achieved by advancing the stiff end of the Terumo wire through the occlusion. The headhunter was advanced over the wire into the ascending aorta and the wire was removed. A 0.014″, 300 cm Platinum Plus wire (Boston Scientific/Scimed, Maple Grove, MN) was advanced into the aorta through the headhunter, after which the headhunter was removed. PTA of the subclavian artery was then performed. Post-PTA angiogram revealed a 70% residual stenosis, creating a channel adequate enough to provide central access for subsequent successful coronary angiography and interventions [17].

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Chapter 20
Renal Artery Interventions

Gianluca Rigatelli, Paolo Cardaioli

General Overview
Non-invasive Evaluation
- Ultrasound
- CT and MRI
- Nephro-photo-scintigraphy

Invasive Evaluation
- Abdominal aortogram
- Diagnostic renal angiography
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- Intravascular ultrasound
- Angiographic and hemodynamic criteria

Indications
Stenting
- Access
- Guides
- Wires
- Filter
- Balloons
- Stents
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- ***Sequential crushing technique for renal bifurcation lesion

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- Restenosis
- **Case report:** Aortic hematoma after stenting

Exotic Complex Interventions for the Urban Week-end Warriors
1. Complex renal stenting in patient with renal artery compromised by aortic aneurysm dissection
2. Exclusion of renal artery aneurysm by covered stent

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complication

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GENERAL OVERVIEW

Renal artery stenosis (RAS) is a relatively common problem, mainly in patients affected by hypertension and peripheral vascular disease. RAS is a progressive condition with a 16% occlusion at 1 year and is the cause of 15% of end stage renal failure in patients over 50 years of age [1].

NON-INVASIVE EVALUATION

Ultrasound: Renal ultrasound imaging and Doppler are the most cost-effective methods for the evaluation of RAS, including functional assessment, such as translesional pressure gradient and parenchymal vascular resistance estimation. The specificity and sensibility are operator-dependent and approach 90%.

CT and MRI: Magnetic resonance angiography (MRA) and computed tomographic angiography (CTA) have the same specificity (98–99%) and sensitivity (92–93%) for the detection of RAS. The choice depends on available equipment and characteristics of the patient (renal failure limits the use of iodinated contrast for CTA and ferromagnetic implants proscribe the use of MRA) [2].

Nephro-Photo-Scintigraphy: Radionuclide angiography or captopril scintigraphy, which rely on differences in renal perfusion between the two kidneys, are not useful for diagnostic screening but for functional evaluation before and after revascularization [3].

INVASIVE EVALUATION

Abdominal Aortogram: At first an abdominal angiogram is done to locate the origin of the renal arteries, identify the accessory renal arteries and presence of significant renal stenosis, especially the ostial lesion which is the most common lesion type of RAS. Also an abdominal aortogram is done to detect any coexistent aortic disease such as aneurysm, dissection, thrombus, etc.

Diagnostic Renal Angiography: Digital subtraction angiography (DSA) of the renal arteries is the "gold standard" for defining both normal vascular anatomy and vascular pathology. It remains the most readily available and widely used imaging technique. RAS more often involves the ostium (ostial RAS), and less frequently, the renal artery itself (true RAS) or both (mixed RAS) [4,5].

TECHNIQUE: Renal Angiography: The usual technique includes the placement of 4F or 5Fr Pigtail catheter level of L1–L2 above the renal artery in a left anterior oblique (LAO) 30° projection. Injection of 25–30 cc of contrast at 10–12 cc rate is usually sufficient with digital subtraction technique to depict both the renal arteries (Figure 20-1). The smallest contrast volume possible should be used when renal angiography is performed in conjunction with cardiac angiography. Selective injection by a Judkins right diagnostic catheter should be done when lesion severity is not known or when renal artery angiography is done

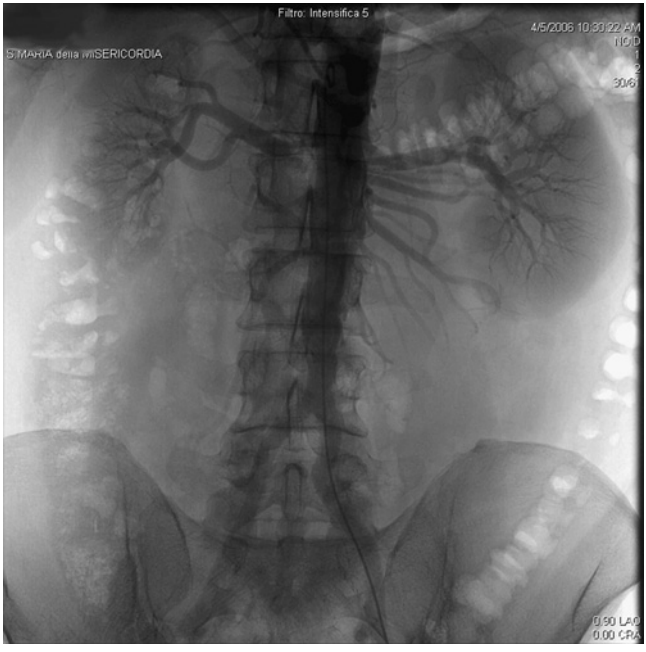

Figure 20-1 An abdominal aortography in a patient with three-vessel coronary artery disease and mild renal insufficiency revealed a tight stenosis of the right renal artery.

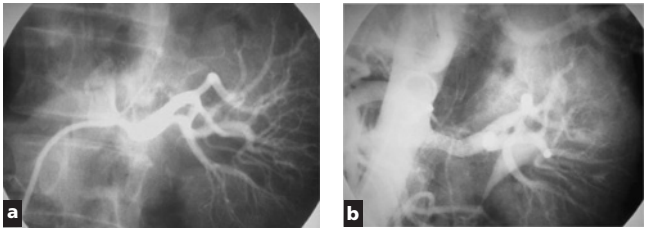

Figure 20-2 Renal artery stenosis is slightly different in patients with coronary artery disease: the normal ostial pattern can be switched to a mixed pattern (a). A mixed renal artery stenosis needs longer stent to be adequately treated (b).

following cardiac angiography. In these situations, especially in patients with borderline or impaired renal function, injection of 5–8 cc for each renal artery is usually sufficient to define any stenosis (Figure 20-2).

In patients with multivessel coronary artery disease undergoing coronary angiography, renal angiography may be useful as a part of global cardiovascular investigation to rule out renal artery disease,

Table 20-1 Indications to renal angiography during coronary angiography

Onset of hypertension >30 years or <55 years
Malignant, accelerated, or resistant hypertension
Unexplained renal dysfunction
Development of azotemia with an ACE inhibitor or ARB medication
Unexplained size discrepancy of >1.5 cm between kidneys
Cardiac disturbance syndrome (flash pulmonary edema)
Peripheral arterial disease (abdominal aortic aneurysm or ABI >0.9)
Multivessel (>2) coronary artery disease

that sometimes may be present also in patients with normal creatinine (Table 20-1) [6].

Fractional Flow Reserve: In unclear cases, lesion gradient assessment with fractional flow reserve (FFR) may be useful. A value of >0.90 can be considered a threshold value below which the stenosis is likely responsible for an up-regulation of renin production and, thus, for renovascular hypertension [7].

TECHNIQUE: Intravascular pressures measurements are performed with the Pressurewire XT (Radi Medical Systems, Uppsala, Sweden), a high-fidelity micromanometer-tipped wire (0.014" diameter). Pressure measurements are recorded 60 sec following selective intrarenal papavarine injection. The pressure measurements are averaged over 15–20 beats. The measured pressures are analyzed with SmartFlow systems (Florence Medical, Wellesley, MA) and the baseline mean pressure gradient, hyperemic mean pressure gradient, and the renal FFR are calculated. Papavarine is administered selectively into the renal artery distal to the stenosis to minimize systemic vasodilatation. A small catheter (4Fr or less) is used to cross the stenosis. Papavarine is diluted in nonheparinized saline at a concentration of 8 mg/ml and is then administered by bolus infusion into the renal artery. The infusion catheter is then withdrawn into the guide and translesional pressure measurements are performed. The results are calculated from the formula below:

Renal FFR = Q_{max} stenosis/Q_{max} normal = $(P_d - P_v)/(P_a - P_v)$. Assumingthe central venous pressure to be zero, this equation can be simplified to renal FFR = P_d/P_a. This equation suggests that in a renal artery without stenosis, the renal FFR will be equal to 1 [8].

Intravascular Ultrasound: Intravascular ultrasound may be needed intraoperatively when severity of lesion and length are not measured accurately or when the ostial disease can not be fully excluded.

Angiographic and Hemodynamic Criteria for RAS: Pullback gradient pressures are unreliable. The translesional pressure should be taken by the two catheter method which involves placement and simultaneous pressure measurement through a catheter (<4Fr) distal to the lesion and a catheter proximal to the lesion (>6Fr).

The pressure measured by the pressure wire distal to the stenosis is also acceptable. The criteria for RAS are either:

1 80% stenosis by QCA
2 Between 50–80% with >20 mmHg peak gradient.

INDICATIONS

To date, no single approach has shown clear superiority either in diagnosis or identification of patients most likely to benefit from revascularization. Revascularization of RAS is recommended for severe/drug-refractory hypertension, preservation of renal function, recurrent flash pulmonary edema, or recurrent severe heart failure, but no clear recommendations can be made for preserving or improving renal function in patients with different levels of renal dysfunction [9,10].

STENTING

For angioplasty of the RAS, there are three techniques have been proposed: the guide (direct) technique; the two catheter coaxial (indirect) technique (a diagnostic catheter is inserted into a larger guide); the wire (indirect) technique in which two femoral punctures are made, one for the wire and one for a pigtail catheter.

Access: Access is usually obtained from the controlateral femoral artery through a 5–6Fr (renal artery diameter 4–6 mm) or 7Fr (renal artery diameter 6–8 mm) sheath. Use whenever possible the controlateral approach which allows easy engagement of the renal ostium and optimal guide support (Figure 20-3). In case of severe kinking of femoro-iliac arteries or acute angulation of the renal arteries, a brachial or radial approach should be used.

Guides: Recommended guides are the renal double curve (RDC), Right Judkins for the femoral approach and Right Judkins, or Multipurpose for the brachial or radial approach. In cases of severely calcified aorta, use a hydrophilic wire with a tip 2 cm outside the guide to ensure a safe catheterization of the renal artery, especially when a 7Fr guide is selected (no-touch technique). This technique helps to avoid dissection of the aorta or the renal trunk.

Wires: Wire selection is a quite important step: 0.14″ coronary soft tip pre-formable wire may be preferable in most cases. Alternatively, a 0.18″ wire can be selected but careful manipulation is suggested due to increased stiffness. Don't use a hydrophilic wire because of higher risk of perforating the distal renal arterioles. Place the wire at the end of a main branch of the renal artery far from the renal cortex (Figure 20-4).

Filter: Distal embolic protection is believed to be helpful in avoiding distal embolization and worsening renal function, but definitive data are still lacking. The balloon occlusion (Percusurge) or eccentric (FilterWire) or concentric filter (Angioguard, Johson & Johnson Medical Corp)

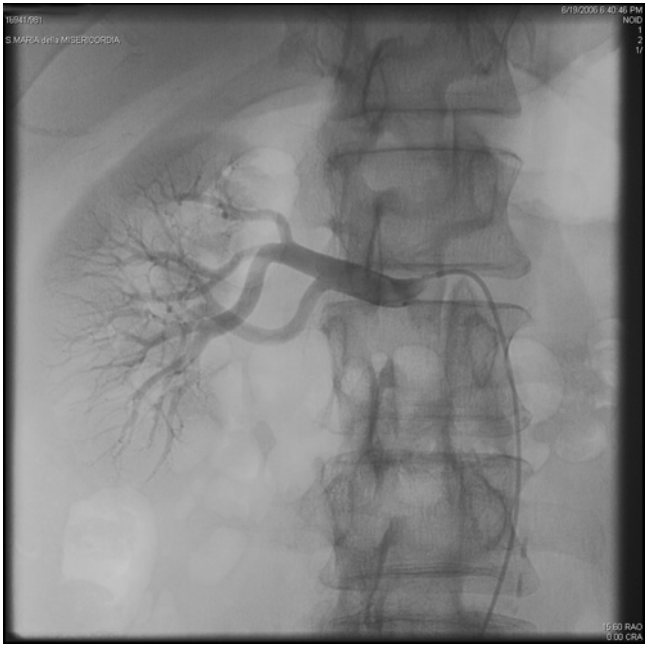

Figure 20-3 Renal artery stenosis is better engaged from a controlateral approach: this allows for obtaining more support from the guide during percutaneous revascularization.

may be selected according to the renal artery size. The eccentric or concentric filters are recommended because they are easy to deploy and less prone to renal perforation, in case of soft, opalescent and ulcerated stenosis (Figure 20-5), when the main renal trunk is long at least 15–16 mm (Figures 20-6 and 20-7). Early bifurcation of the renal artery remains a contraindication to the use of distal protection [11,12].

Balloons: Usually direct stenting is the technique of choice for the majority of lesions but sometimes predilation is needed. Low profile .014″ monorail coronary balloon 1.5–2.5 × 15–20 mm inflated to nominal pressure are usually sufficient for advancement of the stent in case of very tight stenosis. For inflation, use a manometer to inflate balloons especially when facing severe calcified stenosis. Inflate slowly 1 atm/3–4 seconds and watch as the lesion is being modified at maximum pressure of 8–10 atm. Hold the wire and balloon catheter firmly in your hand during inflation to avoid guide or balloon distal migration. Stop inflation as the patient feels pain and check for any dissection or renal artery rupture.

Stents: Proper stent selection is mandatory to achieve excellent immediate and long-term results [13,14]. High radial force

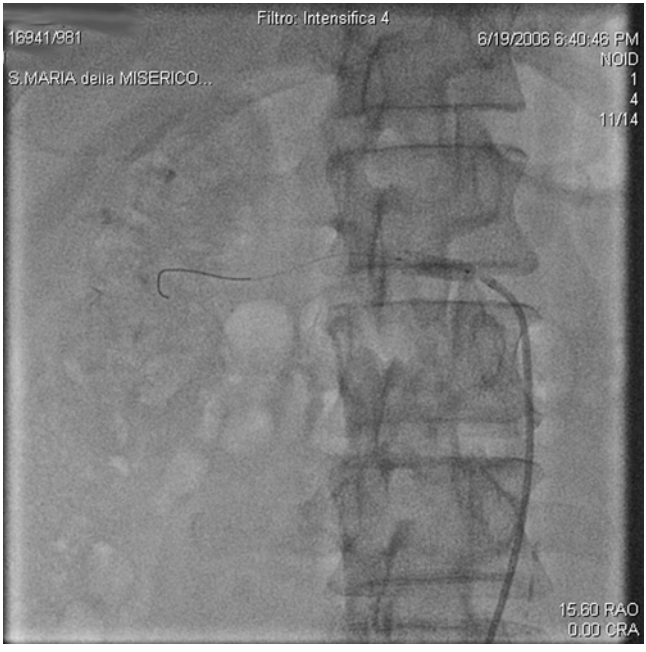

Figure 20-4 The wire should be placed not too much deeply into the renal arterioles to avoid iatrogenic perforation. A non-hydrophilic wire should be used.

stent: 4–6 mm × 12–18 mm stainless-steel balloon-expandable are the best option for focal calcified stenosis in the ostium or the main vessel. They allow precise implantation thanks to their excellent radiopacity and they can be overexpanded if needed with only minimal shortening. Long diffuse soft stenosis are better treated with 4–6 mm × 15–18 mm stainless-steel balloon-expandable stents. Drug eluting stents have been proposed and are currently under investigation.

For RAS, coronary chromo-cobalt stents are preferred compared to stainless-steel stent in case of ostial stenosis and renal artery size of 4–5 mm: their high radial force protects better the ostium. Keep the balloon-expandable stent 2–3 mm beyond the ostium in the abdominal aorta to be sure the ostium is covered. 12–16 atm pressure is usually enough to deploy the stent. After deployment, withdraw the balloon 2–3 mm outside the ostium while gently push the guide until perfect coaxiality is achieved. Then quickly inflate the balloon at high pressure to flare the ostial portion of the stent. Carefully check the final results with generous injection from the guide to assess all segments of the renal parenchyma. Change the guide at the end of the procedure for a diagnostic pigtail catheter in the abdominal aorta. Perform digital subtraction angiography to assess any aortic damage.

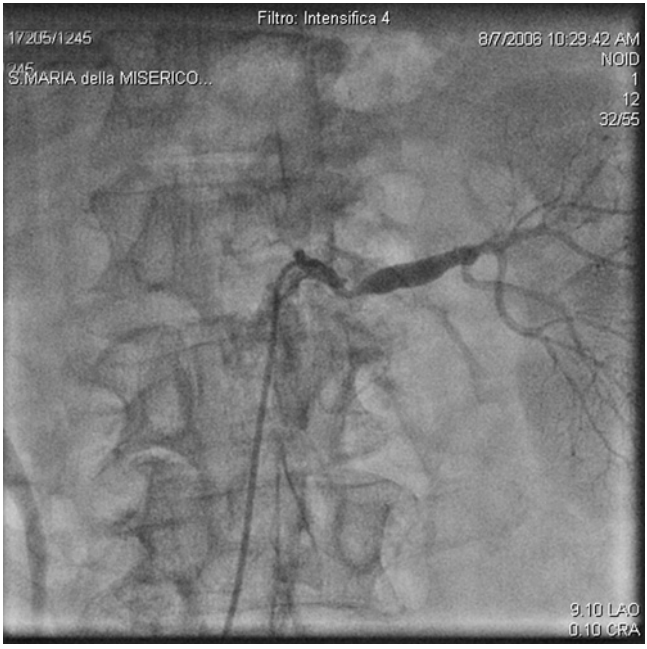

Figure 20-5 A typical renal artery stenosis which should benefit form a filter-assisted percutaneous revascularization: the bifurcation is far form the stenosis and the renal artery lenght is >15 mm.

TECHNICAL TIPS

*****Intervention in Early Renal Bifurcation (Short Renal Artery Trunk):** In case of short renal trunk a kissing balloon technique may be necessary [14]. A 7 or 8Fr guide should be selected. We strongly suggest to stent the main vessel and to dilate the secondary branches through the stent strut as in coronary interventions. Wire both the renal branches. Select the balloons size in order to match the renal artery size (e.g. 3 mm + 2 mm balloons for 5 mm renal artery trunk). After performing a kissing balloon, deploy the balloons simultaneously with two indeflators at the same pressure (10–12 atm), deflate and withdraw both balloons beyond the proximal end of the stent in the abdominal aorta then overdilate with high pressure (12–14 atm) to flare the ostial parts of the stent.

*****Sequential Crushing Technique For Renal Bifurcation Lesion:** In case of renal artery with proximal bifurcations, stenting can be performed in the same fashion as bifurcation coronary stenting. If the guide is a 7Fr guide, then usual technique is used. In case of a 6Fr guide, then sequential crushing technique can be used.

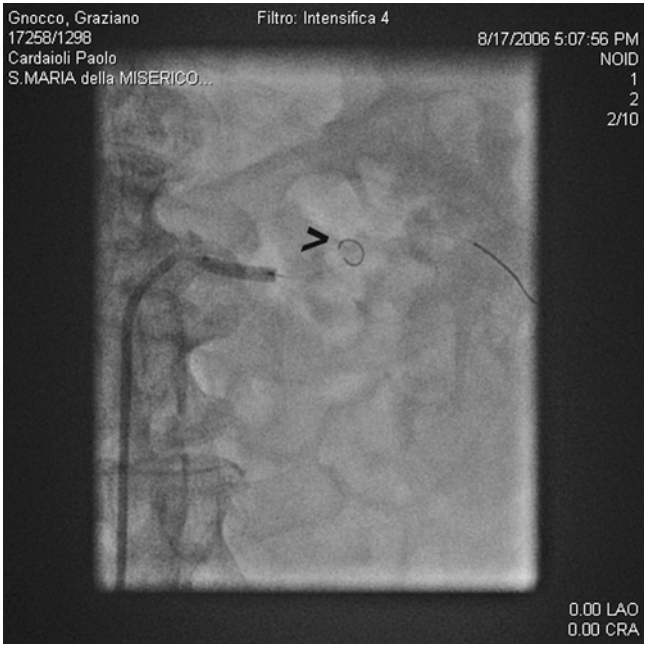

Figure 20-6 A filter wire 3.5–5.5 mm (arrow) has been placed over a .014" nonhydrophilic guide wire. A coronary balloon has been inflated to nominal pressure.

First, a noncompliant balloon is advanced into the main vessel, beyond the bifurcation, then a stent is positioned so that it covered the lesion in the secondary branch while protruding around 4 mm into the main vessel proximal to the bifurcation. The balloon in the main vessel is then retracted so that its midpoint is positioned at the level of the proximal marker of the stent. The stent was deployed at standard pressure. Then the stent balloon and the corresponding wire are removed. At this point, the balloon in the main branch is inflated to 20 atm in order to crush the proximal portion of the stent.

Then, the balloon is withdrawn, a second stent is deployed in the main branch across the bifurcation. Sequential postdilatation inflation and kissing technique is performed as usual [16].

COMPLICATIONS

Although rarely, complications may occur, some of which are potentially lethal. Complications may include distal embolization, aortic dissection, renal artery rupture, renal perforation (Table 20-2). Renal artery dissection can frequently be observed and for itself is not a major problem, being managed with stent implantation. Aortic dissection is a life threatening condition and requires surgery.

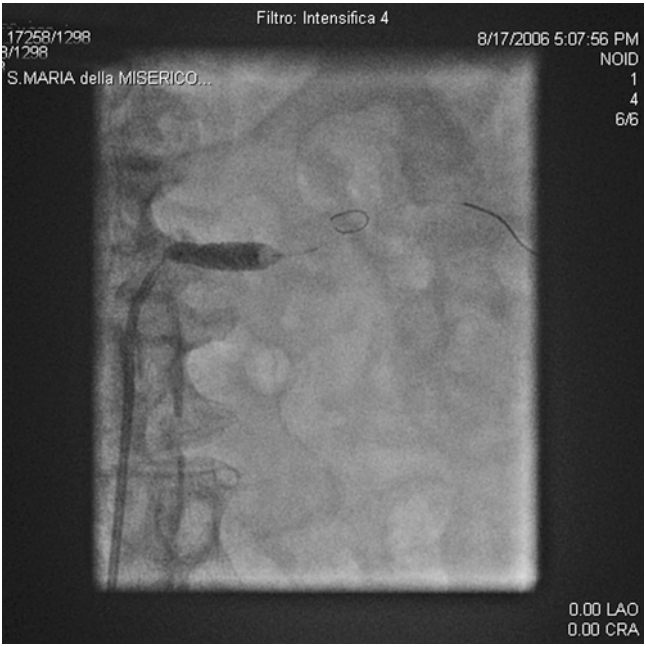

Figure 20-7 Stent deployment: note the incisure at the ostium due to a heavy calcified ostium.

Table 20-2 Management of complications

Complications	Management
Main renal artery rupture	Prolonged inflation of the balloon at low pressure – graft stents
Renal perforation	Immediate embolization
Distal dissection	Ballooning or stenting
Acute and subacute stent thrombosis	Local thrombolysis and redilatation
Cholesterol embolization	Antiaggregants, corticosteroids Cardiac and renal failure treatment

Arterial Rupture and Perforation: Renal artery rupture can be managed with covered stents of which availability in the cardiac catheterization laboratories is a prerequisite for renal artery stenting. Renal perforation needs to be assessed with angiography and possibly with CT scan and can be managed with occlusion coils. Availability of microcatheters is a prerequisite for renal angioplasty. Small coil size

should be selected depending on the perforated vessel size: usually 0.014–0.018″ platinum microcoils are used through 3Fr microcatheters. Alternatively if a microcatheter is not available an over-the-wire small balloon (1.5 mm) can be used to release microcoils. If the condition remains uncontrolled, surgical management is needed.

Restenosis: Renal artery stenting appears to be durable, with only 10% of stented arteries requiring target vessel revascularization (TVR) during clinically-based long-term follow-up. Arteries with a final stent diameter ⩽5.0 mm were more than twice as likely to need TVR, as were patients with a solitary kidney. Recent reports suggest restenosis rates variable from 36% for small renal arteries (vessel diameter $<$4.5 mm) to only 6.5% for larger renal arteries ($>$6 mm) [17]. A recurrence rate from 22–43% has been reported at 6 months after balloon angioplasty for renal in-stent restenosis. Surveillance of implanted stent is warranted by Doppler ultrasound and in particular CT angiography. Follow-up of renal function should be warranted by nephro-scintigraphy and creatinine clearance.

The treatment of in-stent-restenosis in renal arteries remains a challenge with restenting and angioplasty, especially in patients who have aggressive disease. Intravascular brachytherapy has been shown to be effective in coronary and peripheral arteries. Its use in renal arteries is not yet FDA-approved, though case reports in the literature have shown its feasibility in renal arteries. Recurrent renal in-stent restenosis (ISR) was successfully treated with paclitaxel-eluting stent implantation, using intravascular ultrasound guidance, with maintained stent patency at 6 months [19].

CASE REPORT Aortic Hematoma After Stenting: A patient was found to have a 70–80% diameter stenosis in the ostium of the right renal artery. The patient underwent renal artery stenting. After predilation with a 6 $\times$ 20 mm balloon and after a stent was deployed in the stenotic lesion, the patient complained of severe back pain. The systolic blood pressure immediately dropped from 170 mmHg to 80 mmHg. The urgent aortogram showed localized aortic dissection with intimal flap from the right renal artery. There were DeBakey type I acute acute intramural hematoma (AIH: crescentic thickening of aortic wall without contrast enhancement from the ascending aorta to the abdominal aorta), about 5 cm-sized localized aortic dissection arising from the right renal artery ostium.

In patients with hypertension, high blood pressure may injure the arterial wall, which can be dissected with these additional stresses after the balloon injury. There was a possibility of underlying medial degeneration predisposed the vasa vasorum toward hemorrhage and AIH. The aorta with more calcified atherosclerotic component may prevent the progression of the dissection. Surgery is the treatment of choice for the patients with AIH involving ascending aorta. However, in some selected cases, the intensive medical treatment can stabilize the patient [20].

EXOTIC COMPLEX INTERVENTIONS FOR THE URBAN WEEK-END WARRIORS

1 Complex Renal Stenting in Patient with Renal Artery Compromised by Aortic Aneurysm Dissection: A 60-year-old white male with history of a thoracic aortic aneurysm, moderate aortic stenosis, a dissection flap in the descending aorta successfully underwent an aortic valve replacement, with an aortic root graft. Two months later, the patient developed persistent refractory hypertension. MRA of the abdominal aorta demonstrated a sizable dissection plane involving much of the abdominal aorta. The origin of the left renal artery appeared to be significantly stenotic with impairment of blood flow to the left kidney. The dissection extended to involve the distal aorta. The combined lumina measured 2.2 cm in diameter. The patient underwent a thoracic and abdominal angiography, as well as selective right and left renal arteriography. Aortic root injection demonstrated no abnormalities of the previously placed graft. An abdominal aortogram showed the right main and accessory renal arteries to be arising from this lumen, and both appeared normal. No other major branches were seen arising from this lumen. A Storq wire was advanced under fluoroscopic guidance through the left femoral access and through the lumen of the pigtail catheter and advanced to the aortic root to ensure continued access to the ascending aorta. The pigtail catheter was then introduced via the left femoral artery retrograde into what appeared to be the false lumen and another abdominal aortogram was performed. This second abdominal angiogram demonstrated that the celiac, superior mesenteric, and the left renal arteries arose from the false lumen. There was significant ostial compromise of left renal artery. A 7Fr short renal guide was used to engage the left renal artery. Selective angiography demonstrated a proximal critical stenosis (90%) of the left renal artery. The lesion in the left renal artery was predilated with a balloon and stented. There was prompt resolution of the patient's hypertension [21].

2 Exclusion of Renal Artery Aneurysm by Covered Stent: A 63-year-old female with escalating hypertension, referred for workup of renovascular hypertension, was incidentally noted to have an aneurysm of the left renal artery. The berry-shaped aneurysm measured 12–13 mm and was located just proximal to the origin of two interlobar arteries supplying the lower pole of a normal-sized kidney. Renal function was normal and no evidence of atherosclerotic renal artery stenosis was noted. An 8Fr left internal mammary artery (LIMA) guide was manipulated to cannulate the renal artery. A hand-crimped stent graft was successfully delivered to the aneurysm site [22].

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Chapter 21
Endovascular Repair of Abdominal Aortic Aneurysm

David Jayakar, Damras Tresukosol, Thach N. Nguyen, Ramon Llobet

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
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♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

The aorta is the major vascular vessel between the left ventricle and the systemic arterial bed. The abdominal aorta starts below the diaphragm and ends dividing into two iliac arteries. This part of the aorta is further divided based on the origin of renal arteries as supra-renal aorta and infra-renal aorta.

The aorta is composed of three distinct layers: the intima, media and adventitia. The intima is a smooth layer that lines the innermost surface of the blood vessel and prevents clot formation. It can be compared to a velvet lining. The media is a thick elastic layer intertwined with collagen and smooth muscle cells with sufficient elastic strength to withstand the pulsatile stress that occurs during the ejection of blood in ventricular systole. With aging, the elastic elements of the aorta degenerate, reducing elasticity and distensibility. This stiffness makes the aorta vulnerable to mechanical trauma and injury over time and the endothelium disintegrates to expose the media.

As a result, the aorta becomes enlarged. This enlargement is called an abdominal aortic aneurysm (AAA). The common causes of aneurysm include atherosclerosis, cystic medial fibrosis, syphilis, mycotic infection, rheumatic aortitis and trauma.

LaPlace's Law: In medical terms states that the wall tension in an aneurysm is proportional to the product of the arterial pressure within the aneurysm and the diameter of the aneurysm. Larger aneurysms and increased blood pressures are associated with higher wall tension.

Predictors of Rupture: Include aneurysm size and the presence of symptoms. At least five population-based studies have demonstrated an increased risk of rupture when an AAA exceeds 5 cm in diameter [1,2]. (Table 21-1).

Ruptured AAA: Rupture of AAA is life threatening. Mortality is over 90% [3] without and 40–50% with open surgical repair [4]. Up

Table 21-1 Relation between AAA diameter and risk of rupture

AAA diameter	Risk of rupture (percent per year)
<4 cm	0%
4.0–4.9 cm	1%
5.0–5.9 cm	11%
>6 cm	25%

to 62% [3] of patients with a ruptured AAA will die before reaching the hospital [5]. Small observational studies suggest a markedly lower short-term mortality after endovascular repair of the AAA (EVAR) (11%) compared with open repair (40%) [6–8].

Open AAA Repair: Conventional surgical treatment employs open abdominal surgery to expose the aneurysm. Once exposed the blood vessels above and below the aneurysm are taped and cross-clamped to allow resection of the aneurysm. The resected segment is then replaced with Dacron graft prosthesis. The risks associated with open repair of AAA are death, bleeding, infection, heart attack, renal failure, emboli of lower extremities and impotence. The morbidity of having an abdominal incision along with the ileus associated with open repair necessitates a hospital stay averaged of 4–7 days. The incidence of impotence increases with pelvic dissection that is associated with aorto-bifemoral grafting.

Endovascular Repair of the AAA (EVAR): The primary goal of this approach is the exclusion of the diseased aortic segment from the circulation without an abdominal incision. If the aneurysmal segment can be successfully excluded from arterial pressure blood flow, it is hoped that the risk of subsequent rupture will be significantly reduced.

Pre-operative Imaging: Patient anatomy guides the choice of devices and determines additional procedures if needed. The areas of consideration include: (1) the diameter of the aneurysm neck, (2) the length of the normal aortic neck, (3) the location of the mesenteric arteries, particularly the celiac axis and the superior mesenteric artery, and (4) the proximal neck angulation. Distally, anatomic concern includes: (1) the size of the ilio-femoral arteries, (2) the presence of absence of calcification, (3) the tortuosity of the ilio-femoral arteries, (4) the presence of aneurysmal change in the ilio-femoral arteries. Successful exclusion of a common iliac aneurysm in conjunction with EVAR may require embolization of the internal iliac artery (to prevent endoleak) and extension of the endograft into the external iliac artery [9]. The selection criteria for EVAR are listed in Table 21-2.

It is critical to have a good CT scan of the abdomen and pelvis with intra-venous contrast using 3 mm × 3 mm cuts. This will give all the information you need for the selection of cases. Angiography may not allow you to measure the diameter of the neck accurately because of inability to detect thrombus within the neck. Of note we do blood pressure in all four extremities and document complete vascular examinations before the procedure.

Contra-indications for EVAR: The primary factors that determine feasibility of endovascular repair are the diameter and length of the proximal neck of the aneurysm, the tortuosity of the aorta, and the anatomy of the iliac arteries. Unfavorable neck anatomy is the primary factor for exclusion from endovascular repair. If the angle between the neck of the aneurysm and the aorta is too great, the

Table 21-2 Selection Criteria for EVAR

1	Adequate iliac/femoral access
2	Infrarenal nonaneurysmal neck length of at least 1 cm at the proximal ends of the aneurysm, and a vessel diameter 10–20% smaller than labeled device diameter
3	Morphology suitable for endovascular repair
	(a) Aortic diameter >5 cm
	(b) A diameter of 4–5 cm and has increased in size by 0.5 cm in the last 6 months
	(c) Twice the diameter of the normal infrarenal aorta
	(d) Infrarenal neck greater than 10 mm from the lowest renal artery
	(e) Proximal aortic neck angulations less than 60°
	(f) Iliac artery diameter sufficient for placing a 19Fr sheath or greater than 7 mm diameter
	(g) Iliac artery angulations less than 120°

graft may be displaced from its intended position with a subsequent leak at the attachment site. Similarly, if the common iliac arteries are too large, the limb of the stent may not be well opposed to the wall of the artery, and a leak at the attachment site will result [10].

Most operators consider the following criteria relative contraindications for EVAR:

1 Proximal neck <15 mm
2 Infrarenal aortic diameter >26 mm
3 External iliac diameter <7 mm or >16 mm
4 Bilateral internal and external iliac aneurysm.

Endograft: There are many types of endovascular devices for AAA. The two important types are straight grafts and bifurcated grafts. If the aneurysm ends >2 cm above the bifurcation of the aorta to iliacs, then a straight graft can be placed. Otherwise a bifurcated graft is used. Bifurcated grafts have many subtypes. Some of them have three pieces to construct; however, the majority is the two piece system. We prefer the single piece system Powerlink unibody bifurcated stent graft manufactured by Endologix, Inc, Irvine, CA. Mimicking the shape of the natural anatomy of the abdominal aorta, the Powerlink unibody bifurcated stent graft is uniquely designed to allow it to be implanted sitting on the anatomical abdominal aortic bifurcation. This is a single piece bifurcated system that can be safely deployed with a small incision on one femoral artery. The device is accessed percutaneously on the contra-lateral femoral artery. The endografts are composed of prosthetic grafts and vascular stents serving as fixation devices. Further, the stents are divided into self-expanding versus balloon expandable types. The advantages of the self-expanding stent are ease of deployment and ability to accommodate the aortic neck enlargement. The disadvantages are risk of neck enlargement and stent migration. The balloon expandable stent helps to deploy the endograft at an exact location which is needed if the proximal neck is short and angulated.

STRATEGIC MAPPING

When planning procedures, the operators should choose the side of access that facilitates the advancement of devices. The iliac arteries should cause the least degree of twists and crookedness of the device within the proximal neck to maintain the longitudinal and rotational ability with the delivery system. What appears to be an initially easy approach may cause the device to bend awkwardly. The operators should plan the full approach with this in mind. The operators should also be cautious about angulation within the target branch and should design grafts using centerline of flow analysis. Axial images, when incorporating branches, provide inadequate data for device design. When considering branches or fenestrations to improve long-term results, devices should be used that will not migrate. Branch stents will not be enough to prevent migration because they will be sacrificed if the aortic device descends. Even 3 mm of movement is enough to crush the stent. The operators should ensure that the proximal seal and fixation systems are within the healthy aorta. Significant overlap of modular systems can also improve long-term results. Overlap can serve as a stress relief mechanism to minimize migration risk [11].

In our hospital system, we perform the EVAR in the cardiac catheterization laboratory (CCL). We find the imaging quality of the CCL systems and the availability of interventional equipment advantageous for the procedure. The operating room staff helps us along with the CCL staff. This process helps if emergencies do rise for surgical intervention. The operating team consists of the surgeon and an interventional cardiologist.

Pre-operative Preparations: Particular attention is paid to assessment of these patients pre-operatively.

1. PO Mucomyst 600 mg BID.
2. Bactroban to both nares 5 days pre-op and continued through the hospital stay.
3. Peridex mouth and body rinse started the day before the procedure.
4. Lactulose 30 cc PO BID for 3 days pre-op.
5. Bowel preparation with 1 g of neomycin and erythromycin base by mouth once daily, for 2 days.
6. Document pulses and Doppler signals in all the lower extremity arteries.
7. If the patient had prior exposure to heparin and has had heparin-induced antibody documented, we would use bivalirudin during the procedure instead of heparin.

ANESTHESIA PREPARATIONS

An arterial line and a central venous line are placed. Palpable or Doppler pulses are noted on all four extremities. The pre-operative creatinine/glomerular filtration rate (GFR) and the hematocrit is noted. The procedure has been done under local, regional and general anesthesia based on the co-morbidities of the patient. The body is prepped from the xiphisternum to the toes. The upper body is kept warm using bear hugger. We were using 100 units of heparin/kg body weight routinely before the start of the procedure. However, due to the increasing awareness of heparin-induced antibody syndrome and spinal artery thrombosis in face of the heparin-induced thrombocytopenia (HIT) syndrome, we modified our anti-coagulant regime to use bivalirudin.

OPERATIVE PROCEDURE

Access: The procedure starts with bilateral groin injections of 5 cc of 0.5% Marcaine mixed with 5 cc of 2% lidocaine. Usually the right common femoral artery is accessed by an open cut-down. The left common femoral artery is cannulated percutaneously using a 9Fr sheath. Open cut-down should be reserved for patients with small or diseased femoral arteries, with pre-existing plaque or heavy calcification. With a graded pigtail catheter, a preliminary abdominal aortogram is obtained. External markers are avoided as they are frequently misguided by the angulations of the abdominal aorta. Using the graded intra-vascular catheter, the correct length of the aneurysm (based on the intra cavitary catheter length) can be calculated. The Powerlink devices have only two diameters (25 mm or 28 mm) of the main body and proximal cuffs suitable for the abdominal aorta [12]. This process makes it easy to determine the size after aortography, and oversizing 10% to 20% of the aneurysm neck is recommended. Since both these sizes fits most of the clinical cases device management by the staff is greatly expedited.

This is then followed by bilateral iliac artery balloon dilatation using a 9 atm balloon. Once the iliacs are dilated and lifted of the retroperitoneum we place a purse string suture (5–0 polypropylene) in the common femoral artery around the 9Fr sheath on the side of the open cut-down. This helps to minimize blood loss during sheath exchange as the purse string can be tightened without any injury to the blood vessel.

Our intention now is to pass a wire from one femoral artery crossover to the other femoral artery. This is done by using an intra-vascular snare from one femoral artery and placed in the abdominal aorta. The wire fed through the contra-lateral limb is captured and delivered to the ipsilateral femoral artery. Over the wire, the crossover catheter (DL-35-90 Dual Lumen Catheter Powerlink system) is advanced from one groin to the contra-lateral groin. The EVAR device is then attached to the crossover catheter and delivered through the cutdown site into the abdominal aorta. The contra-lateral limb is delivered to the contra-lateral groin using the crossover catheter. If there is difficulty in advancing the endograft, then the operator uses the push/pull

technique: pushing the device forward whilst pulling the wire back. Manual compression on the abdominal wall prevents kinking of the endograft and helps to advance the device.

The device is then deployed and the completion angiograms are done. Frequently using a soft compliant balloon, the proximal and distal ends of the graft are dilated to further secure the graft to the wall of the arteries. Ensuring hemostatsis, the heparin is reversed and the wounds are closed in the standard manner.

Intra-operative Imaging: During the procedure, excellent fluoroscopy and angiography are needed to avoid inadvertent occlusion of the mesenteric and renal arteries and to document the proximal extent of the endograft. After the deployment of the endograft, angiogram could be done simultaneously through both sheaths so enough flow can opacify both iliac arteries.

TECHNICAL TIPS

***Inadvertent Renal Artery Exclusion:** Right after deployment of the endograft, inadvertent renal artery exclusion could happen or could be due to migration of the endograft. If the endograft is not completely deployed, either resheathing or manually pulling the graft caudally may uncover the renal artery. Once the endograft is fully deployed, the only option is to pass a wire across the aorto-iliac bifurcation, from one femoral access and externalize it through the contralateral access. Then pull the wire down to uncover the renal artery. A small catheter should be inserted over the wire before pulling the wire down, so it does not damage the end graft [9].

EQUIPMENT

The Powerlink System: The endovascular graft of choice for its ease of delivery, durability, and solid clinical results. There are various sizes and parts available as shown in Figure 21-1 and Table 21-3.

Post-operative Treatment: The patients recover in the recovery room and are frequently sent to a telemetry floor. They get out of bed and walk the same day, they eat the same day. The Foley is removed by midnight and they frequently go home the next day. Pre-operative medications are resumed. Follow-up CT scan of abdomen and pelvis are done at 6-monthly intervals.

RESULTS

Aneurysm Sac: There was a significant decrease between preoperative and 3-year mean AAA diameter (54.6 +/− 7 versus 47.6 +/− 10 mm, respectively; $p < 0.001$) [13]. Aneurysm sac shrinkage was observed in 32 (55.2%) patients. Twenty-five (43.1%) patients had a stable aneurysm sac diameter. Technical success was achieved in all our cases. There was no post-operative death.

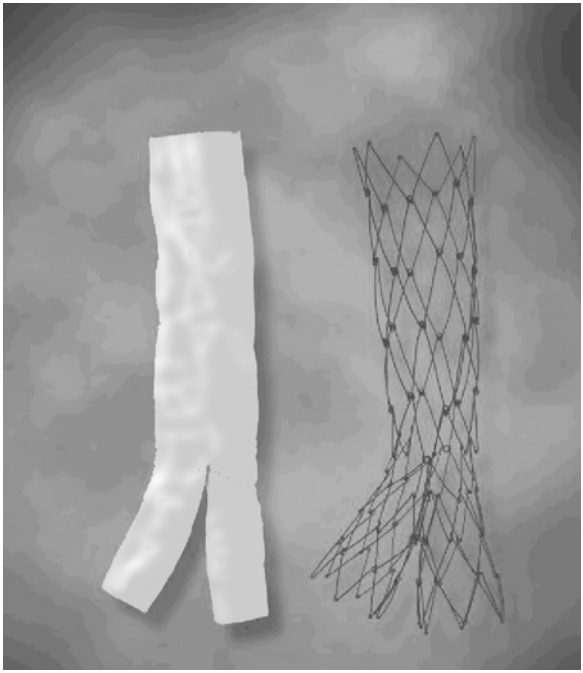

Figure 21-1 The Powerlink system.

Resource Utilization: 70% of our patients left the ICU within 24 hours. 65% were discharged on post operative day 1. The mean operative time was 164 minutes. Operative blood loss had a median of 350cc. In patients who underwent vascular procedures after EVAR we used cell saver technology to minimize blood loss.

Outcomes: The outcomes were very good in the published mid-term results of the multicenter trial of the Powerlink bifurcated system for EVAR, 7 with 97.9% technical success of implantation and only one case of proximal type I endoleak at 1-month CT follow-up. Most endoleaks were type II, and there were no type III and type IV endoleaks [14]. As most recently reported at the Endovascular Congress on Endovascular Interventions in February 2006, there have been no graft material failures or wire fractures through 4-year follow-up [15].

ENDOGRAFT OF DIFFERENT DESIGNS

AneuRx™: The AneuRx™ (Medtronic Inc., Minneapolis, MN) was the first modular device. It consists of a main bifurcated segment and a

Table 21-3 Inventories of the Powerlink System

		Model #	Description	Proximal diameter mm	Distal diameter mm	Total stent length mm	Delivery system outer diameter
Bifurcated devices		28-16-155BL	Infrarenal Bifurcated Stent Graft	28	16	155	21Fr
		28-16-140BL	Infrarenal Bifurcated Stent Graft	28	16	140	21Fr
		28-16-135BL	Infrarenal Bifurcated Stent Graft	28	16	135	21Fr
		28-16-120BL	Infrarenal Bifurcated Stent Graft	28	16	120	21Fr
		25-16-155BL	Infrarenal Bifurcated Stent Graft	25	16	155	21Fr
		25-16-140BL	Infrarenal Bifurcated Stent Graft	25	16	140	21Fr
		25-16-135BL	Infrarenal Bifurcated Stent Graft	25	16	135	21Fr
		25-16-120BL	Infrarenal Bifurcated Stent Graft	25	16	120	21Fr
Infrarenal cuffs		28-28-75L	Infrarenal Proximal Cuff	28	28	75	19Fr
		28-28-55L	Infrarenal Proximal Cuff	28	28	55	19Fr
		25-25-75L	Infrarenal Proximal Cuff	25	25	75	19Fr
		25-25-55L	Infrarenal Proximal Cuff	25	25	55	19Fr

(*Continued*)

Table 21-3 (Continued)

		Model #	Description	Proximal diameter mm	Distal diameter mm	Total stent length mm	Delivery system outer diameter
Lmb extensions		20-20-55L	Limb Extension	20	20	55	17Fr
		16-16-88L	Limb Extension	16	16	88	17Fr
		16-16-55L	Limb Extension	16	16	55	17Fr
Ancillary Products		DL-35-90	Dual Lumen Catheter (90 cm total length)				
		HLS-1012.5	12.5Fr Tear-Away Sheath (packaged and sold in boxes of 5 each)				

contralateral iliac leg. The main bifurcated segment can be deployed through a 21Fr catheter and the contralateral leg can be delivered through a 16Fr catheter. The device is available in aortic diameter of 20–28 mm and iliac diameter of 12–16 mm. Because of the concern over the relatively high rate of endoleaks, surveillance imaging should be performed at 1, 6, and 12 months after implantation and yearly thereafter [11]. The new generation endograft is the Talent which gave good report in European trials.

Excluder™: The Excluder™ (W.L. Gore and Associates Inc., Flagstaff, AZ) is constructed of a durable ePTFE bifurcated graft with an outer self-expanding nitinol support structure to combine both device flexibility and material durability. The function of the endoprosthesis is to internally reline the abdominal aorta, including the bifurcation, and exclude the aneurysm from blood circulation. The device is wrapped around the delivery system and tied with dental-floss-like thread. With the pull of a string the device self-expands to the diameter of the aorta and iliac arteries, sealing off the aneurysm. The device is delivered through an 18Fr access sheath and the contralateral extensions are introduced via a 12Fr sheath (the smallest delivery system available in the US market) [11].

Zenith™: Zenith™ (Cook, Inc., Bloomington, IN) is a supported, bifurcated, self-expandable stent graft with multiple stainless steel Z stents placed inside the graft. It attaches to the vessel wall via barbs. It is unique in having proximal stent for suprarenal fixation to prevent migration and enhance graft-to-vessel sealing. The delivery system utilizes an 18Fr or 22Fr introducer sheath for the main body and a 14Fr or 16Fr introducer sheath for the iliac limbs. The graft is available with proximal neck diameters ranging from 22–32 mm and the iliac extensions come in diameters of 8–24 mm [11].

COMPLICATIONS

Several morphological and structural changes of the endograft leading to device failure have been reported. These changes include: suture breaks between the stent and the graft, fracture of the hooks used to anchor the proximal end of the endograft to the aortic wall, circular and longitudinal stent wire separation, separation of the connection between wire loops, graft fatigue, device migration, component separation, and endograft or vessel thrombosis. Most of these failures manifest with an endoleak. It can be avoided by selecting the common femoral artery or external iliac artery for the entry site of the device. If the vessel is diseased, it may be prudent to have a 7 mm graft cuff anastomosed end to side to minimize the trauma due to repeated catheterization of the device.

Endoleak: Endoleak is defined as a clinical condition associated with endovascular grafts where there is persistence of blood flow outside the lumen of the endovascular graft. This can be diagnosed clinically

by presence of expansile pulsation in the aneurysm or most usually in follow up CT scans. They are classified in three types.

In type 1 blood escapes around the cuff of the stent and into the aneurysm.

In type 2 blood escapes from the branches in retrograde fashion into the aneurysm sac. This group is further classified in type 2A where there is no outflow and type 2B where there is both inflow and outflow.

In Type 3 there is leak through the graft due to tears, disconnection or damage to the graft material.

Meta-analysis: Of the 1189 patients included in 23 studies, 1118 had successful graft placement. Of these, 270 (24%) had an endoleak; 17% were early endoleaks and 7% were delayed. The overall rupture rate was 2%. Most endoleaks arose from the distal stent attachment (36%). More endoleaks occurred with tube grafts (35%) than with bifurcated grafts (18%) or aorto unilateral devices (20%) [16].

The concern over endoleaks is that the systemic arterial pressure is maintained within the aneurysmal sac which may lead to progression and continued expansion of aneurysm. This increases the risk for rupture and is considered to be a failure of treatment. Properly recognized and managed endoleaks rarely are of concern as there are various minor surgical and intervention options available to correct the problem.

For patients with type 2 endoleaks, the primary treatment strategy is observation. Other strategies include coil embolization, thrombin injection, glue injection, laparoscopic inferior mesenteric artery ligation, minilap with lumbar ligation, and surgical conversion. These strategies, however, may not be more effective than observation. Type 3 endoleaks carry a significant risk of rupture as the full force of the aortic flow is brought against the aneurysm sac. Patients with type 3 endoleaks should be treated with additional modular units, a new endograft or open surgical conversion. For type 1 endoleaks, endovascular repair appears to reduce the risk of rupture of large aneurysms despite the persistence of a type 1 endoleak. In a 400-patient cohort treated at Stanford, 5.3% had persisting type 1 endoleaks for longer than 10 months. None of the patients experienced rupture or aneurysm-related death. These endoleaks may indicate inadequate stent–graft fixation. All patients with endoleaks should be monitored, regardless of endoleak status [17].

Graft Migration: The stability of most of the EVAR grafts available is based on the ability of the hooks on the proximal part of the graft to cling to the aortic wall. Due to irregularities in the wall of the aorta and calcifications the ability to secure the graft proximally can be inadequate. This is further exacerbated by the normal forward propulsion of blood flow and causes the graft to migrate distally. The Powerlink (Endologix) system uses a novel approach to minimize this problem. Apart from proximal fixation, the bifurcation of the graft is designed to seat at the normal aortic bifurcation. This gives additional fixation and prevents graft migration. In our experience this has helped graft fixation without endoleak in many patients with abnormal aortic anatomy and angulation (Figure 21-2).

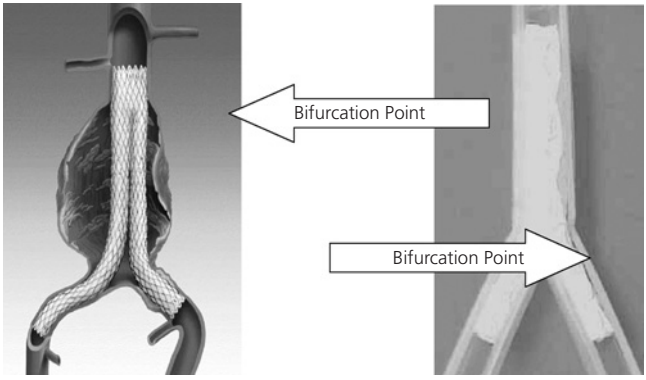

Figure 21-2 The Powerlink Bifurcation system.

In a single-center experience with 297 cases of Powerlink implantation approximately one-third of them were implanted sitting on the aortic bifurcation (anatomic fixation). The remaining were implanted using infrarenal or transrenal fixation with the bifurcation in the sac (non anatomic fixation). This may be another reason for the 4.4% type 1 endoleak. They had seven cases of distal migration in total, but all of the devices were not implanted sitting on the aortic bifurcation. There were no cases of distal migration observed in the patients receiving anatomic fixation [17].

Endograft Limb Occlusion: Occlusion of the limb of the endograft can happen, usually 2 months after implantation. The patient presents with atypical leg discomfort, new onset claudication or acute limb ischemia. The treatment includes open or percutaneous thrombetomy

in order to reestablish the flow to the lower extremities. Then the cause of the occlusion should be investigated and addressed such as limb kink, stenosis, distal dissection etc. The predisposing factors of limb occlusion are smaller limb diameter, limb stenosis, unsupported endografts, and the extension of the endograft into the external iliac arteries [18].

Internal Iliac Artery Occlusion: The internal iliac artery could be embolized as a stage procedure or it is excluded during procedure. The complications are erectile dysfunction, hip or buttock claudication. Other complications include scrotal skin sloughing, sacral decubitus ulcer, and intestinal ischemia. These complications happen more frequently if the profunda femoris artery is diseased, because the profunda is a major source of collaterals to these areas. So the decision of occlusion of the internal iliac artery should be evaluated well for risks and benefits [9].

EMERGENCY EVAR FOR RUPTURED AAA

As EVAR becomes an established modality for the treatment of AAA, EVAR was tried on patients with ruptured AAA (rAAA) because of its low morbidity and mortality. The only difference in the technique is the insertion of a 33 mm or 40 mm aortic occlusion balloon positioned in the supraceliac abdominal aorta. The balloon is positioned there uninflated. It will be inflated if there is conversion to surgery. After that, the deployment of EVAR is conducted in the usual fashion. The other management difference is the induction of hypotension in order to minimize ongoing hemorrhage. Because many patients were so hemodynamically unstable so a CT scan could not be done, the selection of the endograft was based on intra-operative angiographic findings. The goal of emergent EVAR is to exclude the rAAA at presentation and get the patient through the initial high risk episode, even at the cost of a second elective procedure, or conversion to open surgery once the patient is hemodynamically stable [19].

FOLLOW-UP

Studies performed in patients who have undergone AAA treatments has shown that up to 30% of the patient can have progressive dilatation of untreated vascular segments proximal and distal to the graft [18]. Thus, the potential exists for a delayed failure unless the endoprosthesis has the capability to continually expand. We had one such instance which was recognized post-operatively on routine follow up and fixed with a proximal cuff.

In the prospective comparative trials on FDA-approved devices, perioperative mortality is similar for both endovascular repair and surgical groups and falls within the range of 0–2.7%. Perioperative major morbidity seems to occur with greater frequency in those undergoing open surgical repair and statistically significant reductions in pulmonary and gastrointestinal adverse events. Of note in most studies the patients resumed to normal eating the same day of surgery. These are confirmed by large studies with the Powerlink EVAR device [20].

Persistent endoleaks are observed in approximately 15–30% of endovascular repair-treated subjects and the potential for progressive dilatation of the aorta raises concern about the long-term efficacy of endovascular repair. As expected, older patients and patients with aspirin (ASA) class III or IV risk have a higher risk of complications with both surgical and EVAR but EVAR had significantly lower complications.

It is still debated as to the superiority of EVAR over open repair [21]. Preliminary studies and meta-analysis have shown that EVAR is superior to open repair [22]. In a prospective study, there is a previously underestimated incidence of sub-clinical myocardial damage associated with surgery for infrarenal AAA, which is lower after endovascular than open repair [23]. Pearson *et al.* found that an attenuated glucocorticoid surge characterizes the reduced stress response experienced by patients undergoing EVAR compared to open repair and suggested

that a reduction in the occurrence of Systemic Inflammatory Response Syndrome is a feature of a more favorable postoperative course after an endovascular approach [24]. These factors strongly support the increasing use of EVAR in patients presenting with suitable anatomy.

INVESTIGATIONAL MANAGEMENT OF ENDOLEAK

CASE REPORT: A 65-year-old man presented with a symptomatic, expanding abdominal infrarenal aortic aneurysm. The patient underwent surgery with a polyester graft (Sulzer Vascutek, Switzerland) implanted with an oblique proximal anastomosis just below the renal arteries and a distal anastomosis at the level of the aortic bifurcation. Two months later he returned with back pain, anemia, and collapse. CT angiography of the abdomen showed a high flow proximal para-anastomotic leak with active extravasation into the aneurysm sac which had expanded to a diameter of 9 cm. The communication was notable for the presence of a narrow neck bridging two larger lumens and was situated immediately adjacent to the origin of the left renal artery and in close proximity to the superior mesenteric artery. Consultation with interventional radiology and interventional cardiology led to an attempt at percutaneous closure utilizing an atrial septal defect occluder.

The patient was taken to the catheterization laboratory and under local anesthesia the aorta was cannulated utilizing a right femoral percutaneous approach. The leak was located utilizing a Simmons 2 catheter (Cook; Bloomington, IN), and hand injections of contrast. The communication appeared to have a large neck which tapered down to ~4 mm in diameter prior to entry into a large aneurysmal sac.

The leak was cannulated utilizing a 6Fr right Judkins catheter and a hydrophyllic wire. The catheter was advanced into the aneurysm and the hydrophyllic wire exchanged for a more supportive wire. Through the guide was advanced an oversized 8 mm waist diameter Amplatzer ASD Occluder (AGA Medical, Golden Valley, MN). Contrast injection through another catheter placed in the aorta from the femoral approach confirmed satisfactory placement. The device was deployed with the distal disc within the aneurysm and the proximal disc in the aortic neck, Aortography showed no visible contrast entering the aneurysm [10].

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Chapter 22
Iliac Artery Stenosis

Gianluca Rigatelli, Paolo Cardaioli, Rosli Mohd Ali

GENERAL OVERVIEW

Aorto-iliac artery disease is a common presentation of atherosclerotic cardiovascular disease. Because of the high correlation between coronary artery disease and aorto-iliac atherosclerosis, a global work-up including coronary artery disease screening should be performed whenever possible [1]. With excellent results of angioplasty and stenting of the aorto-iliac vessels, endovascular approach has become the first line therapy in the majority of these patients.

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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NON-INVASIVE EVALUATION

The new guidelines of peripheral artery disease (PAD) management identified asymptomatic and symptomatic patients for both of whom measurement of ankle-brachial index (ABI) is of paramount importance. An ABI $>$ 0.91 or $>$ 1.30 suggests more sensitive and sophisticated investigations, such as treadmill exercise test with and without ABI measurement and 6-minute walk test, Doppler ultrasound and for candidates to revascularization procedure, MRI, CT and invasive angiography [2].

Treadmill Exercise Test With and Without ABI: Exercise testing may be extremely useful (a) in establishing the diagnosis of lower extremity PAD when resting measures of the ABI are normal, (b) to objectively document the magnitude of symptom limitation in patients with lower extremity PAD and claudication, (c) to objectively measure the functional improvement obtained in response to claudication interventions, (d) to differentiate claudication from pseudo-claudication in individuals with exertional leg symptoms, and (e) to provide objective data that can demonstrate the safety of exercise and to individualize exercise prescriptions in patients with claudication before initiation of a formal program of exercise training. Dedicated personnel and equipment are needed.

Ultrasound: Doppler ultrasound is useful to provide an accurate assessment of lower extremity PAD location and severity, to follow lower extremity PAD progression, and to provide quantitative follow-up after revascularization procedures. Unfortunately in the aorto-iliac segment its efficacy is limited by the poor acoustic window of obese patients and for the deep localization of the vessel in the abdomen.

Computed Tomography Angiography: Computed tomographic angiography requires intravenous injection of iodinated contrast, which opacifies the arteries. The angiographic image is constructed from multiple cross-sectional images and then presented as a maximum-intensity projection, similar to the appearance of standard arteriography. The image can be rotated three-dimensionally in space to view any oblique projection. This is particularly useful in the aortoiliac segment, where Doppler ultrasound often is suboptimal because of technical difficulty.

Magnetic Resonance Angiography: MRA of the extremities can be used to diagnose the anatomic location and degree of stenosis of PAD. Magnetic resonance angiography evaluation is based on imaging the arteries, similar to standard arteriography. Assessment of the accuracy of MRA depends on the MRA technique used and the standard against which it is compared. MRA techniques continue to evolve and improve. The techniques include 2D time of flight, 3D imaging, and contrast enhancement with gadolinium, subtraction, cardiac gating, and bolus chase. These techniques may be used in combination, because each has its advantages and disadvantages. The technique is particularly useful in elderly patients with poor renal function but is limited by presence of metallic prosthesis and cardiac implants [2].

INVASIVE EVALUATION

Diagnostic Aortoiliac Angiography: Digital subtraction angiography (DSA) is the "gold standard" for defining both normal vascular anatomy and vascular pathology. It remains the most readily available and widely used imaging technique. Because of possible distal embolization or the effect of below-knee distal run-off on short and long term iliac stent patency, a pre- and post-procedural lower extremities angiogram should be done and compared [3].

TECHNIQUE: The usual technique includes the use of 4 or 5Fr Pigtail catheter placed above the renal artery if flat panel radiological equipment is available or infrarenal if cardiac equipment is used [3]. Injection of 25–30 cc of contrast at 10–12 cc rate is usually sufficient with digital subtraction technique to depict the aorto-iliac bifurcation and the external iliac arteries. Ispilateral 20–30° oblique view can be selected in case of eccentric lesions. Pressure gradient measurement should be obtained with manual pull-back to detect any significant stenosis: >30 mmHg.

Place a side-hole catheter at level of L1–L2. Use the smallest contrast volume possible especially when aortoiliac angiography is performed in conjunction with cardiac angiography. Use 20° controlateral angulation with 20° caudal angulation injection when a detailed relation between the internal/external iliac arteries is needed (Figure 22-1).

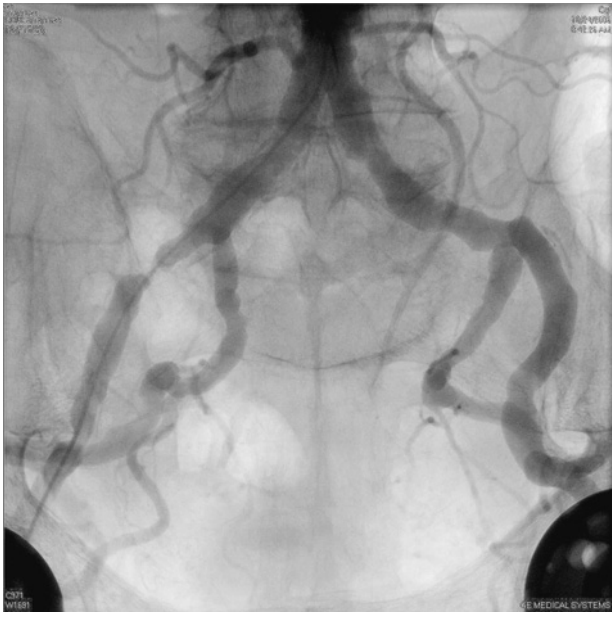

Figure 22-1 Aortoiliac angiography during coronary angiography in a 70-year-old patient with multivessel coronary artery disease.

Intravascular Ultrasound: Intravascular ultrasound may be needed intraoperatively when the severity of lesion and length is not easily measured or when the ostial disease can not be fully excluded: 6Fr peripheral intravascular catheter and sometimes in case of severely ectatic iliac arteries, 9Fr intracardiac ultrasound probe can be used [4,5].

INDICATIONS

Following the new guidelines [2] some clear indications for iliac artery occlusive disease endovascular management should be observed in case of claudication or chronic ischemia with an increasing degree of evidence from C to A levels (Tables 22-1 and 22-2).

STENTING

Access: Access is usually obtained from the ispilateral femoral artery [6,7]. Sometimes the controlateral approach is preferable especially in case of total occlusion without a nipple or when the ipsilateral femoral artery is diseased itself. Use whenever possible the ipsilateral approach.

Table 22-1 Indications for Iliac Artery Interventions

1	Endovascular procedures are indicated for individuals with a vocational or lifestyle-limiting disability due to intermittent claudication when clinical features suggest a reasonable likelihood of symptomatic improvement with endovascular intervention and (a) there has been an inadequate response to exercise or pharmacological therapy and/or (b) there is a very favorable risk-benefit ratio (e.g. focal aortoiliac occlusive disease) *(Level of Evidence: A)*
2	Endovascular intervention is recommended as the preferred revascularization technique for TransAtlantic Inter-Society Consensus (TASC) type A iliac and femoropopliteal arterial lesions *(Level of Evidence: B)*
3	Translesional pressure gradients (with and without vasodilation) should be obtained to evaluate the significance of angiographic iliac arterial stenoses of 50–75% diameter before intervention *(Level of Evidence: C)*
4	Provisional stent placement is indicated for use in the iliac arteries as salvage therapy for a suboptimal or failed result from balloon dilation (e.g. persistent translesional gradient, residual diameter stenosis greater than 50%, or flow-limiting dissection) *(Level of Evidence: B)*
5	Stenting is effective as primary therapy for common iliac artery stenosis and occlusions *(Level of Evidence: B)*
6	Stenting is effective as primary therapy in external iliac artery stenoses and occlusions *(Level of Evidence: C)*

Table 22-2 Current Aortoiliac Lesions Classification

Lesion type	Lesion characteristics
TASC type A iliac lesions	Single stenosis less than 3 cm of the CIA or EIA (unilateral/bilateral)
TASC type B iliac lesions	Single stenosis 3–10 cm in length, not extending into the CFA Total of 2 stenoses less than 5 cm long in the CIA and/or EIA and not extending into the CFA Unilateral CIA occlusion
TASC type C iliac lesions	Bilateral 5–10 cm long stenosis of the CIA and/or EIA, not extending into the CFA Unilateral EIA occlusion not extending into the CFA Unilateral EIA stenosis extending into the CFA Bilateral CIA occlusion
TASC type D iliac lesions	Diffuse, multiple unilateral stenoses involving the CIA, EIA, and CFA (usually more than 10 cm long) Unilateral occlusion involving both the CIA and EIA Bilateral EIA occlusions Diffuse disease involving the aorta and both iliac arteries Iliac stenoses in a patient with an abdominal aortic aneurysm or other lesion requiring aortic or iliac surgery

TASC TransAtlantic Inter-Society Consensus; CIA Common Iliac Artery; EIA External Iliac Artery; CFA Common Femoral Artery.

Select a 6 or 7F sheath (at least 23 cm long). This enables sufficient contrast injection and optimal visualization of the lesion by the sheath itself without using a controlateral pigtail catheter (Figures 22-2 and 22-3).

Wires: Wire selection is a quite important step: A 0.35″ soft tip preformable wire may be preferable in most cases. Hydrophilic .035″ wires should be used in case of total occlusion. Sometimes when dealing with very tight stenosis a coronary high-support hydrophilic wire 0.014″ may be used to facilitate predilation with small coronary balloons. Stiffer wires such as the .035″ Supracor or Amplatz wires may be limited to cases in which the balloon passage is difficult. In such cases it is useful to change the wire for a hydrophilic exchange catheter such as the 4Fr Glidecath (Terumo Co, Tokyo Japan).

When using a hydrophilic wire, careful attention to the tip is needed. Follow the advancement of the tip under fluoroscopy to avoid access to aberrant renal artery with potentially severe renal damage. The wire tip is curved as a large C rather than a J. Usually it is sufficient to cross the lesion without vessel damage. Place the wire into the thoracic descending aorta: there are fewer branches for erroneous entry.

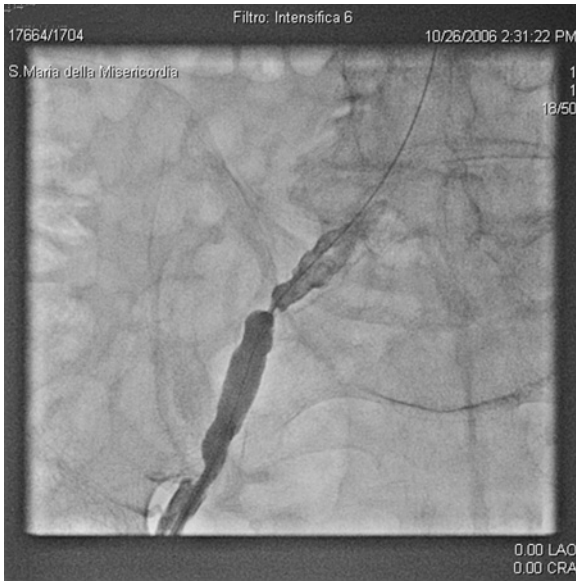

Figure 22-2 A 23 cm 7F sheath has been placed through the femoral artery into the esternal iliac artery: a pre-formable .035″ soft tip wire has been advanced through a external iliac artery tight stenosis.

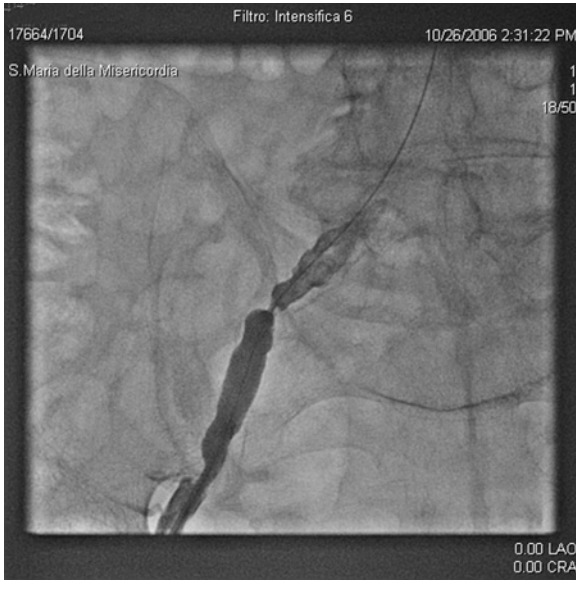

Figure 22-3 A small contrast injection through the 23 cm 7F sheath visualized the tight stenosis: thus, a road map technique allow for correct placement of the stent.

Balloons: Usually primary stenting is the technique of choice for the majority of lesions but sometimes predilation is needed. A low profile 0.014″ monorail coronary balloon 2.5–3.5 × 20–30 mm inflated to nominal pressure would help to advance the stent in case of very tight stenosis. In all the other cases, a normal peripheral 0.035″ balloon 4–6 mm × 20 or 30 mm depending on the lesion length can be used.

Use manometer to inflate balloons especially when facing with calcified stenosis. Inflate slowly 1 atm for 3–4 seconds and watch as the lesion is modified. Stop inflation as the patient complains of pain.

Stent: Stent implantation is probably the essence of modern iliac artery interventional technique. Proper stent selection is mandatory to achieve good immediate and long-term results. Focal calcified stenosis should be managed with high radial force stent: 8–10 mm × 20–30 mm stainless-steel balloon-expandable are the best option as well as for focal and ostial stenosis (Figure 22-4). The balloon expandable stents allow precise implantation thanks to their excellent radiopacity. They can be overexpanded with only minimal shortening. Long diffuse soft stenosis is better treated with 8–10 mm × 40–80 mm self-expandable nitinol stents: its open–close design is be preferred because of low risk of distal embolization and optimal conformation to vessel anatomy (Figure 22-5).

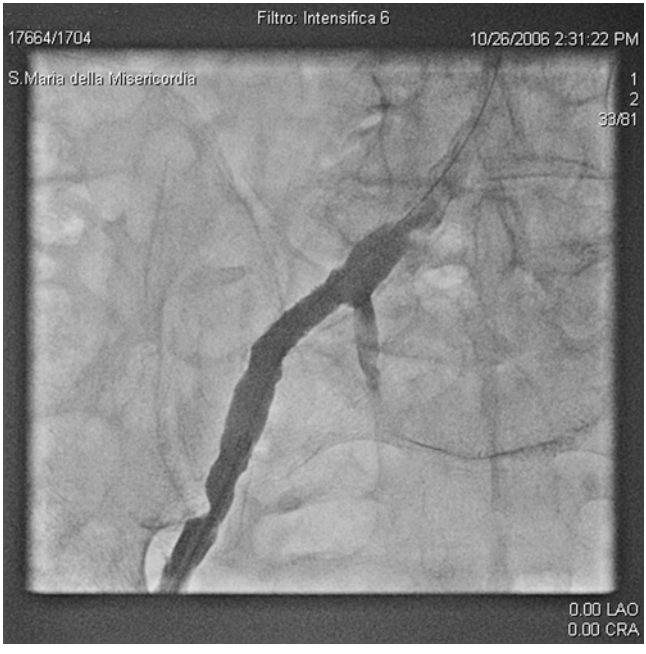

Figure 22-4 A 8 × 30 balloon-expandable stainless-steel stent has been correctly placed with a good angiographic result.

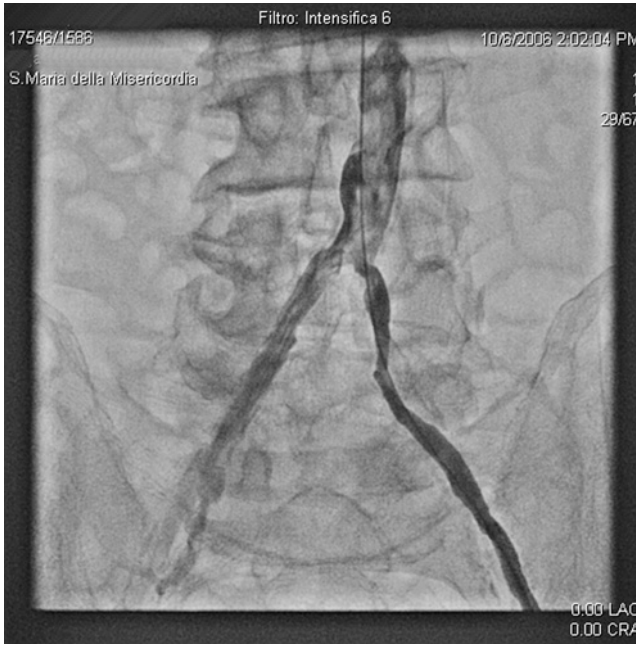

Figure 22-5 An aortogram during coronary angioplasty showed a long , diffused stenosis of the left common iliac artery in a patients with multivessel coronary artery disease and 50 m claudication. The pressure gradient through the lesion was about 35 mmHg.

TECHNICAL TIPS

****Perfect Stenting:** Use roadmapping technique when possible if balloon-expandable stenting is planned. Inject 5–8 cc of contrast if the iliac ostium is to be covered. Keep the balloon-expandable stent 2–3 mm beyond the iliac ostium in the abdominal aorta to assure full coverage of the ostium. Deploy the stent very slowly: if the stent tends to advance, gently withdraw the stent, check position and continue to slowly deploy (Figures 22-6–8). Alternatively, advance the sheath over its dilator through the lesion within the distal abdominal aorta, take a picture with the road-map technique, withdraw the sheath and deploy the stent. Avoid kissing stent with self expandable stents: it is difficult, cumbersome, and often the results are not satisfying [8]. When performing kissing stent technique, deploy the stents simultaneously with two indeflators at the same pressure (8–9 atm for most stents). Deflate the balloons then advance both balloons beyond the distal end of the stent in the abdominal aorta and overdilate with high pressure (10–12 atm) to flare the ostial segments of the stents. Don't

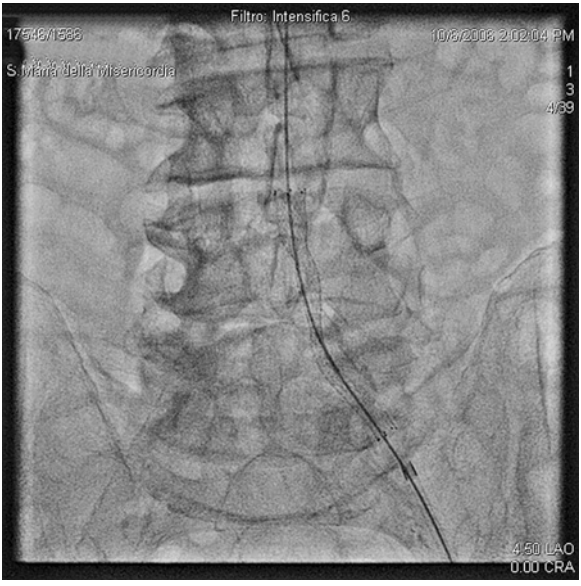

Figure 22-6 A self-expandable stent was deployed to cover the ostium and the entire lesion through a 7F 23 cm sheath.

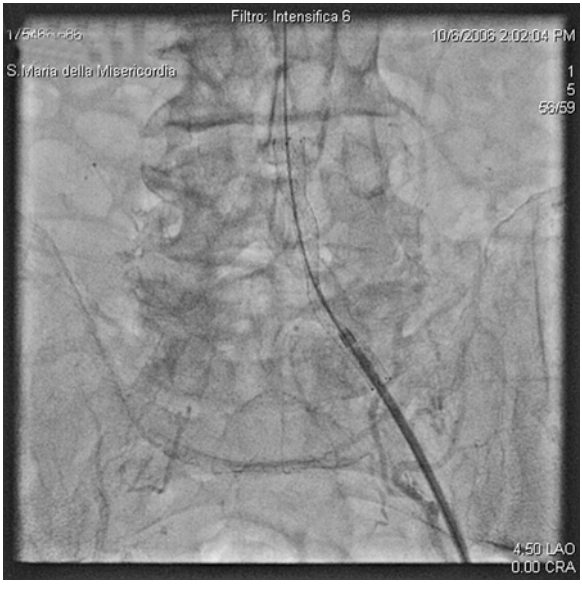

Figure 22-7 The sheath is gently advanced over its dilator through the stent to allow the balloon safely advances into the stent.

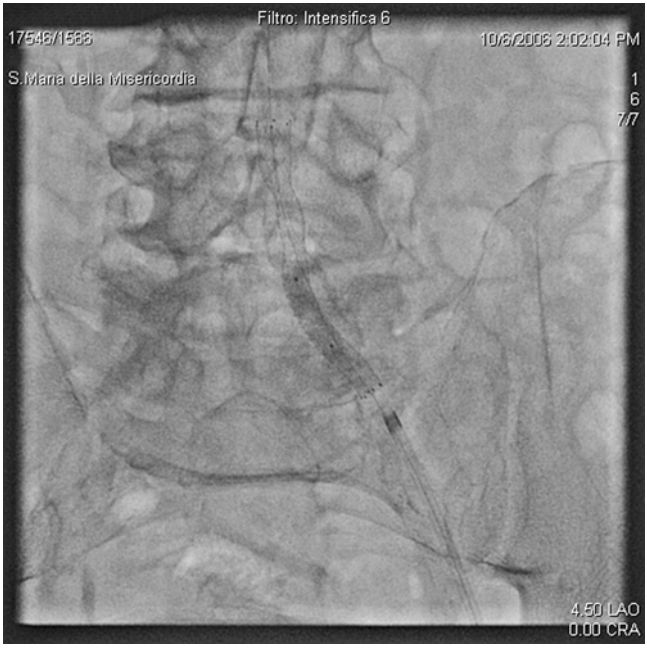

Figure 22-8 The balloon is slowly inflated at nominal pressure until the patient feels pain.

worry if 4–6 mm of the proximal end of the stent lies in the aorta: usually it is uneventful and some operators even prefer it (Figure 22-9) [9].

Check final results carefully with an aortogram from a diagnostic pigtail catheter (with digital subtraction) technique to assess the results and any aortic damage.

Brachial Access: In rare cases the procedure cannot be accomplished through the femoral approach: new alternative access sites have been proposed for a wide spectrum of endovascular interventions, such as the radial approach for renal and carotid interventions and axillary approach for subclavian and aortic aneurysmal repair. Unfortunately, the axillary route often requires surgical cutdown and the diameter of the radial artery should be carefully measured before the procedure in order to minimize arterial complications when large sheaths are to be used. Recently, a case of radial access for ipsilateral iliac stenting has been reported [10,11]. A brachial access may have the advantage of being able to accommodate larger sheaths and is more likely to be effective in reaching the aorto-iliac segment in most patients. The disadvantage of being required to use stiff wires, especially in the case of a calcified and elongated aorta, may be corrected by using a protective 4Fr catheter and replacing the catheter with an

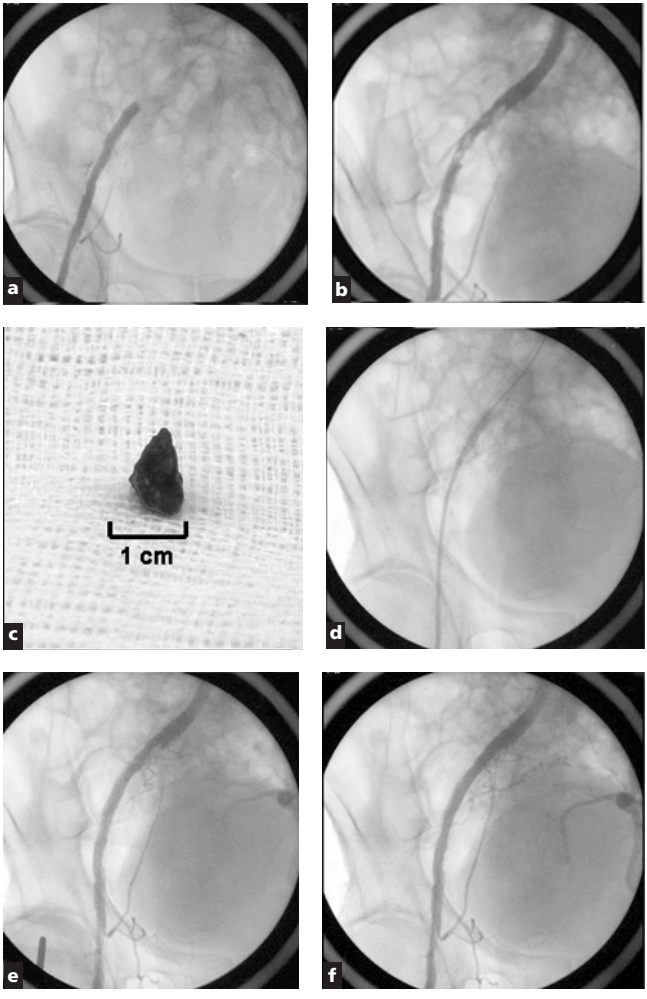

Figure 22-9 Subacute occlusion of the right common iliac artery after prolonged counterpulsation in patients underwent coronary surgery (a). Result of 12 h infusion of Tirofiban (b). Manual aspiration (c) and balloon inflation (d). The residual dissection (e) has been covered with a balloon-expandable stent (f).

at least 85 cm long sheath. This technique is safer and offers more support than using two different guides, reduces the stress on the arterial vessel at the subclavian site and enables a stiff balloon or stent catheter to be advanced even through a very elongated and calcified aorta without the risk of dislodging the stent.

The technique can be performed by a guide technique with Judkins Right or Multipurpose 6Fr guide as usually done for angioplasty in other arterial segments or in case of very tortuous aorta or very calcified lesion with 90 cm-long sheath (Figures 22-10–12). In cases of kissing stenting a 6Fr Brite tip sheath (Cordis, Jonhson & Jonhson Medical, Miami Lakes, FL, USA) is inserted into both brachial arteries and a standard 4Fr Judkins Right diagnostic catheter inserted over a 260 cm 0.038" Terumo Stiff wire (Terumo co. Tokyo, Japan) through both the sheaths. The catheter is navigated up to the aortic bifurcation and after selecting the common iliac artery ostia, the wires are navigated through the lesion and moved up to the ipsilateral superficial femoral arteries. The catheters should then be advanced over the wires beyond the occlusions and the Terumo wires were replaced by two 0.038" 260 cm Supracor wires (Boston Scientific, San Jose, CA, USA). In order to facilitate the forward movement of the stents without the risk of dislodging them, two 6Fr 90 cm Shuttle Flexor introducer long sheaths (Cook Co, Bloomington, IN) can be advanced over the Supracor wires until they reach the common iliac artery ostia. A roadmap technique is used to check the ostia position in order to properly deploy the selected stent (Figures 22-13–15).

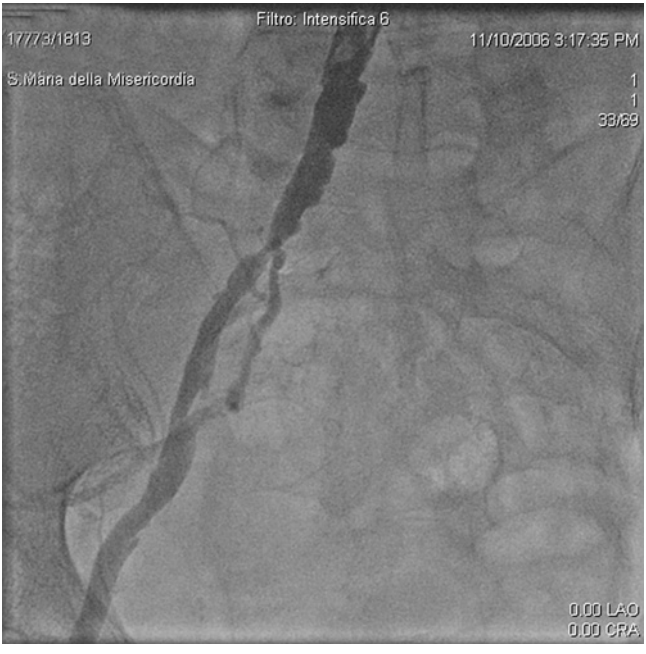

Figure 22-10 Iliac angiography through a diagnostic catheter advanced from the brachial artery.

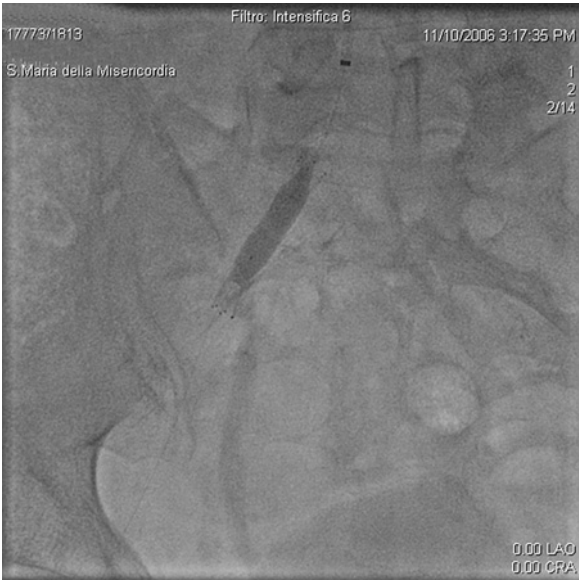

Figure 22-11 After changing the catheter with a long 6F 90 cm sheath, a soft tip 260 cm wire is advanced through the lesion and the stent is correctly deployed under road-map technique.

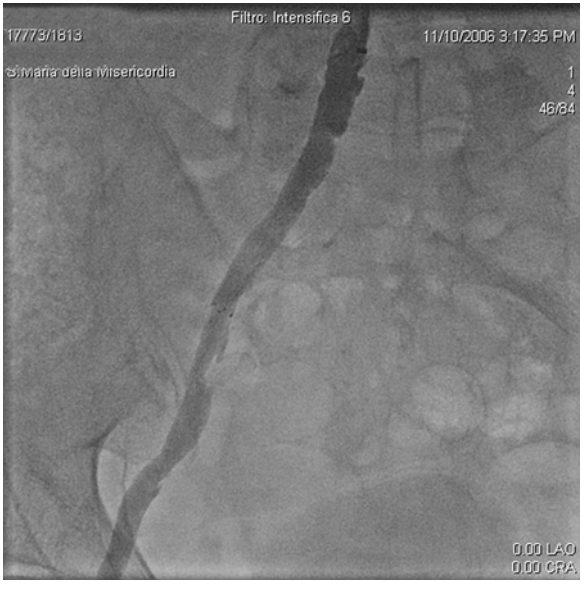

Figure 22-12 Final result by direct injection through the 6F 90 cm sheath.

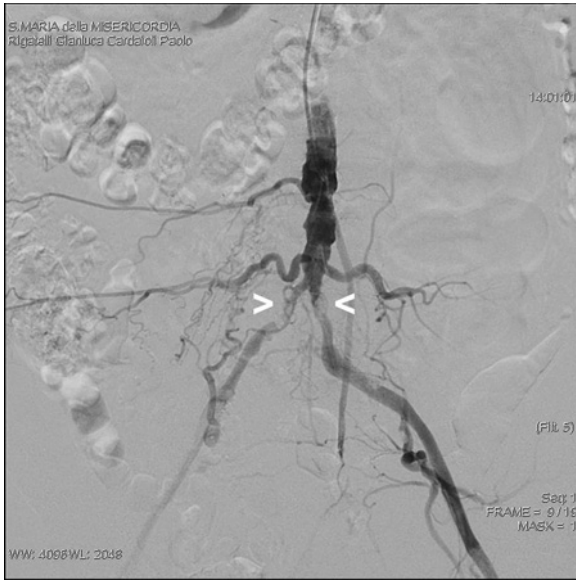

Figure 22-13 Aortoiliac angiogram in 20° left anterior oblique projection showing severe aorto-iliac occlusive disease (arrows).

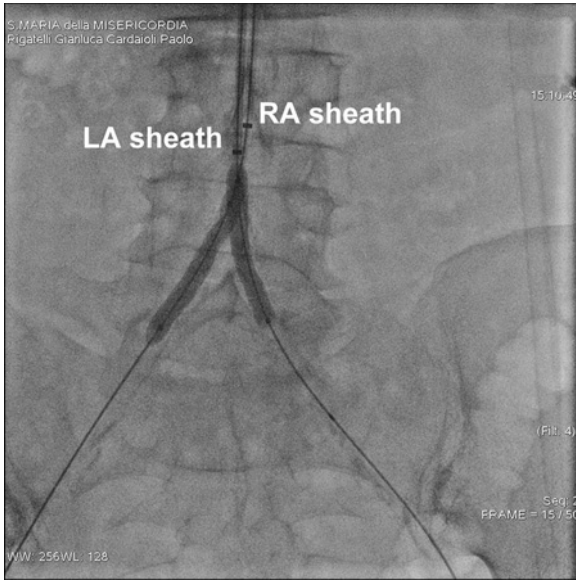

Figure 22-14 Kissing stenting with two 6.0 × 39 mm balloon-expandable stent through the long 6F brachial sheaths.

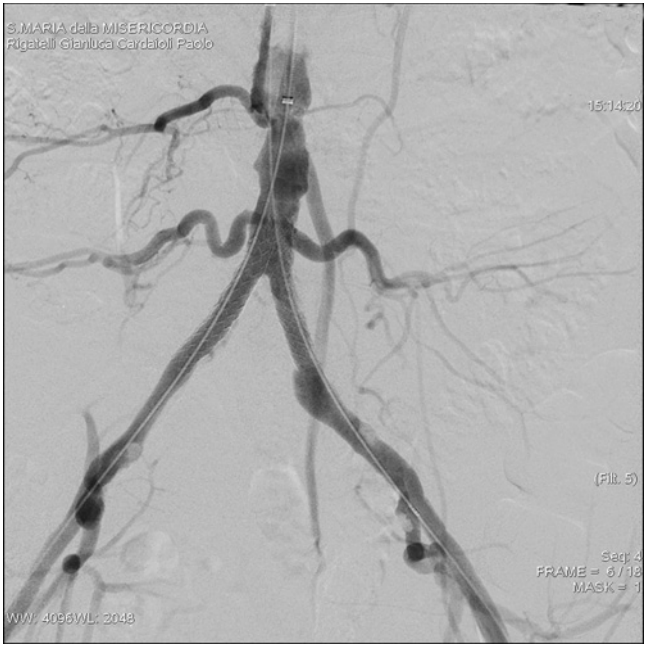

Figure 22-15 Final angiographic results after kissing stenting.

COMPLICATIONS

Although rare, complications may occur, some of which are potentially lethal [12,13]. Complications may include distal embolization, stent migration, iliac artery dissection, iliac artery rupture.

Perforations: Covered stent availability in the cardiac catheterization laboratories (CCL) is a prerequisite for iliac artery stenting as well as large occlusion balloons that may often keep the patient stable until reaching the operating room. Surveillance of implanted stent should be warranted by Doppler ultrasound and in particularly by CT and MR angiography. Poor infrainguinal runoff is the main risk factor for decreased primary patency stenting to treat TASC type B and type C iliac lesions. The presence of poor runoff, external iliac artery disease, and female gender are independent predictors of poor outcome after iliac stenting, and therefore these risk factors should determine the need for surgical reconstruction: careful patient selection is to be recommended.

Acute or Subacute Occlusion: Despite its rare occurrence, acute or subacute occlusion of the common or external iliac artery may occur, especially due to iliac artery dissection during diagnostic femoral procedures or caused by long-term aortic counter pulsation [9]. In case of acute occlusion, local thrombolytic therapy should be used whenever

possible, perfused through a 5–6F infusion catheter from the retrograde controlateral or brachial access. In case of subacute occlusion, declotting with manual or rheolytic thrombectomy could be successfully performed. Protection with distal filter should be used whenever possible by placing a large 6–8 mm filter from the controlateral approach in the femoral artery. Infusion of antiplatelet agents such as glycoprotein 2b3a antiplatelets agents may be helpful and suggested if there is significant residual thrombotic burden after mechanical thrombectomy.

TECHNICAL TIPS

****Damage Control:** Use the brachial access to place an infusion catheter whenever possible to minimize the risk of bleeding. In case of subacute thrombosis, first perform a manual aspiration thrombectomy with a 6Fr large lumen guide and then use any thrombectomy catheter available: this strategy may help to obtain maximal declotting (Figure 22-9). When using rheolytic catheter (usually 6Fr peripheral device), be sure not to aspirate more than 200–250 cc each time because hemolysis can occur.

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Chapter 23
Infrainguignal and Infragenicular Interventions

Prakash Makam, Thach N. Nguyen

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
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GENERAL OVERVIEW

Lower-extremity peripheral artery disease (PAD) is very common, presented as claudication and can progress to critical limb ischemia (CLI) if left untreated. The goals of endovascular intervention in PAD patients with severe ischemic changes in the lower extremity include an improvement in symptoms of rest pain and restoration of blood flow to the foot necessary for the healing of ischemic ulcers or ischemic gangrene [1].

Anatomic–Clinical Correlation: At the level of the inguinal ligament, the external iliac artery becomes the common femoral artery (CFA) just distal to the lateral circumflex and inferior epigastric arteries. At the lower edge of the femoral head, the CFA subsequently divides into the deep profunda femoral artery (PFA) and superficial femoral artery (SFA). The laterally situated PFA is an important source of collateral flow to the distal vessels. Significant occlusive disease of the PFA, in conjunction with severe SFA disease, can lead to CLI. This fact must be recognized and patency of the PFA be preserved when working on the SFA [2].

The tibial or infrapopliteal arteries provide blood flow to the gastrocnemius and soleus muscles of the calf, as well as to the arterial arcade of the foot. While these vessels are common sites of PAD, single or multiple stenosis of one crural vessel rarely provokes claudication. Rather, significant disease of all three infrapopliteal arteries (anterior tibial, peroneal, and posterior tibial) is usually required to provoke symptoms in the absence of proximal flow-limiting lesions of the ileo-femoral system. In the setting of limb salvage, the majority of patients will present with multi-level and multi-lesion disease [2].

NON-INVASIVE EVALUATION

Ankle-Brachial Index: Non-invasive studies such as ankle-brachial indices (ABI): Values below 0.3–0.4 are associated with severe claudication. Non-healing ulcerations or gangrene occur in the presence of ankle pressures of less than 50 mmHg. Toe systolic pressures are particularly useful in diabetic patients where the ABI may be falsely elevated due to arterial calcification. Values less than 30 mmHg suggest CLI and portend poor tissue viability. Other modalities of testing include ultrasound, Doppler, CT angiogram and MR angiogram.

INVASIVE EVALUATION

Angiography: It is important to take multiple angiographic views of the anatomy both before and after the interventional procedure. This allows the operators to better visualize the location and type of lesions, and identify the origin of occlusions (the "stump") when managing the chronic total occlusions (CTO) and to assure no occurrence of significant distal embolization compromising the distal flow. If the anterior-posterior (AP) view shows the external iliac artery (EIA) overlapping the internal iliac artery (IIA) then a 20° caudal and 20° left anterior oblique (LAO) angulation would separate the IIA and EIA. To separate the SFA and the PFA, or to highlight the origin of the SFA, an ipsilateral 30° oblique (or steeper) angulation would help.

Many patients have identical arterial anatomy in both limbs therefore an angiogram of the less diseased, contralateral limb often helps the operator to determine the size and location of arteries in the limb receiving treatment. This "roadmap" feature plays a vital role in the accuracy and efficiency of an intervention.

Due to the frequent presence of flow-limiting proximal disease, it can be very difficult to deliver sufficient contrast to opacify the distal vasculature. When severe multi-level disease is present, it is necessary to place a small diagnostic catheter in the common femoral artery, or even at any level (iliac, femoral, popliteal or infra popliteal) for a more detailed image of a particular arterial segment [2].

CRITICAL THINKING

Angiography with Knee Bend in Order to Detect Hinge Point: Conventional angiography (CA) (with the knee in extension) followed by dynamic angiography (DA) (with the knee in flexion), is an attempt to identify the exact place where the popliteal artery (PA) has a hinge point (HP) during knee flexion. An infusion of nonionic, low osmolality contrast in two boluses of 10 cc each in 1 second is administered through a pump. Images are captured with 35 mm film at 30 photos per second. The DA has a static phase and a dynamic phase. During the static phase, the patient lies on the side to be studied, with the knee in flexion at 100º (grade 0º is defined as the axis of the leg that continues to the axis of the thigh). Focus is on the knee joint, using lateral and medial projection. Once opacification of the PA has occurred, the leg is passively extended to total extension (dynamic phase). Evaluation of the artery takes place during the entire recording process using the same projection and focal point [3].

INDICATIONS

The accepted indication for PAD endovascular interventions remains primarily the treatment of CLI. Most patients with chronic PAD maintain lower extremity perfusion adequate for tissue viability, but insufficient to prevent ischemic pain during exertion (claudication) [2].

CLI is defined by the Transatlantic Inter Society Conference (TASC) as persistent recurring ischemic rest pain requiring opiate analgesics for at least 14 days, ulceration or gangrene of the foot or toes and ABI <0.40, toe pressure <30 mmHg, systolic ankle pressure <50 mmHg, flat pulse volume waveform, and absent pedal pulses. Most patients with CLI have multilevel, multivessel disease, often with three-vessel tibioperoneal occlusive disease [4].

The subacute or chronic manifestations are ischemic rest pain, trophic skin changes, tissue loss or simple claudication. Ischemic rest pain is characterized by discomfort in the toes, forefoot, or heel that is not relieved with the use of simple analgesics. Ischemic rest pain is predominantly nocturnal, and patients will often need to dangle their feet over the side of the bed to get relief. Minor trauma to the skin or nails usually precedents a poor-healing ulcer. An ulcer is classified as nonhealing when 8–12 weeks of wound dressings and wound care fail to result in complete healing. Gangrene can ensue when the reduced arterial supply to the limb does not meet the metabolic demands of the ischemic leg. Late stages of CLI are characterized by sensory loss and muscle weakness. Infection may precede or may complicate the nonhealing ulcer [4].

MANAGEMENT

All patients must receive aggressive medical management, including antiplatelet drugs, aggressive lipid-lowering therapy, optimal blood

glucose control, adjunct wound care, antibiotics, partial debridement, and analgesics [4]. The interventional goal is to restore the "straight-line flow" to the pedal arch in one or more tibial arteries. Dilatation of a proximal lesion in the setting of a distal occlusion will not be adequate for wound healing. Success of therapy is measured by relief of rest pain, healing of ulcers, and avoidance of major amputation. Because treatment of CLI is largely linked to the quality-of-life issue of being able to walk, the most important aspect of life quality for CLI patients is preservation of the limb. The primary goal, above all others, is to leave the patient with an ambulatory foot, so the patient can walk without prosthesis [3].

ACCESS

Contralateral Retrograde Approach: This is the preferred approach for interventions in the CFA, SFA, PA, proximal peroneal and proximal tibial arteries. This approach is also preferable in patients with a large pannus.

TECHNIQUE Crossing the Aortic Bifurcation: When performing this approach, use an internal mammary or pigtail catheter. Hook onto the bifurcation with this catheter and advance a stiff glide wire as distally as possible to the contralateral side. Remove the catheter and advance a sheath and dilator (Ansel [Cook, Bloomington IN] Raabe [Cook, Bloomington IN], or Pinnacle Destination [Boston Scientific, Natick MA] are recommended) over the stiff glide wire. If the sheath cannot be advanced over the bifurcation, then the operator should attempt to pull back the dilator while advancing the sheath forwards, or the operator can remove the sheath and exchange the glide wire for a stiffer wire, such as the Amplatz [Boston Scientific, Natick MA], and attempt to cross again.

The disadvantage of the loss of wire control when very distal lesions are approached from this remote location can be overcome by placing a 6Fr guide in the distal SFA akin to coronary angioplasty. The working length of wires will have to be 300 cm and the balloon working length of at least 120 cm will be required when tackling the infrapopliteal vessels from this approach [2].

Ipsilateral Antegrade Approach: This is the preferred approach when addressing distal anatomy where length of interventional devices becomes an issue such as the distal tibial vessels, the dorsalis pedis, and the plantar arteries. This approach is also used in patients where the contralateral approach is not feasible, such as in steep iliac bifurcations (where a sheath will not cross over) or patients who have had an aorto-femoral bypass.

The operators should always prepare the patient for a high stick when performing this type of access. Retract the pannus in large patients and put a small rolled towel under the ipsilateral hip in order to lift the hip up. Identify the femoral head under fluoroscopy and puncture in a horizontal coaxial fashion. When treating distal tibial disease, the operator can inject contrast through a long 6Fr or 7Fr sheath for better images while conserving contrast.

TECHNICAL TIPS

****Entering the Superficial Femoral Artery:** Various maneuvers are possible if the wire is persistently directed toward the PFA. After documenting that the entry point was into the CFA, the needle can be redirected to the contralateral wall, and the wire readvanced into the SFA. Alternatively, the floppy tip of a moveable core wire can be advanced into the PFA with the wire pushed to herniate into the SFA. A third method is to leave the wire in the PFA, exchange the entry needle for a small 4 or 5Fr angled catheter, remove the wire, and withdraw slowly the angled catheter while dye is injected to redirect it into the SFA. A variation on this maneuver would be to similarly exchange the needle for a 4Fr dilator, place a 0.018" wire in the PFA through the dilator and attached Tuohy-Borst adapter, and then with dye injections and/or roadmapping, advance a second 0.018" wire through the dilator into the SFA. Finally, through special order from Cook, a Saddekni-Cope dilator catheter with dual lumen side and end holes allows for wire catheterization of the SFA after the PFA is inadvertently entered [2].

ANGIOGPLASTY

Percutaneous transluminal angioplasty (PTA) is defined as the localized stretching of a vessel wall with a pressurized balloon to break apart plaque and restore blood flow. The advantages of PTA include reasonable technical and clinical success rates (some $>$90%) and low major complication rate ($<$10%). The main disadvantages of PTA include: (1) frequent occurrence of dissection that may require stenting; and (2) the need for frequent patient follow-up/surveillance. The technical success and patency also vary by lesion location and morphology (e.g. often better results in TASC A and much worse in TASC C or TASC D lesions) [5].

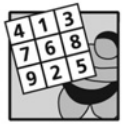
STENTING

Stenting is often considered the primary therapy for focal iliac and diffuse SFA lesions, it is becoming increasingly common as a "bail-out" for infrapopliteal PTA. The main advantages of stenting include: (1) improved midterm patency (compared with PTA in particular); (2) predictable angiographic results; and (3) near absence of acute and subacute occlusions. The main disadvantages of stenting include stent fracture and restenosis which frequently involves the entire length of the stent and is often longer than the original lesion [5].

ATHERECTOMY

Other leading non-stent modalities include debulking by the excimer laser (ClirPath Excimer Laser, Spectranetics Co, Colorado Springs, CO) and the excisional atherectomy (FoxHollow Technologies, Redwood City, CA), as well as innovative balloon technologies such as cryoplasty (CryoVascular PolarCath System, Boston Scientific Co, Natick, MA). These modalities are needed for crossing a CTO lesion or complimentary to angioplasty or stenting. Atherectomy with a Silverhack (FoxHollow Technologies, Redwood City, CA), can be a stand-alone procedure.

STRATEGIC MAPPING



Common Femoral Artery: The CFA overlies the femoral head and hip articulation and is the most frequently used vascular access for diagnostic and interventional procedures. Endovascular stenting of the CFA may make future vascular access and femoropopliteal bypass surgery at this site difficult or impossible. In addition, because of flexion of the joint, stent fracture as well as the durability of vessel patency after percutaneous intervention are legitimate concerns in this location. For this reason, the use of endovascular stents is advocated only after failed balloon angioplasty (provisional stenting) [2].

Ostial SFA: Most operators consider debulking using the Silverhawk Plaque Excision Catheter [Foxhollow Technologies, Redwood City, CA] as the best option for this particular lesion type. It will recanalize this area while preserving the PDA (no plaque shift). This area does not respond well to angioplasty (high recoil and restenosis rate) and is not a good area to stent (risk of shutting off the PFA and preventing future access).

Mid SFA: Intervention in the SFA systems is plagued with excessive restenosis rates. The SFA lesions are generally diffusely diseased, often occluded, and lie in an area of the body subject to substantial motion. The SFA undergoes multiple stresses including extension, contraction, compression, elongation, flexion and torsion. So stenting is not ideal here. Atherectomy is best option [3].

Distal SFA: The proximal and mid-segments of the SFA are relatively immobile, but the distal segment of the SFA after it exits the adductor hiatus into the popliteal is not well supported by external tissues and undergoes substantial bending and twisting. The areas of bend are not easily identified during straight leg angiography. It was generally assumed that arterial bend occurs in the area of the joint space. The portion of the artery that exhibits the most motion is the area just distal to the adductor hiatus. Stenting across this area of bend with relatively stiff nitinol stents (stents that do not compress well longitudinally) can exacerbate the bend in the native artery at the ends of the stent and cause traumatic interaction between the native artery and the stent edge [3].

Popliteal Lesions: Operators should avoid stenting in this area if possible. Silverhawk plaque excision or laser atherectomy followed by angioplasty with a low profile balloon (Ampheron Deep, Submarine [EV3, Minneapolis, MN]) are the best options of these popliteal lesions [3].

Infragenicular Lesions: Most operators treat this area with atherectomy and angioplasty as needed. In some cases, stenting may be a useful modality here. For in-stent restenosis, a debulking strategy is best by using laser or directional atherectomy and subsequent angioplasty and stenting if needed [3].

CHRONIC TOTAL OCCLUSION OF THE SFA

Most long SFA occlusions begin with a proximal stump followed by varying degrees of distal vessel reconstitution by way of collaterals from the PFA. Angiographic assessment of the proximal stump requires a 35° to 40° ipsilateral lateral angiogram. In this view, one can determine the length of the proximal stump and the possible access options, including antegrade, contralateral, or even brachial approaches. If the proximal stump or cap is less than 3–5 cm long, management of the ostial SFA segment must be factored into device selection [6].

Piercing the Hard Proximal Cap: If a stump is not visible in the AP view, take a 35° ipsilateral view and study the contralateral limb angiograms to help identify the SFA origin. Roadmap the reconstitution site to provide a target when wiring. Study the anatomy of the contralateral limb when it is available. The initial cap of CTO's is usually crossed with support catheter such as a Glide Catheter or Quickcross catheter [Spectranetics, Colorado Springs CO] and an angled or straight stiff Glidewire. In rare cases, a operator may choose to use a stiffer wire, such as the Confianza [Abbott Laboratories, Evanston, IL] or the back end of a glidewire, or the Frontrunner Catheter [Cordis Corp, Miami, FL]. Do not do angiograms through support catheters that have not re-entered the true lumen as this will stain the subintimal space and impair distal visualization. In patients with a difficult proximal cap, an operator might attempt recanalization through the popliteal access (retrograde popliteal stick).

TECHNIQUE The SafeCross Wire: The SafeCross wire (IntraLuminal Therapeutics, Menlo Park, CA) has the unique property of an optical coherence reflectometer. This wire is coupled with radiofrequency energy that is delivered from the tip if the reflective signal obtained by the near-infared sensor identifies a luminal position, signified by a green indicator. Radiofrequency is not deliverable if the reflective signal is red, suggesting wire proximity to the endoluminal wall. The benefit of this technology is the theoretical advantage of remaining in the intraluminal space, thus reducing the dissection plane of a long occlusive lesion. Certain anatomical situations favor its use. (1) A flush occlusion of the SFA with no visible nub, (2) occlusions across the knee joint, and (3) occlusions at the site of a prominent collateral channel are three clinical scenarios in which intraluminal passage with the SafeCross wire may produce greater success [6].

Crossing a Long CTO Segment of the SFA: Many long occlusions of the SFA are composed of a focal proximal cap followed by a long segment of gelatinous debris culminating in a distal fibrous cap. Some operators use a stiff angled glide wire and create a small loop, supported by straight glide or Quickcross catheter. Make sure the loop is not larger than 5–6 mm (the diameter of most SFA's). If the loop increases in size, it often means significant dissection into the subintimal space. The success of reliable re-entry into the true lumen diminishes

as the diameter of the wire loop increases. When the wire loop size increases, the operator should stop advancing, retract the wire into the support catheter, then probe with the wire and re-engage the occlusion at this point with a smaller wire loop with catheter support [6].

Distal Re-Entry: When the wire tip is near the distal cap, the operator should retract the wire, and probe again without the wire loop if possible first before attempting to re-enter with a loop. The operators should never spend more than 15 minutes attempting to cross the distal recanalization site, especially in heavily calcified vessels. Do not continue to create a more distal subintimal channel with more dissection if the wire does not appear to be in the true lumen at this point. This is where re-entry devices are utilized. Further dissection with the Glidewire would lead to progressive subintimal channel creation, reducing the likelihood of successful true lumen re-entry. Upon re-entry into the true lumen, the operator should be able to remove the wire and aspirate blood through the support catheter. If this occurs, the operator can then take an angiogram through the catheter with diluted contrast to evaluate the distal anatomy [6].

TECHNICAL TIPS

****Piercing the Distal Cap:** The location of the distal reconstitution strongly influences device choice for re-entry catheters. Re-entry catheters have the highest degree of success in lesions that reconstitute above the adductor canal, where the vessel is relatively large. If the occlusion does not collateralize until the popliteal artery or even lower, re-entry becomes more challenging. Even more important is the progressive dissection below the knee compromising important distal collaterals and potentially worsening claudication symptoms. Remember that a 7F sheath will be necessary for most of these devices. Once a contralateral sheath is in place, either the Pioneer catheter (Medtronic Vascular) or the Outback (Cordis Co, a Johnson & Johnson Co, Miami, FL) can be used [6].

TECHNIQUE The Pioneer Catheter: The Pioneer device is a 6.2Fr catheter with two wire ports, each 0.014-inch compatible, one with a hollow core nitinol needle that is guided by an integrated 64-element, phased-array intravascular ultrasound device and is connected to a Volcano (Volcano Corporation, Rancho Cordova, CA) intravascular ultrasound (IVUS) console, enabling vessel imaging. The device is delivered through the subintimal plane and is placed with the distal tip at the level of the SFA re-entry site. By slowly rotating the catheter, the ultrasound image is maneuvered until the tip of the nitinol needle is oriented toward the true lumen and is lined up at the 12-O'clock position on the ultrasound image. The needle is plunged into the lumen at a controlled depth, typically between 3–5 mm. The soft-tipped, nonhydrophylic, .014-inch floppy-tipped wire in the monorail port of the catheter is sent through the needle into the distal vessel and is confirmed with angiography. The catheter is carefully

removed without pulling out the re-entry wire and routine intervention is performed. Difficulty in successful re-entry is encountered with highly calcified vessels, poorly visualized distal reconstitution, deep subintimal catheter location, and poor wire angle of the floppy wire [6].

TECHNIQUE The Outback Catheter: The Outback catheter (Cordis, Miami FL), used for re-entry, is a 6Fr compatible catheter with a hollow 22-gauge cannula for distal vessel entry using fluoroscopic imaging. The device is delivered to the distal subintimal space adjacent to the reconstitution of the vessel and two orthogonal angiographic views are taken. The proprietary locate, tune, and deploy technique is used to increase successful distal re-entry. An L-shaped fluoroscopic marker provides reproducible orientation of the tip toward the re-entry target site. The "T"une fluoroscopic marker, combined with a 90° orthogonal view, confirms the desired alignment to fine tune positioning at the target re-entry site. Lastly, the 22-gauge, nitinol re-entry cannula is plunged into the distal vessel to re-enter into the true lumen [6].

DIFFUSELY DISEASED SFA LESIONS

The operators can use a combination of Silverhawk Plaque Excision [Foxhollow Technologies, Redwood City, CA] followed by angioplasty if needed. As an alternate approach, operators can debulk using the Spectranetics 2.5 mm Turbo Laser [Spectranetics Corp, Colorado Springs, CO] in conjunction with post laser angioplasty in most cases. If neither of these techniques is adequate, the operator can choose to stent. Spot stenting is preferable to multiple overlapping stents. In longer lesions, its best to use a flexible fracture resistant stent such as the Everflex [EV3, Minneapolis, MN] rather than multiple overlapping stents to reduce restenosis and stent fracture.

TECHNIQUE The Foxhollow Atherectomy: The SilverHawk [Foxhollow Technologies, Redwood City, CA] is a monorail catheter with a carbide cutting blade at the tip. When activated, this blade spins at 8000 rpm. The SilverHawk is a forward-cutting atherectomy catheter. Plaque is excised as the catheter is advanced through the lesion. The excised plaque is collected into the nose cone of the catheter and can subsequently be removed from the body. The nose cone can be cleared and additional passes can be made with the catheter. The SilverHawk catheter offers the advantage of more effectively debulking lesions compared to excimer laser or other atherectomy devices. In many cases, stand-alone atherectomy can be performed.

Debulking strategy with the Foxhollow atherectomy is preferred for diffuse disease and CTO of both above and below the knee prior to further treatment. In the femoral artery, debulking reduces the need for more aggressive balloon dilation and the need of stenting which has its own limitation in the femoral artery. Lesion below the knee is best managed by debulking and PTA if needed and stenting as the last resort [6].

TECHNICAL TIPS

****Safe Atherectomy with the Silverhawk:** During atherectomy with the Silverhawk, advance the cutter slowly and cut and pack every 6 cm to avoid any embolization. Take a look after four quadrants for any perforation. Avoid excessive torquing because of wire wrapping. The Silverhawk catheter also allows preferential plaque cutting in a specific vessel wall.

TECHNIQUE Eximer Laser: The 308 nm excimer laser utilizes flexible fiberoptic catheters to deliver intense bursts of ultraviolet (UV) energy in short pulse durations. Tissue is ablated only on contact, with minimal surrounding thermal injury. A unique feature of UV light is its ability to ablate plaque and thrombus and thus reduce the potential for embolic complications. Debulking with the excimer laser prior to adjunctive balloon angioplasty offers the advantage of a better angiographic result with less dissection and reduced need for stenting. This may be particularly advantageous for long (>20 cm) occlusions in which multiple stents would be required. Furthermore, using the "step-by-step" technique (wireless laser-assisted angioplasty) in which the laser is used to create a wire channel, can be very useful in total occlusions that are refractory to traditional wire crossing techniques [6].

CAVEAT

Importance of Distal Reconstitution Point:
When approaching the distal reconstitution point, it is critical that the operator does not force the wire more than a few millimeters distal to the reconstitution point. Failure to stop at this point can lead to a less favorable surgical option (e.g. if the loop is pushed below the knee in a patient that has a reconstitution point above the knee, the patient could be forced to have a Femoral Below Knee Bypass versus an Above Knee Femoral–Popliteal as a surgical option if the percutaneous revascularization fails). Operators should take extra care re-entering the distal reconstitution point in areas that are not "stent friendly" such as the popliteal artery.

Operators should always make an attempt to recanalize a CTO in a critical limb ischemia patient. In these patients, if unsuccessful on the first attempt (e.g. if one cannot re-enter or a false lumen is created), one should consider re-intervention again in 4–6 weeks due to the dire consequences (amputation) of failure to recanalize in these patients.

Short CTO Segment of the SFA: In short segment occlusions with frequent collaterals, the operator can attempt to advance the Quickcross with the wire retracted inside in an attempt to bluntly

dissect through the occlusion, then re-advance the wire. Some operators can also use a 0.9 mm laser catheter [Spectranetics Corp Colorado Springs, CO] and wire to ablate and traverse the initial cap of a SFA CTO using a step by step approach (ablate 1 mm, advance wire until meeting resistance, advance the laser and ablate 1 mm, advance wire, etc).The SafeCross catheter [Medtronic Minneapolis, MN] can also be used to cross the initial cap of an SFA CTO. If the guidewire continues to select a large collateral, one may obturate the collateral with a small balloon to redirect the wire into the main vessel [6].

TECHNIQUE Blunt Microdissection: The Frontrunner catheter [Cordis Corporation, a Johnson & Johnson Co, Miami, FL] is a blunt microdissection device that takes advantage of the elastic properties of adventitia versus inelastic properties of fibrocalcific plaque to create fracture planes. This technique may be advantageous in penetrating hard fibrous caps of the SFA occlusions. The device separates atherosclerotic plaque in various tissue planes, creating a passage through the CTO [6].

INFRAPOPLITEAL CTO

When selecting wires, the operators should start with small diameter 0.014 wires such as the Miracle Bros wire series [3 g, 4.5 g, 6 g tips Abbott Laboratories, Deerfield IL] or the Confianza [9 g tip Abbott Laboratories, Deerfield IL]. If this is not successful, one can switch to 0.018 wire and 0.018 support catheter (Quickcross or balloon catheter). If these combinations are not successful, then one can try an 0.035 wire system. For catheter support, one should consider starting with an 0.035 crossing system to allow for more wire options such as 0.035 stiff angled Glidewire and size down to smaller catheters if necessary.

CAVEAT

Priority of Vessels: Evidence of distal reconstitution in the ankle or foot helps in successful re-entry into the true lumen, however, in CLI patients one should attempt to wire distally regardless of the existence of this distal target. In treating below-the-knee (BTK) lesions in patients with non-healing wounds or gangrene one should preferentially treat vessels in the following order of priority: (1) vessel directly feeding the symptom (e.g. if a patient has gangrene in the great toe, the operator should treat the vessel feeding that area of the foot, generally the anterior tibial and dorsalis pedis); (2) totally occluded arteries as there is no negative consequence of unsuccessful intervention of this artery (occluded artery remains occluded so no change in patient prognosis); (3) the dominant vessel feeding the majority of the foot.

TECHNICAL TIPS

****Further PTA after Proximal Opening:** Often after successfully recanalizing one BTK vessel, improved flow through the treated vessel often exposes reconstitution of subsequent BTK vessels. This often allows an operator to successfully cross and treat subsequent BTK vessels that initially appeared too difficult to attempt. Operators may choose to attempt retrograde access using a micropuncture set through the distal post tibial artery or dorsalis pedis if standard attempts to wire an artery in an antegrade fashion are unsuccessful. If one successfully crosses a vessel in this fashion, the operator should snare the wire and remove it through the antegrade sheath and use a small catheter to perform a wire exchange and reverse the wire direction and intervene through the larger antegrade sheath.

COMPLICATIONS

Access Complications: When using a contralateral approach, operators should avoid excessive sheath manipulation to avoid distal embolization. When performing antegrade puncture, insert the needle at a 45-degree angle and coaxially to avoid intra-operative sheath kinking. Operators should be sure to use fluoroscopy to identify the femoral head to avoid a high or low stick. High sticks can lead retroperoneal bleeds, and low sticks can lead to intraoperative thrombosis.

TECHNICAL TIPS

****Avoiding Plaque Excision Complications:** When performing plaque excision in the SFA, operators should always take a picture after cutting in four quadrants to avoid a deep wall cut or a perforation. One should always take care not to overcut around artery bends and heavily calicified vessels. When encountering resistance, operators should not force the device distally. Instead, apply slight forward pressure and slowly rotate the catheter to find a plane with less resistance. Then return to the resistant area and try again. One should always pack the larger devices after cutting the length of the nosecone (e.g. if the nosecone is 4 cm, pack every 4 cm). Some operators choose to use distal protection when performing atherectomy in heavily calcified vessels with single vessel run-off.

****Avoiding Laser Atherectomy Complications:** One should never push the laser catheter if there is any resistance. When operators encounter resistance, they should pull the catheter back and let the laser work at the point of resistance for a few seconds, or downsize to a small catheter.

****Avoiding Angioplasty Complications:** Operators should always choose the proper balloon size to the size of the artery. Operators should never over-inflate the balloon. One should always be sensitive to patient feedback while performing an angioplasty. If the patient complains of anything but minor pain then the operator

should user lower pressures when inflating the balloon, or in post dilating a stent.

Perforations: For minor perforations (particularly wire perforations) in the SFA many operators elect to continue the procedure and reduce or reverse anticoagulation. For minor perforations below the knee, one should reverse the anticoagulation and compress externally with an ACE wrap to avoid compartment syndrome. For perforations from atherectomy, operators can often manage the situation with long, low pressure balloon inflations for a few minutes and a reversal of anticoagulation. If this does not work after a few attempts, one can use a covered stent, such as the Viabahn [W.L. Gore, Flagstaff AZ].

Arterial Spasm: Operators often see spasm in the tibial/peroneal arteries which can be relieved with intra-arterial nitroglycerin. If this does not resolve on the first attempt, one should deliver several 100 micrograms through a catheter directly into the artery in spasm. One may also try a combination of nitroglycerin and a calcium channel blocker for greater efficacy. Some operators elect to use papaverine to treat spasm due to its longer half life.

Acute Thrombosis: Operators can manage interoperative thrombosis with the use of mechanical thrombectomy catheters such as the Export Catheter [Medtronic Inc, Minneapolis, MN] or Rheolytic Catheters such as the Angiojet [Possis Medical, Minneapolis, MN]. Operators can also choose to deliver lytics locally through an infusion catheter for a few minutes or overnight. Laser is also another option in these patients.

Distal Embolization: Operators can use manual aspiration catheters such as the Diver [EV3], Pronto [Vascular Architects, Nashville, TN], Quick Cat [Kensey-Nash, Exton, PA]. If this is unsuccessful one can attempt to tack the embolus using a low profile angioplasty balloon. If residual embolization exists, one can consider using anticoagulation for a longer period of time and/or a bolus of a 2b3a inhibitor.

Compartment Syndrome: This syndrome refers to the situation in which the pressure in a closed space, usually one of the enclosed myofascial compartments of an extremity, becomes high enough to restrict tissue perfusion and oxygen delivery. It usually follows prolonged ischemia and often results from both the original ischemic insult, as well as reperfusion.

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Chapter 24
Inoue Balloon Mitral Valvuloplasty

Jui Sung Hung, Kean-Wah Lau

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

Percutaneous balloon mitral valvuloplasty (BMV) introduced in 1984 by Inoue *et al.* [1] has ushered in a new dimension in the treatment of patients with mitral stenosis. Extensive clinical studies have established this minimally invasive, non-surgical procedure to be a safe and effective therapeutic modality in selected patients with mitral stenosis

[2–8] and is equivalent to or even better than surgical commissurotomy [9–13].

With successful balloon valve enlargement, there is generally a two-fold increase in the mitral valve area [2–8] and an associated dramatic fall in transmitral valve gradient, left atrial pressure and pulmonary artery pressure. These hemodynamic benefits are mirrored in post-procedural improvement in the patients' symptoms and exercise tolerance [14]. The long-term results of BMV are excellent, especially when the acute results are optimal and in the presence of good valve morphology [14–19].

Besides the original Inoue technique using size-adjustable, self-positioning balloon catheters, various other techniques using fixed-sized balloon catheters have been developed for performing BMV. These include the antegrade (transvenous) approaches with one or two balloon catheters through one or two inter-atrial septal punctures [20,21] or the retrograde (transarterial) approaches with or without transseptal access [22]. However, the Inoue balloon catheter system via the transvenous approach has remained the principal BMV technique used today.

Our extensive experience in Inoue BMV has demonstrated that incremental operator experience and ongoing evolving technical refinements in BMV techniques have resulted in a nearly 100% technical success rate and a significant diminution in complications despite the presence of a significant number of technically demanding scenarios and high-risk co-morbid conditions [4,14,23–25]. This chapter focuses on the discussion of the pitfalls and tricks in Inoue-balloon BMV to ensure success and to minimize complications of the procedure. We hope that this chapter will be beneficial to all Inoue BMV operators at different levels of experience. The instrumentation of the Inoue balloon catheter system has been extensively described in previous publications [1–3], and thus is omitted from this chapter.

TRANSSEPTAL ACCESS

Transseptal catheterization is a vital component of BMV. Transseptal puncture must be not only executed safely to avoid cardiac perforation, but also made at an appropriate septal site to facilitate balloon crossing of the stenosed mitral valve.

To avert cardiac perforation during transseptal catheterization, some operators have resorted to routine intra-procedural transesophageal [26,27] or intracardiac [28] echocardiography to facilitate optimal transseptal needle placement. However, even with the echocardiographic guidance, cardiac perforation may still occur [26]. Therefore, acquisition of basic transseptal skill is essential. To perform transseptal procedure, biplane fluoroscopic equipment is preferable, but single-plane fluoroscopy is usually sufficient. The essential instruments for transseptal access are listed in Table 24-1.

Catheter/Needle Fitting Exercise: A catheter/needle fitting should be performed before its insertion into the patient. First, fully

Table 24-1 Instruments for Septal Puncture

1	A Brockenbrough needle
2	A 7F or 8F dilator catheter
3	An outer sheath catheter (optional)*

*The use of the sheath is optional, but its utility is recommended, especially for inexperienced operators, for two reasons: (1) to prevent inadvertent perforation of the dilator by the needle during its insertion, and (2) to prevent left atrial perforation during insertion of the catheter/needle into the left atrium because the sheath tip works as a safety stopper at the septum.

insert the transseptal needle until its tip extends beyond the catheter (Figure 24-1a). Then withdraw the needle until its tip is concealed slightly (2–3 mm) within the tip of the catheter. The operator should fix his/her right index finger on the needle between the direction indicator and the catheter hub to prevent the needle from accidentally moving forward and protruding from the catheter tip. This is of vital importance during *in vivo* manipulation of the catheter/needle. The direction indicator on the needle is held between the thumb and the index finger (Figure 24-1c).

Landmarks for Optimal Puncture Site: To select an optimal transseptal puncture site, there are two imaginary reference lines that need to be defined first: (1) the vertical "midline;" and (2) the horizontal M-line. The target site for septal puncture is, as a rule, located at the intersecting point of the vertical "midline" and the horizontal M-line.

1 Definition of the Vertical "Midline"

(a) Inoue's Angiographic Method: Inoue has devised a specific transseptal puncture technique designed for the Inoue-balloon BMV, incorporating the concept of a vertical "midline," a line assumed to divide the intra-atrial septum into anterior and posterior halves [27]. This line is defined based on the landmarks obtained from right atrial angiography during normal respiration in the frontal plane (Figure 24-2a,b).

(b) Hung's Modified Method: Because in most cases of mitral stenosis, the left atrial silhouette is visible under fluoroscopy, this author (JSH) has modified Inoue's method of defining the "midline." In this method, the aortic valve instead of the tricuspid valve is used as a landmark because of their proximity. Therefore, point T is substituted with the tip of an pigtail catheter (Figure 24-2c, point A) touching the aortic valve (usually the non-coronary sinus of Valsalva) in the frontal view. A horizontal line is drawn from point A to L, where the line intersects the right lateral edge of the left atrium. The "midline" thus derived is usually identical to that from the Inoue's angiographic method.

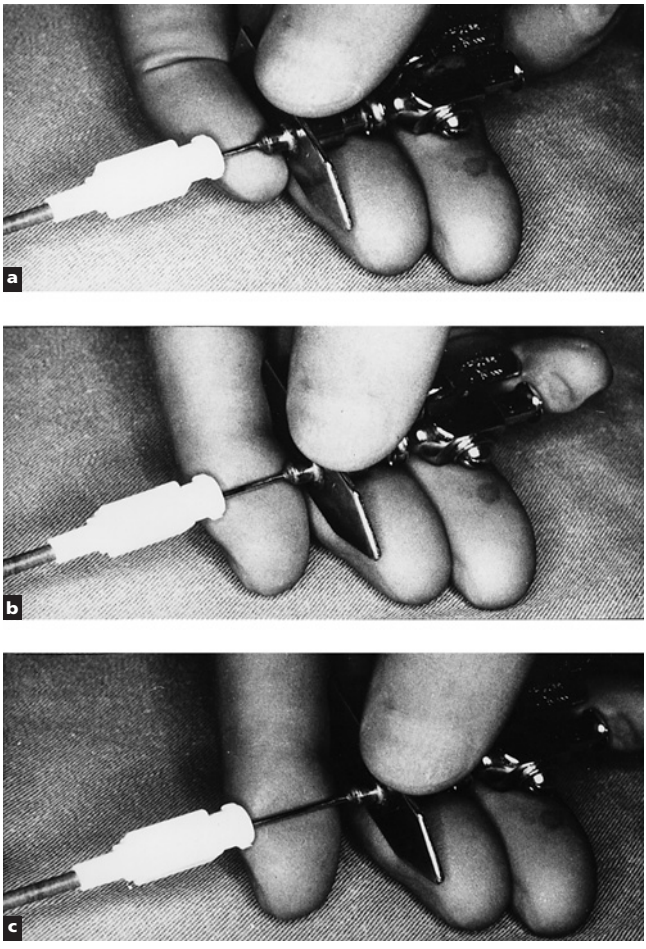


Figure 24-1 Catheter/needle fitting exercise. (a) First, fully insert the transseptal needle until its tip extends beyond the dilator tip. (b) The needle is then withdrawn until its tip is aligned with the dilator tip. (c) It is pulled back farther, thus the needle tip is concealed slightly (2–3 mm) from the dilator tip. The index finger is fixed as a stopper on the needle between the direction indicator and the catheter hub to prevent the needle from moving forward and protruding from the dilator tip. This is of vital importance during in vivo manipulation of the catheter/needle. The depth and the angle of the stopper-finger (c) are adjusted according to the distance between the direction indicator and the catheter hub in each catheter/needle set. Each side of the direction indicator is held by the thumb and the index finger, respectively. This makes rotation of the indicator easier, and also allows the blunt part of the direction indicator visible to the operator (and the tutor, if any). (Courtesy of www.ptmv.org.)

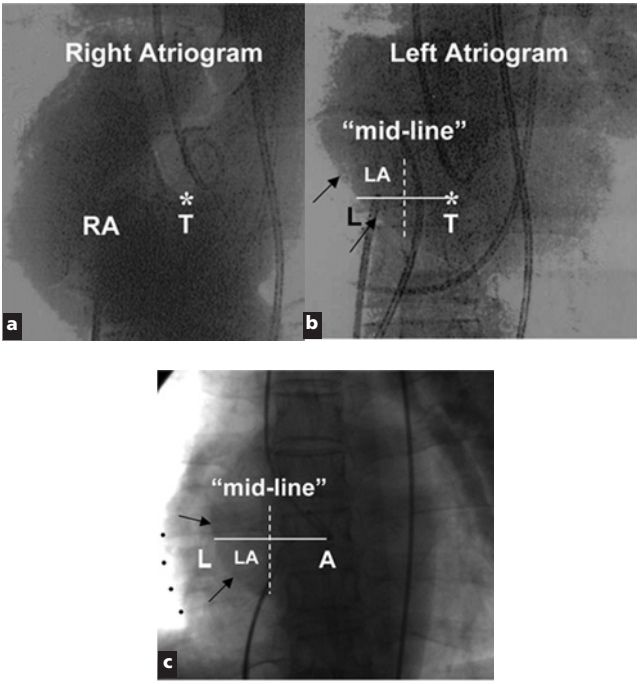
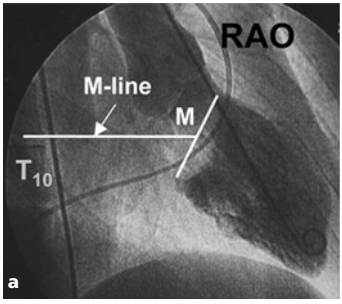


Figure 24-2 The "mid-line." Angiographic method (a and b): the upper end of the tricuspid valve at systole (point T, marked as asterisk) is determined on a stop-frame frontal right atrial (RA) angiographic image (a) and translated to a stop-frame left atrial image (b). On the latter image, an imaginary horizontal line is drawn from point T until point L, where the line intersects the lateral border of the atrium encountered first (usually the left atrium, as in this case). Point L is assumed to be the posterior limit of the septum. The dotted vertical line crossing at the mid-point between T and L is the "mid-line". Fluoroscopic method (c): a horizontal line is drawn from the tip of the pigtail catheter (point a) to L, the LA silhouette (black arrows), to define the "midline." The dotted line indicates the right atrial silhouette. (Courtesy of www.ptmv.org.)

TECHNICAL TIPS

****Variances of the Mid-Line:** The septum lies within the superimposed area between the two atria, and therefore there is no septum outside this area. The lateral (or posterior) limit of the septum is the lateral border of the medial atrium, usually the left atrium. Infrequently (such as in patients with giant left atria), the lateral border of the right atrium is medial to that of the left atrium, and thus the point L should be on the right atrial border because there is no septum laterally beyond this point.



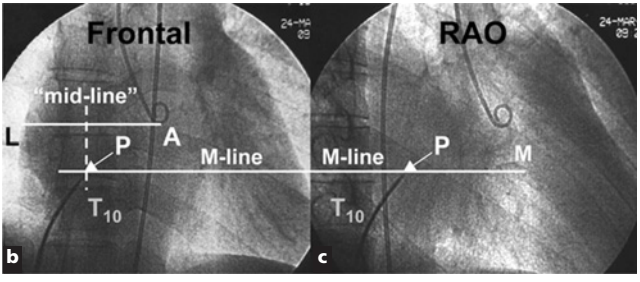


Figure 24-3 Defining horizontal M-line, vertical "midline" and target puncture site. The M-line is obtained using the stop frame RAO view left ventriculogram (a). This line is memorized in relation to the vertebral body T_{10}, and transcribed in the frontal (b) and RAO views (c). The vertical "midlines" is defined in fluoroscopic frontal view. The target site for septal puncture (point P) is located at the intersecting point of the vertical "midline" and the horizontal M-line (b). A, pigtail catheter tip; RAO, right anterior oblique; T_{10}, 10th thoracic vertebra. (Courtesy of www.ptmv.org.)

BMV can be performed with the patient in a semi-recumbent position under an urgent situation. In this setting, the "midline" can be defined in frontal view with appropriate caudal tilting [29]. The frontal image intensifier needs to be tilted in a caudal angle corresponding to the degree of semi-recumbency to negate the patient's tilt and "normalize" the positional relationship of the various intrathoracic structures. For example, if the patient is lying at 30° to the horizontal, the frontal image intensifier should be rotated to 30° caudally.

2 Definition of the Horizontal "M-Line": The "M-line" is a horizontal line crossing the center of the mitral annulus (point M) as visualized in a left ventriculographic diastolic stop-frame obtained in 30° RAO projection (Figure 24-3a). This line is memorized in relation to the vertebral body; there is no need to plot it on the image monitor screen. The stop-frame angiogram is also used as a road map during transseptal puncture and balloon catheter manipulation.

In individual cases, the puncture point in relation to this line may have to be adjusted. For example, in patients with a more vertically

oriented left ventricle, the puncture site is chosen slightly above the horizontal M-line. In patients with giant left atria, the operator is often forced to make septal puncture more caudal to the M-line.

TECHNICAL TIPS

****Appropriateness of the Inoue Method:** Inoue's angiographic method is suited in following situations: (1) for operators inexperienced with the transseptal puncture technique; (2) in cases in which atrial silhouettes are not well visualized under fluoroscopy; (3) in extremely difficult cases of transseptal puncture, e.g. in the presence of a giant left atrium [29], or kyphoscoliosis [30]. In these cases, it may be necessary to perform biplane (frontal and lateral) right angiography to properly visualize the atrial septal orientation and relative anatomic relationships of the both atria, the tricuspid valve, and the aorta.

SEPTAL PUNCTURE

Placement of Transseptal Catheter/Needle: The catheter/sheath is inserted via the right femoral vein over a guide wire into the superior vena cava to the level of the carina. The catheter is aspirated and flushed after removal of the wire. Then, the Brockenbrough needle, attached with a 5cc plastic syringe containing pure contrast medium, is inserted into the catheter and carefully advanced under fluoroscopic view until its tip reaches at the predetermined position (refer to "catheter/needle fitting exercise" above). The needle is allowed to rotate freely during its passage. The right-hand stopper-finger is now firmly kept between the catheter hub and the direction indicator of the needle to prevent the needle from moving forward (Figure 24-1). Extreme care should be taken not to let the needle slip forward during subsequent manipulation of the catheter/needle.

Catheter/Needle Manipulation: Under frontal fluoroscopic view, the needle-fitted transseptal catheter with its direction indicator pointing about 4 O'clock is slowly withdrawn downwards (caudally) from the superior vena cava. A clockwise rotation is applied to the direction indicator to align the catheter/needle on the "midline." The catheter/needle is further withdrawn until its tip reaches the level of the pigtail tip touching the aortic valve (Figure 24-3b).

Under lateral view the catheter/needle is further withdrawn caudally while contrast medium is being injected (septal flush method) [27], by an assistant or using the operator's right hand while fixing the catheter hub and the direction indicator using the left hand, to outline the right atrial margin of the septum (Figure 24-4c,d). The catheter tip is finally set at the curvilinear portion of the septum at the altitude of the M-line (Figure 24-4e).

Subsequently, the catheter needle/needle tip position is viewed under 30° RAO projection, contrasted with the left ventriculogram road map, to confirm optimal septal puncture site as well as to avoid puncture of other structures (Figure 24-4d). The catheter/needle tip

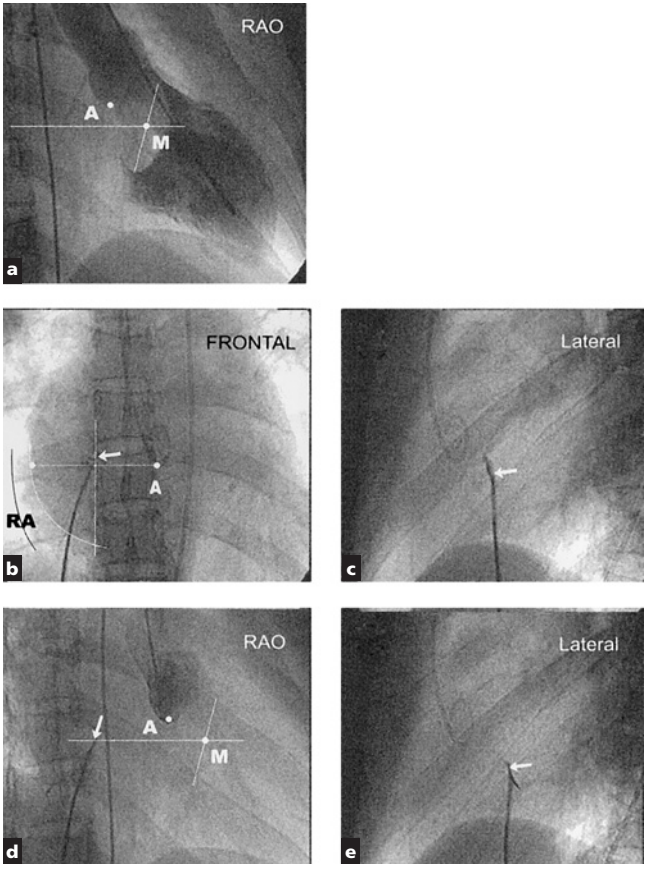


Figure 24-4 Catheter/needle manipulation. (a) Stop-frame left ventriculogram in 30° RAO view shows a horizintal line, M-line, crossing the center of the mitral annulus. (b) Under frontal fluoroscopic view, the needle-fitted transseptal catheter is slowly withdrawn downward (caudally) from the superior vena cava to align the catheter/needle on the vertical mid-line. The catheter/needle is further withdrawn until its tip reaches the level of the pigtail tip touching the aortic valve (point A). (c) Under lateral view, the catheter/needle is further withdrawn caudally while contrast medium is being injected (septal flush method) to outline the right atrial margin of the septum. (d) The catheter/needle is further withdrawn to set its tip at the curvilinear portion of the septum at the altitude of the M-line. At this point, the catheter/needle is observed to be pointed dorsally. (e) Subsequently, the catheter/needle tip position is viewed under 30° RAO projection, contrasted with the left ventriculogram road map (a), to confirm optimal septal puncture site as well as to avoid puncture of other structures (the aorta, the coronary sinus and the tricuspid valve). The catheter/needle tip is now seen on the M-line, usually just anterior to the vertebra.

is now seen on the M-line, usually just anterior to the vertebra, and away from the asending aorta, the coronary sinus and the tricuspid valve. While frontal and lateral views are sufficient for experienced operators, the RAO view is especially vital for inexperienced operators.

TECHNICAL TIPS

****Exact Positioning of the Catheter/Needle Tip:** In most cases of mitral stenosis, a sudden sharp movement of the catheter/needle towards the left is not observed when the tip of the transseptal assembly falls over the limbic ledge and enters the fossa ovalis. This is because the atrial septum bulges markedly towards the right atrium makes the fossa ovalis more shallow. When the septal bulge begins in the upper septum, the catheter/needle being withdrawn from the superior vena cava takes a lateral course to the "midline". In this case, turning the needle to the 3 O'clock position may lead the catheter/needle to a medial position. If not, the needle alone can be withdrawn slightly, and the floppy tip of the catheter should tend to flip medially. Then the needle is advanced slowly and carefully to bring its tip back to the original position while keeping the catheter tip in the medial position. If the above means also fail to place the catheter/needle medially, the latter is withdrawn further downward and close to the lower edge of the left atrium (passing the caudal end of the bulge). With the needle pointing toward the left (about 3 O'clock), the catheter tip is allowed to shift medial to the "midline" and then carefully advanced cephalad. A clockwise twist is made to the needle and the catheter tip is steered to or near the target point.

****Exact Positioning of the Catheter/Needle Tip in Giant Left Atrium:** If the atrial septum bulges markedly towards the right atrium, especially in cases of a giant left atrium; it is difficult to align the catheter tip with the "midline" and perpendicular to the septum. The catheter tip faces a strong resistance at 4 O'clock when it touches the bulged septal surface. As the needle is being rotated clockwise, the catheter/needle will give way suddenly. In effect, the needle tip flips over the crest of the bulge and points towards the right side of the patient at 9 O'clock. To prevent this, the catheter should be pressed slightly against the septum as the needle is being rotated clockwise to 6 to 7 O'clock. At the same time, a slight counter-clockwise twist is applied to the catheter with the left hand to counter any excessive clockwise rotation of the needle. If the crest of the bulge happens to be at the "midline," it is not possible to make a puncture on the line. In this case the puncture site is settled in the region slightly lateral to "the midline."

****Repositioning the Catheter/Needle Tip After Failed First Attempt:** If the initial pass of the transseptal catheter/needle is not successful in engaging it at an appropriate puncture site, the needle is removed from the catheter and the second attempt is begun by repositioning the catheter in the superior vena cava over a guide wire.

For experienced operators, the alternative is to reposition the catheter/needle high in the right atrium. This is done by setting the needle in the 12 O'clock direction (ventrally) and carefully moving the catheter/needle upward (cephalad) while slightly rotating the direction indicator of the needle clockwise and counter-clockwise to make certain the catheter tip is free and not caught against the right atrial appendage or its free wall.

****Needle Tip Reshaping:** Reshaping of the distal needle to make it more curved may be necessary in the following situations: (1) when the catheter/needle tip tends to take a more lateral course to the "mid-line" despite counter-clockwise rotation of the direction indicator to 3 O'clock direction; and (2) at the intended puncture site, there is a sharp angle between the direction of the catheter/needle tip and the septum, therefore making septal penetration impossible or causing septal dissection when the needle is advanced forward.

The Technique of Septal Puncture: When the operator is satisfied with the intended puncture site, the catheter/needle is pressed firmly against the septum. Usually cardiac pulsations (so-called septal bounce) are felt by the right hand holding the catheter/needle. While keeping the catheter firmly against the septum to prevent it from slipping away from the puncture site, the operator releases the stopper-finger and advances the needle forward. The needle is aspirated and contrast medium is injected to confirm its entry into the left atrium. If no blood is aspirated, the needle either has dissected the high septum or is caught in the thickened septum. Staining of the septum with injection of a small amount of contrast medium (septal stain method) [27] easily distinguishes between the two (Figure 24-4). This type of septal staining is of no consequence since contrast medium is absorbed rapidly. When the high septum is dissected, it is stained in more vertical fashion. In this situation the needle is withdrawn and septal puncture is made at a slightly more caudal site.

TECHNICAL TIPS

****How to Puncture a Thick Septum:** When the needle is caught in the thick septum (usually, in the muscular septum), the stain takes more oblique orientation (Figure 24-5c,d). In this case the catheter/needle is carefully forced across the septum as described below or the puncture is attempted at another site. When the catheter/needle is being advanced, a "tenting" of the septum is observed before the septum is entirely pierced by the catheter/needle. With pressure monitoring, it is not possible to differentiate dissection of the high septum from entrapment of the needle in the thick septum. This is another reason why the authors perform the transseptal puncture without constant pressure monitoring.

When marked resistance is encountered during septal puncture, a sustained force is applied to the catheter/needle. After several cardiac beats, not infrequently a "give" is felt or seen under fluoroscopy when

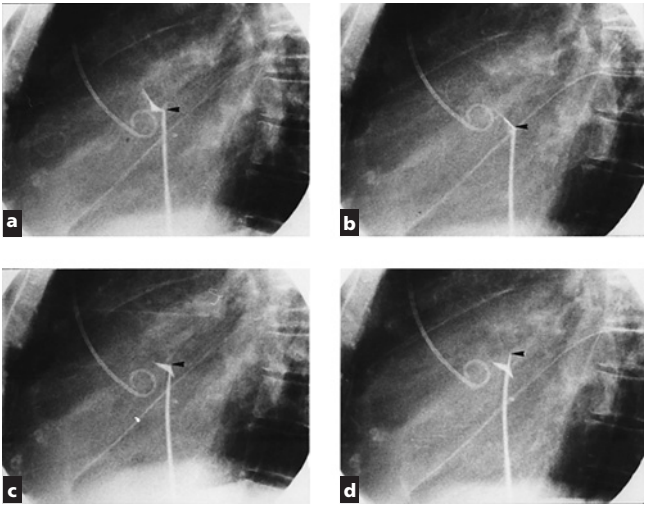


Figure 24-5 Septal flush/stain method illustrated in lateral views. (a) As the catheter/needle assembly is withdrawn caudally it is flushed with contrast medium which outlines the right atrial margin of the septum. The tip of the catheter/needle (black arrow) is at the high anterior septum. (b) The catheter/needle is withdrawn to set its tip at the puncture target site, and the needle is advanced. (c) Since the puncture is made in the thickened muscular septum, the needle is caught in the septum as demonstrated by oblique septal stain. When the catheter/needle is advanced, a "tenting" of the septum is observed. (d) The needle is carefully forced through the septum. (From [27]).

the catheter/needle finds its way into the left atrium. If this method fails to place the catheter/needle across the septum, a Bing stylet, which has a blunt tip, is inserted and extended beyond the needle. The catheter/needle is carefully forced through the tough septum by a forward push with the right hand while applying counter resistance with the left hand. During the process the operator must be prepared to withdraw the needle as soon as the catheter enters the left atrium, so that the excessive forward momentum does not carry the needle forward and perforate the left atrial wall, causing cardiac tamponade.

****How to Avoid Puncturing the Aorta, Tricuspid Valve and Coronary Sinus:** When the catheter/needle is set on the "midline," puncture of these structures can be avoided. Being confirmed in the RAO projection, the intended puncture site is clearly separated from the aorta, the tricuspid valve and the coronary sinus (Figure 24-3c). Inadvertent puncture of the aorta, as confirmed by contrast injection or pressure recording, is usually uneventful if the needle is withdrawn immediately; however, should the operator unknowingly

advance the catheter into the aorta, it should not be withdrawn. The patient should be sent for emergency surgery with the catheter left in the aorta.

****Avoid Puncturing Medial to the "Midline":** When a puncture is made medial to the line, there is a risk of puncturing the aorta, tricuspid valve or coronary sinus. More importantly, the puncture site thus made is too close to the mitral valve, and this makes balloon crossing of the mitral valve difficult or even impossible, unless the posterior-loop method (refer to "Crossing the mitral valve") is employed. Slight lateral deviation of the puncture site to the "midline" is permissible, especially in patients with relatively small left atria.

****How to Avoid Puncturing the Right Atrium:** To avoid injury of the right atrium, the catheter/needle should be carefully manipulated and the needle tip always kept inside the catheter tip. When right atrial perforation is detected by contrast opacification of the pericardial space, do not advance the catheter and withdraw the needle/catheter immediately. Usually cardiac tamponade does not ensue, and the operator may proceed with puncture attempt at the optimal site. It is important to note that there may not be septum in an area near the inferior (caudal) border of the left atrium caudal to the M-line because the atrium often bulges caudally beyond the true septal boundary. This is especially true in patients with a large left atrium. If this region is punctured, the catheter/needle may perforate through the right atrial wall and then enter the left atrium (the so-called "stitching" phenomenon) [27]. After the guide wire is placed in the left atrium and the catheter is withdrawn, cardiac tamponade ensues. To avoid puncturing the right atrium, setting of the catheter/needle tip at the septum can be confirmed by (1) observing the "septal bounce", and (2) the septal flush/stain method, as discussed above (Figures 24-4 and 24-5).

Confirmation of Left Atrial Entry: After entry of the needle in the left atrium is confirmed, first by contrast medium injection followed by pressure recording, the needle direction is set toward 3 O'clock (left side of the patient). If there is no or little resistance, the catheter/needle is advanced forward about 2 cm into the left atrium. Then, the catheter alone is advanced another 2 cm (or until the tip of the sheath meets a resistance at the septum), while the needle is being withdrawn.

Heparinization: Upon removing the needle after the catheter is placed in the left atrium, heparin, 100 units/kg body weight, should be given immediately through the catheter. After baseline hemodynamic studies, including simultaneous measurement of cardiac output, BMV is performed. If the patient has been on warfarin prior to BMV, the drug is discontinued 2–3 days before the procedure and substituted with intravenous unfractionated or subcutaneous low-molecular weight heparin until before the procedure.

Table 24-2 Catheter Selection and Balloon-sizing Based on Patient Height and Valvular Status

Reference size (RS) (mm)

Height (cm) (rounded to nearest 0) × 1/10 plus 10, e.g. height = 147 cm
RS = 150 × 1/10 + 10 = 25 mm

Catheter selection

Valvular Status	Balloon Catheter
Pliable	RS matched (e.g. PTMC-26 for RS = 25 mm)
Calcified/SL	One-size < RS matched (e.g. PTMC-24 for RS = 25 mm)

Balloon sizing

Valvular status	Initial	Increments
Pliable	(RS−2) mm	1 mm, or 0.5 mm in high pressure zone** (if MR or unilateral commissural split)
Calcified/SL	(RS−4) mm	1 mm (low pressure zone*) 0.5 mm (high pressure zone**)

MR, mitral regurgitation, preexisting or increased; RS matched, catheter with its nominal balloon size = RS; SL, severe subvalvular lesions.
*Low pressure zone = balloon diameter <2 mm of nominal balloon size
**High pressure zone = balloon diameter within 2 mm of nominal balloon size

SELECTION OF BALLOON CATHETER

Selection of an appropriate-sized balloon catheter for the controlled stepwise dilatation technique is extremely important in order to avoid creating severe mitral regurgitation during BMV. Our balloon catheter selection methods have evolved from our continuing efforts to minimize this complication [4,14,23–25] (Table 24-2).

Selection guidelines are based on balloon reference size derived from patient height, transthoracic echocardiographic findings of the mitral valve, fluoroscopic presence of valvular calcification. The reference size (RS) is calculated according to the simple formula [14,23]: patient height (in cm) is rounded to the nearest zero and divided by 10, and 10 is added to the ratio to yield the RS (in mm); e.g., if height = 147 cm, then RS = 150/10 + 10 = 25 mm. In patients with pliable, noncalcified valves, and with angiographic mitral regurgitation ⩽1+, a catheter with a nominal balloon size at least that of the RS (an RS-matched catheter) is used. In contrast, in patients at high-risk for creating severe mitral regurgitation (valvular calcification and/or severe subvalvular lesions), a balloon catheter one size smaller than an RS-match is selected. Therefore, in the above example with an RS of 25 mm, a PTMC-26 catheter would be selected for a pliable, noncalcified valve, and a PTMC-24 catheter for a calcified valve and/or a valve with severe subvalvular disease.

TECHNICAL TIPS

****Pretesting for Balloon–Syringe Mismatch:** Although the volume predefined by red marks on the syringe and its corresponding balloon size at full inflation have been tested by the manufacturer, balloon–syringe mismatch may occur. While this mismatch is usually mild, gross mismatch may take place when the catheter and the syringe are from different packaging or reused after sterilization. The mismatch, if undetected, may result in either underinflation or overinflation of the balloon. The former may result in suboptimal valvular dilatation, and the latter, in severe mitral regurgitation. Therefore, before inserting the balloon catheter into each patient, the balloon diameters should be confirmed using a two-step test. First, the syringe should be filled with diluted contrast to the mark corresponding to the balloon diameter chosen for the first inflation (see "Balloon sizing" below). The balloon should then be fully inflated, and its diameter measured with a caliper. If there is a mismatch, the difference should be noted and adjusted during the second step of testing when the balloon is inflated to its nominal diameter.

After the pretesting exercise, the syringe is disconnected from the balloon catheter for two reasons. One is to purge the syringe of any remaining air, and the other, to avoid any inadvertent overinflation of the balloon at its nominal size. After the catheter has been inserted into the left atrium, the air-free syringe filled with diluted contrast corresponding to the predetermined initial balloon diameter is reconnected to the catheter.

ADVANCEMENT OF THE BALLOON CATHETER

Insertion of the stretched Inoue balloon catheter over the 0.025 inch stainless steel coiled-tip guide wire into the right femoral vein is smooth in the majority of patients. Occasionally, difficulties arise from resistance to the catheter at the femoral access site, or at the interatrial septum.

TECHNICAL TIPS

****Resistance at Groin Access Site:** To avoid creating a long subcutaneous tunnel which may pose some resistance during insertion of the balloon catheter, the puncture needle is angled more vertically than usual during the initial vascular access (at about 60° to the skin surface instead of 45°). After transseptal puncture and insertion of the coiled-tip guide wire into the left atrium, the subcutaneous track is then well stretched with an artery forceps along the guide wire. This is followed by use of the 12Fr dilator (enclosed with the Inoue balloon assembly), which is also used to dilate the atrial septum. Finally, when inserting the stretched balloon catheter, firm compression with the flat of the fingertips cephalad to the puncture site and over the subcutaneous track may be needed to aid catheter entry.

If significant resistance is encountered during insertion of the stretched balloon catheter, it is inserted into the vein at a more obtuse angle of about 90° until the catheter tip meets the posterior venous wall. The catheter is then tilted more horizontally and advanced over the wire. During the latter process, to facilitate catheter insertion and avoid bending the guide wire, firm compression should be applied cephalad to the puncture site and over the subcutaneous track (as described above), and the guide wire should be held taut by an assistant. If this technique fails, the subcutaneous track and the vein should be re-dilated with the 14Fr dilator. If these precautionary measures are exercised, the need for a 14Fr intravascular sheath for insertion of the balloon catheter is rare in our experience, even in patients with the right groin scarred from previous catheterization. However, one should not hesitate to use a 14Fr vascular sheath to avoid bending the guide wire or metal tube, when a difficulty is encountered during the catheter insertion process.

It is also important to note that during insertion of the catheter into the femoral vein, the catheter should never be twisted, lest the metal tube be bent. If the tube is inadvertently bent, it should be replaced with a new one. On the other hand, if the guide wire is bent, the 12Fr dilator is reinserted over the wire and carefully left into the left atrium. The wire is then replaced.

TECHNICAL TIPS

****Septal Resistance:** After atrial septal puncture and placement of the coiled-tip guide wire in the left atrium, occasionally, there may be some difficulty in advancing the balloon catheter across the septum, particularly when the latter is markedly thickened at the puncture site. When this occurs, forceful action is to be avoided as the catheter may curve excessively in the inferior vena cava, resulting in abdominal discomfort for the patient. Rather, the balloon catheter should be turned slightly, usually in a clockwise direction as it is pushed forward (screwdriver maneuver) to overcome septal resistance. In the rare instances when this method also fails, the septum is re-dilated with the dilator. After passage across the septum, it is also important not to push the catheter tip up against the left atrial roof, or the guide wire may be bent into an acute angle, making subsequent catheter manipulation difficult.

****Deep Catheter Placement in Left Atrium:** The balloon catheter is introduced under frontal fluoroscopic view into the atrium over the coiled-tip guide wire to form a large loop with the tip medial to the mitral orifice, pointing in a 6 to 7 O'clock direction (Figure 24-6, upper panel a). This placement has the following advantages: (1) the catheter thus positioned is less likely to flip to the left atrial appendage when the stylet is advanced to the catheter tip; (2) the catheter will not enter the pulmonary veins; and (3) in subsequent manipulations to cross the mitral valve, the deep-seated catheter will need only

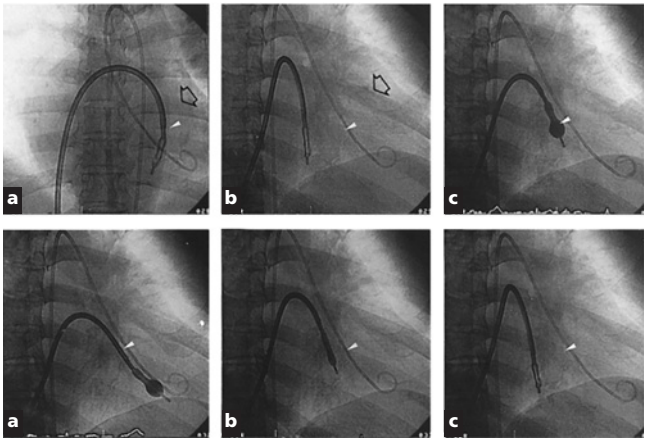


Figure 24-6 A balloon catheter is introduced under frontal fluoroscopic view into the left atrium over the coiled-tip guidewire to form a large loop with the tip medial to the mitral orifice, pointing 6 to 7 O'clock direction (upper panel a). After the deep catheter placement, the projection is changed to a 30° right anterior oblique view (upper panel b), and with the stylet inserted into the catheter tip, the partially inflated distal balloon is directed toward the anteriorly located mitral orifice. The catheter is then withdrawn gradually, to direct the balloon to the mitral valve (white arrowhead) (upper panel c) and to cross the mitral valve (bottom panel a). After balloon inflation procedure, the catheter balloon is then withdrawn to the left atrium. In subsequent crossing of the mitral valve with the stepwise dilation technique, the stylet is inserted on the catheter tip (bottom panel b) and the catheter is advanced to deep-seat the balloon (bottom panel c). During balloon catheter manipulation under a 30° right anterior oblique fluoroscopic view, the catheter tip should always be kept to the left of the pigtail catheter preplaced in the left ventricle to avoid trespassing on the left atrial appendage area (open arrowhead).

to be withdrawn. Thus, potential entrapment by a tough septum, which is only encountered during catheter advancement, is avoided (see "Catheter entrapment at atrial septum" below).

CROSSING THE MITRAL VALVE

After deep catheter placement in the left atrium, the fluoroscopic projection is changed from a frontal to a 30° right anterior oblique view (Figure 24-6, upper panel b) that displays the left ventricular long axis in profile. In patients with giant left atria, additional use of lateral fluoroscopic view may be needed to facilitate crossing of the valve.

Methods of Crossing: With the stylet inserted to the catheter tip, the partially inflated distal balloon is directed towards the anteriorly located mitral orifice. The balloon is directed anteriorly by applying a

counter-clockwise twist (usually 180°) to the stylet with the right hand (in a right-handed operator). The catheter is then withdrawn slowly, using the left hand, until a horizontal bobbing motion of the balloon is noted, indicating close proximity of the balloon to the mitral valve. Mitral valve crossing is then attempted using four methods in descending order of preference: (1) the vertical, (2) the direct, (3) the sliding, and (4) the posterior loop method. The vertical method is the most frequently successful crossing method.

1 The Vertical Method: Upon further slight retraction of the catheter, the balloon is observed to move in (during diastole) (Figure 24-7a,c) and out (during systole) of the left ventricle (Figure 24-7b,d) even though the catheter is not aligned with the orifice-apex axis. Coincident with diastole, only the stylet is withdrawn. To accomplish this, the operator must carefully watch the rhythmic motion of the heart. This allows the distal segment of the catheter to take on a more horizontal orientation to cross the valve and enter deep in the left ventricle (Figure 24-6e–g). If the distal portion of the catheter is still vertically oriented and points to the inferior wall of the left ventricle (Figure 24-7g), the catheter is carefully withdrawn to align it with the orifice-apex axis (Figure 24-7h). During the process, the distal bal-

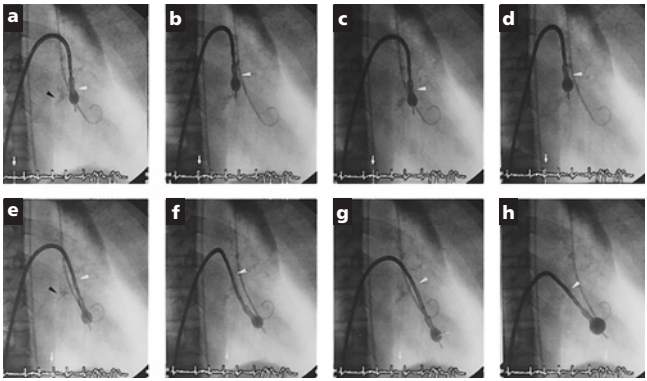


Figure 24-7 Vertical method. Fluoroscopic 30° right anterior oblique views during manipulations of Inoue balloon catheter to cross mitral valve. (a–d) During diastole (a and c) the catheter balloon crosses calcified mitral valve (black arrowhead) into left ventricle while during systole (b) and (d), it pops back into left atrium. (e) During diastole of the same cardiac cycle in (d), only the stylet is withdrawn and the distal catheter thus adopts a more horizontal orientation, permitting the balloon to enter into the left ventricle. (f–h) The catheter is retracted to align along the left ventricular long axis. White arrowheads indicate stylet tip position. White arrows at bottom of each frame depicts timing of cardiac cycle on the electrocardiogram. (See text for discussion). (From [31].)

loon may need to be inflated further to prevent it from popping out of the ventricle.

This vertical approach keeps the catheter from inadvertently flipping into the appendage, thus minimizing the risk of catheter encroachment into the left atrial appendage in cases with thrombi confined to the appendage [31].

2 The Direct Method: When the vertical method fails, the balloon catheter is further withdrawn until the catheter balloon is near the valve and the catheter is well aligned with the orifice-apex axis. At this time a "woodpecking" sign is observed as the balloon moves away from the mitral orifice in systole, and toward it in diastole along the mitral-apex axis. Once this sign is evident, the balloon is in position to cross the mitral orifice. With careful attention to the rhythmic motion, the operator jerks the stylet back slightly (4–5 cm) as the balloon approaches the orifice, and simultaneously advances the catheter with the left hand to drive the balloon flow across the valve deep into the left ventricle. Because timing is critical, in an operator's early experiences with BMV, selection of patients with sinus rhythm is recommended, as it is then easier to make use of the regular cardiac cycle to advance the balloon across the mitral orifice.

TECHNICAL TIPS

****Optimal Position of the Stylet:** In the vertical and direct methods, it is important to insert the spring-wire stylet all the way to the balloon catheter tip to straighten the latter. Occasionally, however, the stylet may be too short to reach the catheter tip, thus making a slight bend in the catheter tip. If this occurs, the rubber grip at the proximal end of the stylet can be pulled further back to lengthen the exposed segment of the stylet or, failing which, the rubber grip is cut at 1–2 mm from its distal end and removed. To maintain the anterior orientation of the balloon (toward the mitral valve), the stylet must be kept twisted at all times. An extra counter-clockwise twist is occasionally needed to direct the catheter tip anteriorly, especially in cases with giant left atria. In these cases, the septum is displaced markedly to the anterior, and thus the balloon catheter tends to point more posteriorly. An added lateral fluoroscopic view will facilitate manipulation of the catheter/stylet for balloon crossing of the mitral valve in these instances.

3 The Catheter Sliding Method: When the vertical or direct method fails, another technique that may be useful for crossing the mitral valve is the catheter sliding method [23]. This method has proved to be effective in cases when the septal puncture is made too caudally and/or the left ventricle takes a more horizontal orientation (Figure 24-8a). The balloon is first directed toward the mitral valve by keeping the stylet twisted counter-clockwise. The distal catheter segment is then made more flexible by withdrawing the stylet clear out of the balloon segment (Figure 24-8b). Once the slightly inflated balloon is

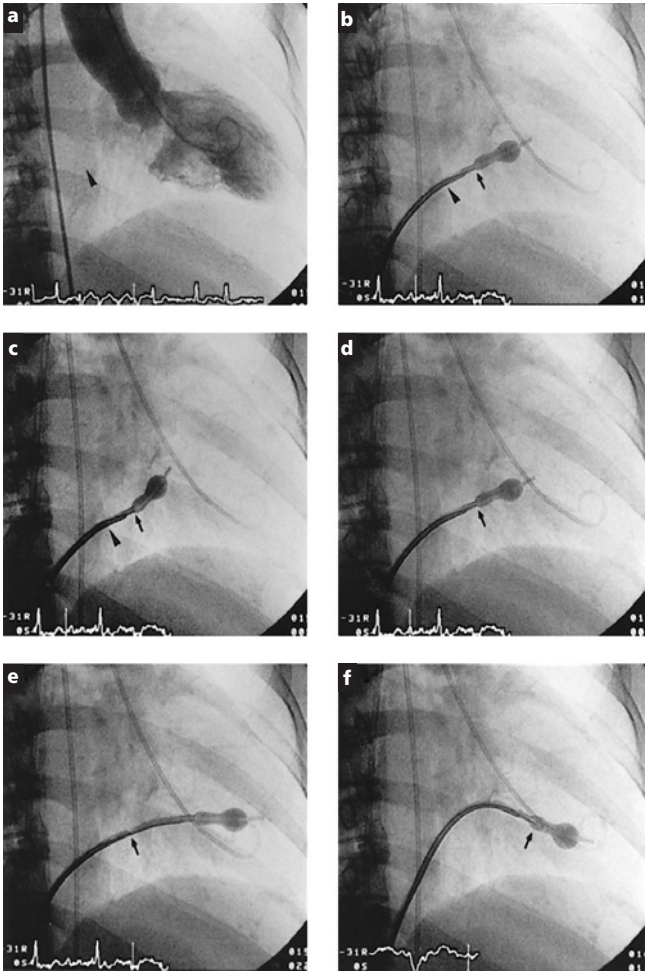


Figure 24-8 Catheter sliding method. (a) Left ventriculogram in 30° right anterior oblique view, showing position of the transseptal puncture site (arrowhead), quite caudal to the mitral orifice; the left ventricle is more horizontally oriented. (b) The stylet (arrow) is slightly withdrawn from the balloon segment. (c,d) During a cardiac cycle, the balloon segment bobs up and down in systole and diastole, respectively, indicating proper positioning of the catheter tip at the mitral orifice. (e) During diastole, when the balloon bobs down and is aligned with the distal catheter (as in d), only the catheter is advanced forward (with the stylet kept fixed) to place the balloon into the left ventricle. (f) Thereafter, the stylet is carefully advanced to align the catheter with the mitral orifice/ventricular apex axis before initiating the balloon inflation procedure. (From [23].)

at the mitral orifice, cardiac contractions will cause the balloon segment to tilt upwards during systole (Figure 24-8c). In diastole, the balloon segment aligns with the catheter shaft (Figure 24-8d). With the operator carefully watching the rhythmic motion of the cardiac cycle, only the catheter is advanced forward (with the stylet kept fixed) during diastole to cross the valve (Figure 24-8e). The stylet is then advanced to help align the catheter with the orifice-apex axis (Figure 24-8f).

4 The Posterior Loop Method: Crossing the valve with the balloon catheter may be difficult with the above mentioned methods in patients with giant left atria, or when the atrial septal puncture has been made inappropriately either too cephalad or too anterior in relation to the mitral valve. In such circumstances, the loop approach may be used. This method, which has been well described previously [2,3], is infrequently used in this authors' experience.

TECHNICAL TIPS

****Stylet Reshaping:** The J-tipped stylet with its original curve will, in most instances, steer the balloon toward and across the mitral orifice. However, when it is difficult to direct the balloon toward the mitral orifice by aligning the catheter with the orifice-apex axis, the stylet should be reshaped according to the positional relationship between the septal puncture site and the valve orifice. For example, in patients with a giant left atrium where puncture site is often made more caudally and laterally in relation to the mitral orifice, the distal segment of the stylet can be shaped into a larger smooth curve to facilitate passage of the balloon across the mitral valve. Conversely, in those with a relatively small left atrium, when the puncture site is made suboptimally, either too medially or anteriorly (in relation to the mitral valve), the stylet can be reshaped into a tighter loop (or the posterior loop method described above can be employed).

BALLOON INFLATION

Assuring Free Balloon Movement in the Left Ventricle: One of the most dreaded complications of BMV is the development of severe mitral regurgitation requiring surgery. Once the mitral valve has been crossed, the free movements of the partially inflated distal balloon in the left ventricle should be ascertained to prevent the disastrous consequences, i.e. rupture of chordae, papillary muscles or leaflets, stemming from its subsequent full inflation between the chordae. This is done by simultaneously pushing the catheter and pulling the stylet slightly in opposite directions ("accordion" maneuver) [23] to ensure that the partially inflated distal balloon slides freely along the orifice-apex axis.

TECHNICAL TIPS

****If the Balloon Strays Among the Chordae:** After crossing the mitral valve, the catheter balloon may point more vertically

and deviate away from the orifice–apex axis. This suggests that the catheter has strayed among the chordae. To correct this situation, the distal balloon is inflated larger to prevent the balloon from being inadvertently retracted into the atrium, and the catheter is carefully pulled back to assume a more horizontal orientation. After satisfactory alignment of the catheter with the orifice–apex axis, the catheter is advanced toward the apex, and the previously described accordion maneuver is performed before initiating the inflation procedure. Similarly, a twist in the balloon during the inflation process may also indicate that the catheter has tethered among the chordae. In this case, the inflation should be promptly aborted and the balloon repositioned.

Reassessment of Subvalvular status: Before BMV, mitral valvular status is determined by pre-procedural transthoracic echocardiography and fluoroscopy (for the presence of valvular calcification), and an appropriate balloon catheter is then chosen accordingly (see "Selection of balloon catheter" above). Extensive subvalvular disease has been found by various investigators to be a predictor for significant mitral regurgitation. Because echocardiography (either transthoracic or transesophageal) often underestimates the severity of subvalvular disease [24], severe mitral regurgitation may be created during BMV despite the presence of an apparently favorable valve morphology. Therefore, during the actual balloon dilatations, vigilance is required to identify the presence of previously undetected severe subvalvular disease. We and others have found other more reliable signs of significant subvalvular involvement [14,25,32]. Even in patients in whom no severe subvalvular disease is demonstrated by pre-procedural echocardiography, when any of the signs described below are observed, the balloon dilatation protocol is altered accordingly as described below (see "Balloon sizing" below).

TECHNICAL TIPS

****Severe Subvalvular Disease Undetected by Echocardiography:** The following signs suggest or indicate the presence of severe subvalvular disease.

1 Difficulty in Performing the Accordion Maneuver: This occurs because of resistance at the subvalvular level. If this difficulty is not appreciated, subsequent full balloon inflation will be within the left ventricle as the balloon is not anchored at the mitral valve. Hence, it is the subvalvular apparatus and not the mitral valve that is dilated. Although severe mitral regurgitation may result from such an accidental subvalvular dilatation [32], the inflation is usually harmless. However, it should be promptly recognized and the balloon quickly deflated. The size of the distal balloon is then reduced during subsequent attempts to anchor the balloon at the mitral valve.

2 Gross Indentation of the Inflated Distal Balloon: (balloon compression sign) (Figure 24-9). This indicates severe subvalvular disease

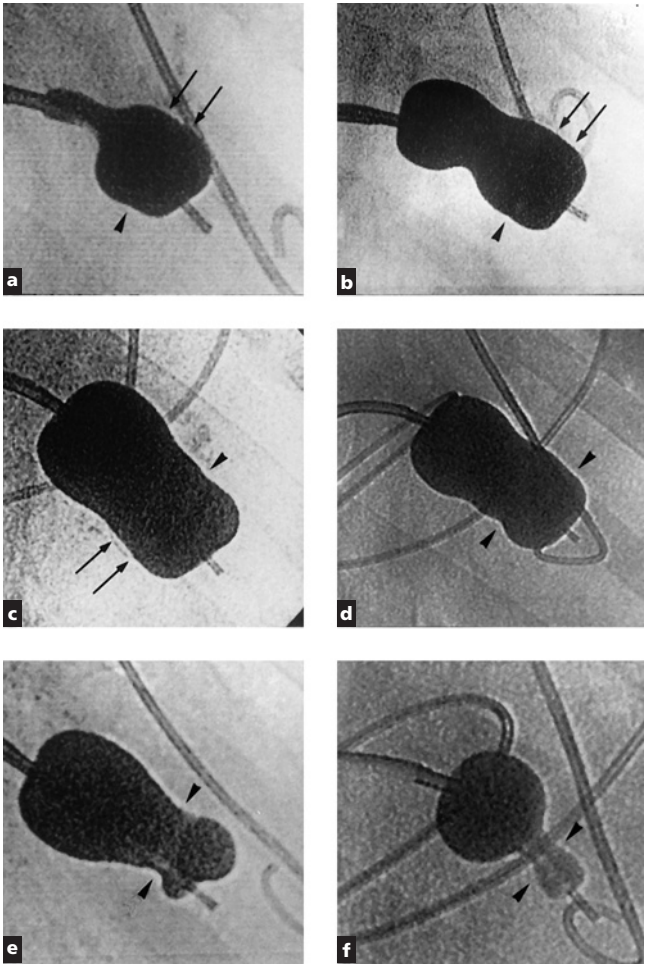


Figure 24-9 Various deformities of inflated balloon caused by severe subvalvular disease. (a–e) Indentation (arrowheads) and compression (arrows) are observed on the distal balloon segment. (f) During in-situ test inflation of balloon at the mitral valve, the proximal but not the distal segment is inflated as the latter is compressed (arrowheads) by severe subvalvular disease (see text for discussion). (From [23].)

[14,25]. As soon as compression is observed on the distal balloon, the inflation procedure is aborted and the inflation strategy reassessed.

3 "Balloon impasse": (Figure 24-9f) In cases of tight mitral stenosis, valve crossing may be difficult even when the catheter, with its distal balloon partially inflated, is properly aligned with the long

axis of the left ventricle. If this occurs, the balloon size is gradually reduced until it is accommodated by the mitral orifice. In rare and extreme instances, even when the balloon is not inflated, the catheter is checked (or entrapped) at the mitral valve. This finding, which we have termed "balloon impasse," reflects resistance caused by severe obstructive subvalvular lesions [25]. In the presence of this sign, BMV performed with the usual catheter selection and balloon sizing is likely to tear the mitral leaflets and/or chordae and thus create severe mitral regurgitation. Our experience in a limited number of patients suggests that in addition to stepwise dilatations previously emphasized, the use of smaller balloon catheters may prevent the complication of severe mitral regurgitation [25].

4 Cogwheel Resistance: Rarely, while withdrawing the partially inflated balloon to anchor it at the mitral valve, cogwheel resistance may be encountered. This suggests the presence of subvalvular disease.

CONTROLLED STEPWISE DILATATIONS

In order to avoid or minimize the complication of severe mitral regurgitation, the selection of an appropriate balloon catheter (discussed above) and the controlled stepwise dilatation technique are mandatory. In addition, one should be familiar with the pressure–volume relationship and inflation limit of the less compliant balloon of the second-generation catheter now in use [24].

Pressure–Volume Relationship: The intra-balloon pressure transits from the "low-pressure" to the "high-pressure" zone as the balloon is inflated to within 2 mm of its nominal size, e.g., the 24–26 mm zone in a 26 mm-balloon catheter. Each catheter can be safely inflated to a maximal diameter of 1 mm above the nominal size because of the built-in safety margin. Initial balloon inflation is never to be performed with balloon diameter in the high pressure-zone regardless of the valvular morphology.

Balloon Sizing: Balloon sizing for the stepwise dilatation technique is crucial in avoiding the complication of severe mitral regurgitation (Table 24-2). Our balloon sizing methods have evolved through our continuing efforts to minimize this complication. By adhering to the cautionary methods outlined below, especially in patients with severe subvalvular disease, creation of significant mitral regurgitation (increase of $\geqslant$2+ angiographically) can be minimized [4].

TECHNICAL TIPS

****Balloon Sizing in Patients with Pliable, Noncalcified Valves:** In patients with pliable, noncalcified valves and no severe subvalvular lesions, as determined by the subvalvular reassessment outlined above, an RS-matched balloon catheter is selected as stated previously. The initial inflated balloon diameter is RS minus 2 mm. In subsequent dilatations, the balloon size is increased by 1 mm. When

there is preexisting mitral regurgitation or any question of increase in the degree of mitral regurgitation, the increment should be 0.5 mm in the high-pressure zone. This approach also applies when unilateral commissural splitting occurs during the previous dilatation, as observed by asymmetrical balloon waisting on fluoroscopy. The final diameter is best kept within 1 mm above the RS to avoid oversizing: our previous study [14] showed that oversizing of the balloon is a risk factor for creating severe mitral regurgitation in this group of patients.

****Balloon Sizing in Patients with Calcified Valves and/or with Severe Subvalvular Disease:** In patients with either fluoroscopically visible valvular calcification, or severe subvalvular lesions as observed by transthoracic echocardiography, instead of an RS-match, a balloon catheter one size smaller than the RS-match is selected at the outset. For those whose subvalvular lesions are not detected by preprocedural echocardiography, the RS-matched catheter already placed in the patient may still be used if the dilatation procedures are carried out with extra care. Ideally, the catheter should be exchanged for a smaller one, but this is quite costly.

For the first dilatation, a balloon diameter 4 mm less than the RS is used. For subsequent dilatations, the balloon size is increased by 1 mm in the low pressure zone and by 0.5 mm in the high-pressure zone until satisfactory results are obtained or until mitral regurgitation develops. In cases where the gradient has already been reduced to one-half and several more dilatation attempts have failed to reduce it further, the procedure is terminated to avoid creating severe mitral regurgitation [24]. Reducing the mitral valve gradient by one-half should result in a 41% increase in the mitral valve area, as calculated by the Gorlin formula, provided that heart rate and cardiac output remain the same. Our previous study [24] suggests that a 40% improvement in the valve area is sufficient for symptomatic improvements in patients with a more sedentary life style.

****Balloon Sizing in Cases of "Balloon Impasse":** If balloon impasse (Figure 24-9f) is encountered, the initial catheter is exchanged for a smaller PTMC-18 or -20 catheter to predilate the valve and the subvalvular structures, regardless of the echocardiographic findings of the mitral valve [25]. We no longer force the usual-sized balloon through the valve to the left ventricle by slenderizing and stretching the deflated balloon segment, as previously recommended [2]; nor do we recommend advancing the balloon across the mitral valve over a guide wire preplaced in the left ventricle. Both maneuvers may cause the catheter to stray among the chordae, and with larger-sized balloon catheters, it is difficult or impossible for the operator to execute the precautionary "accordion" maneuver to ensure that the catheter is not tethered among the chordae.

However, if a smaller PTMC-18 or -20 catheter also fails to cross the mitral valve with the catheter uninflated, the balloon segment of this small balloon catheter is slenderized and stretched for crossing the

mitral valve. Before the balloon inflation procedure, it is mandatory to exercise the "accordion" maneuver with the distal balloon slightly inflated to ensure that the balloon catheter is free in the left ventricle. This maneuver would not have been possible with larger-sized catheters. The initial inflation is then performed with the balloon diameter at its nominal size. If further dilatations are required, the catheter is exchanged for one a size larger, and stepwise dilatations are done according to the sizing method in patients with severe subvalvular lesions, as discussed above.

Exchange for Different-sized Balloon Catheters: Exchange of balloon catheters is carried out for two reasons. The first, as alluded to above, is to downsize the catheter because of the "impasse" posed by severe subvalvular distortions.

The second reason occurs in the rare instance when there is a need to upsize the balloon catheter to one that is one size larger because of inadequate hemodynamic improvement. In such a situation, before exchanging for a larger catheter, it is vital that the initial catheter's final balloon diameter be re-measured and re-verified after its complete removal from the patient, particularly when it has been inflated beyond its nominal size. This precautionary exercise is essential because, not uncommonly, despite pre-testing, the balloon size is smaller than what it is supposed to be after *in vivo* usage. When this occurs, the original balloon catheter is retested to determine the actual volume of diluted contrast in the syringe necessary to achieve maximum balloon size (as mentioned above, the Inoue balloon tolerates about 1 mm in excess of its nominal size before rupturing), the original balloon catheter is reintroduced into the patient, and the dilatation process is repeated. However, if the balloon matches its predefined size, an exchange for a larger-sized catheter is made and dilatations with the larger balloon are performed. Failing to re-verify maximum balloon size before inflating a much larger balloon creates the risk of severe mitral regurgitation.

TECHNICAL TIPS

****Balloon "Popping" to the Left Atrium:** When the mitral valve has already been enlarged by dilatations, the balloon may occasionally slip into the left atrium during subsequent inflations with larger balloon diameters. To prevent the latter from occurring, the stylet is advanced far into the balloon segment to stiffen the catheter, and before the catheter is retracted to anchor the balloon at the orifice, the distal balloon is inflated to a diameter slightly larger than the previous one. As soon as the balloon assumes an hourglass configuration, the catheter is advanced slightly to prevent it from jerking out into the left atrium, and full balloon expansion is then executed. With this extra dilatation, although the mitral gradient may be unchanged, further shortening of the A2-opening snap interval and enhanced splitting of the commissures, as assessed by echocardiography are often observed.

The balloon "popping" signals enlargement of the mitral orifice with wide splitting of the commissures. It is usually encountered in patients with pliable, noncalcified valves and foretells excellent BMV results. However, suboptimal hemodynamic results are occasionally observed despite the balloon "popping" sign, especially in the presence of atrial fibrillation. In these cases, although the mitral valve with split commissures can be forced to accommodate the fully inflated balloon, the effective mitral valve area is, in reality, limited by the thickened and stiff leaflets, and by ineffective atrial contractions in the beating heart.

****Subsequent Valve Crossings and Dilations:** After the initial balloon inflation procedure, the catheter balloon is then withdrawn to the left atrium, while keeping the catheter tip to the left of the pigtail catheter (Figure 24-6, lower panel 2). The effects of the balloon dilation is assessed by observing the left atrial pressure wave forms and measuring the transmitral gradient and also by auscultation. If mitral regurgitation is suspected by the advent of a large v-wave and by a new or worsening systolic murmur, echocardiography or left ventriculography may be performed. In subsequent crossings of the mitral valve with the stepwise dilation technique, the stylet is inserted to the catheter tip and the catheter is advanced to deep seat the balloon (Figure 24-5, bottom panel 3). Thereafter, the above manipulations are repeated to cross the mitral valve for valve dilations.

****Catheter Entrapment at Atrial Septum:** When the septal puncture site is thick and tough, the catheter may be entrapped by the septum, thereby making manipulations difficult during subsequent attempts at crossing the mitral valve. The operator should be alert to the possibility of this entrapment when marked resistance is encountered at the septum during septal puncture. This vexing problem usually does not occur during the first crossing of the valve because, as alluded to earlier, the catheter is already deeply placed and coiled in the left atrium. However, entrapment may occur during subsequent crossings, when it becomes necessary to advance the catheter, which has been inadvertently withdrawn too far back into the atrium after valvular dilatation and caught at the thick septum. If the catheter cannot be advanced with the stylet inserted all the way to the catheter tip, a clockwise twist is applied to the stylet, directing the catheter tip posterolaterally to align it more or less perpendicular to the septal plane. The catheter may then be advanced forward together with the stylet (Figure 24-10, panels 3 and 4). If even this approach fails, the coiled-tip guide wire should be reinserted to facilitate deep placement of the catheter in the left atrium.

****Avoiding the Left Atrial Appendage:** Left atrial appendage thrombus may be unsuspected when BMV candidates are screened only with insensitive transthoracic echocardiography. To minimize the risk of inadvertent thrombus dislodgement and systemic embolism, the anterolateral appendage region must be avoided.

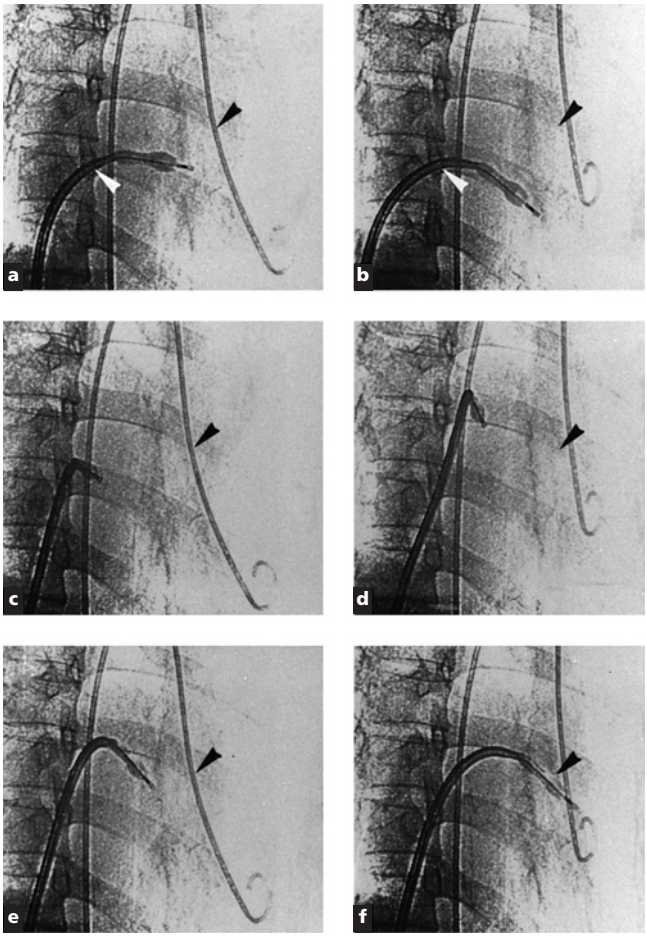


Figure 24-10 Sequence of steps to disengage a catheter entrapped in a thick septum. (a) Balloon catheter with stylet inserted to the tip is entrapped at the atrial septum (white arrowhead). (b) When the catheter is pushed, it does not advance forward, but rather turns downward. (c) Slight clockwise twist is made to the stylet to direct the catheter tip posteriorly. (d) The catheter, now aligned more or less perpendicular to the septal plane, can be effectively advanced farther into the left atrium. (e) Counter-clockwise twist is now made to the stylet to direct the catheter tip anteriorly. (f) The catheter is carefully withdrawn to bring its tip toward the mitral orifice (black arrowhead).

During balloon catheter manipulation performed under 30° right anterior oblique fluoroscopic view, the catheter tip should always be kept to the left of the pigtail catheter pre-placed in the left ventricle (Figure 24-6). After the precautions detailed below have become

rote, it may be possible to perform BMV safely even in the presence of left atrial appendage thrombi [33,34]. The alternatives are either to subject patients with appendage thrombi to mitral valve surgery, or to defer BMV for stable patients until resolution of the thrombi after warfarin treatment [34].

****Withdrawing the Catheter from the Ventricle:** After each balloon inflation procedure, in order to exert better control over the catheter tip and to prevent it from encroaching on the left atrial appendage, the stylet is advanced halfway into the balloon segment, and a slight clockwise twist to the stylet is applied as the catheter is withdrawn back to the left atrium. The balloon catheter, with its tip thus directed posteriorly, can then be safely pulled to the atrium by cautiously withdrawing the catheter and the stylet in steps. The catheter, however, should not be withdrawn too far during the process (see "Catheter entrapment at atrial septum" above). The stylet is then removed entirely from the catheter for left atrial pressure measurement, leaving the deflated balloon segment pointing vertically. Again, during hemodynamic measurements, care should be exercised to avoid accidentally pushing the catheter forward into the appendage.

****Subsequent Crossings:** The catheter, after having been withdrawn from the left ventricle, stands fairly straight up without looping. Thus, for the next crossings of the mitral valve, extra care is needed to keep the catheter to the left of the pigtail catheter. The stylet is carefully inserted to the catheter tip to bend the catheter downwards into a generous arch with the distal catheter segment oriented more vertically (Figure 24-6, lower panel 3). Then a counter-clockwise twist to the stylet is made, and the catheter is slowly withdrawn to direct the partially inflated balloon towards the mitral valve.

****Avoid Entry into the Left Atrial Appendage:** It should be noted that the catheter has a propensity to enter the left atrial appendage if the catheter tip is more horizontally oriented, and the stylet is pulled back too vigorously during a failed crossing attempt. To avoid this, the stylet should not be withdrawn too much during any manipulation in the left atrium, particularly when the sliding or posterior loop method is used.

****Minimizing Atrial Septal Injury:** Inherent in the antegrade BMV approach is the creation of an atrial septal defect. Fortunately, most of these defects are small and of no clinical consequence and they tend to close spontaneously with time.

To minimize the occurrence of these defects and to avoid septal avulsion, a number of precautionary steps should be adopted. First, prior to full balloon inflation, i.e. after the balloon has attained its hourglass configuration and is securely anchored at the mitral valve, the distal segment of the catheter shaft (between the septal puncture

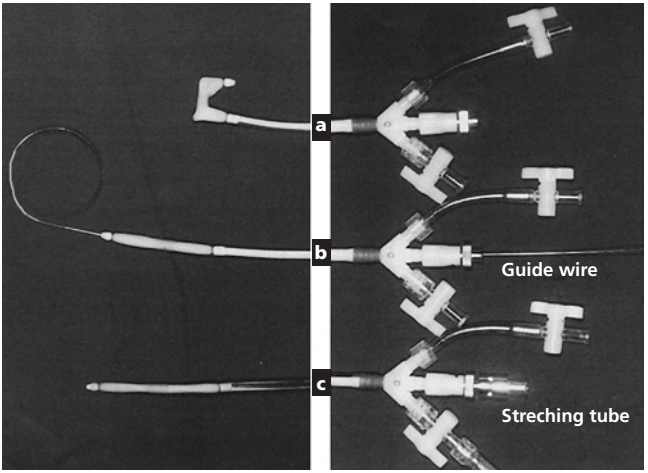


Figure 24-11 Kinked balloon. (a) Kinking of unsupported balloon segment occurs when inner tube is pushed to stretch the catheter balloon. (b) Balloon segment is supported by guide. (c) Balloon segment is supported by stretching tube.

site and the balloon) should be allowed to take on a gentle curve by releasing the tension exerted on the balloon catheter during its placement across the mitral valve. Second, it is mandatory to adhere to the standard practice of balloon slenderization during balloon passage across the septum (on both entry into and withdrawal from the left atrium). Third, before removing the stretched balloon catheter from the left atrium to the right atrium, the guide wire should be withdrawn, leaving only its soft distal floppy segment exposed. This may avoid "slicing" the septum by the stiff portion of the wire during withdrawal of the catheter/wire assembly.

****Bent Balloon Tip:** Kinking of the stretched balloon occurs during placement of the catheter in the left atrium if the guide wire is withdrawn before releasing the inner tube from its locked position. The balloon segment, unsupported by either the metal tube or the guide wire, may also be inadvertently bent by advancing the inner tube alone. Once the tip is bent, subsequent attempts at crossing the mitral valve with the catheter may be extremely difficult, if not impossible. In addition, it may be impossible to reinsert the guide wire to retrieve the balloon catheter from the left atrium. This problem may be overcome by (1) pulling the inner tube to its limit to shorten the balloon segment, (2) carefully inflating the entire balloon in the left atrium to sufficiently straighten the kinked inner tube, and (3) passing the guide wire through the deflated balloon to re-establish its natural shape (Figure 24-11).

INDICATIONS

The Selection of patients for BMV procedure is a complex decision involving consideration of multiple variables, including clinical profile, valve morphology and operator skill.

BMV procedure is best applied to patients with symptomatic moderate-to-severe mitral stenosis (mitral valve area $<1.5\,cm^2$) and favorable mitral vale morphology (pliable, non-calcified valve without significant subvalvular disease). In this subset of patients, BMV predictably yields excellent results and a low risk of resultant severe mitral re-gurgitation. BMV can be performed in asymptomatic patients with the favorable valve anatomy prior to non-cardiac surgery or planned pregnancy.

Inoue BMV is technically less demanding and clearly simpler to perform than the double-balloon approach, thereby engendering a shorter procedural and irradiation time [35]. This advantage is vital in pregnant patients where the hazards of irradiation to the fetus is of paramount importance, and for patients in pulmonary edema in whom swift and expeditious BMV is clearly desirable [29]. However, to minimize the hazards of fetal irradiation, it should be performed after the mid-second trimester, with adequate total abdominal and pelvic shielding, minimal use of fluoroscopy (by omitting diagnostic right heart catheterization and left ventriculography), only by interventional cardiologists skilled in the transseptal and valvuloplasty techniques.

The utility of BMV in patients with adverse valve morphology (calcified mitral valves and/or with severe subvalvular disease) is unclear and controversial [35]. Most operators contend that these types of patients are better served with surgery which often means mitral valve replacement because BMV in this setting is associated with an increased risk of complications and inferior long-term results [14,36,37]. In patients who pose a prohibitively high risk for valve surgery, BMV may be a better option than surgery, and may occasionally be the only therapeutic modality available for some of these patients. On the other hand, some experienced operators [38,39] advocate more liberal use of the procedure because of a low risk of major complications, in particular, resultant severe mitral regurgitation, and the procedure continues to offer sustained functional benefits in a substantial number of patients. Notwithstanding, it cannot be overemphasized that BMV in these patients can be technically demanding, and does require a higher level of technical skill and extra caution in executing the procedure.

CONTRAINDICATIONS

There exist two absolute contraindications in BMV: (1) severe ($\geqslant$grade 3+) angiographic mitral regurgitation, and (2) the presence of left atrial cavity thrombus. The treatment for patients with $\geqslant$grade 3+ mitral regurgitation is clearly that of mitral valve replacement. Patients with left atrial thrombus are subjected to open mitral commissurotomy or valve replacement, depending on the mitral valve status. Those patients with mobile thrombi in the left atrium are at a high risk of systemic embolism, and require urgent mitral valve surgery.

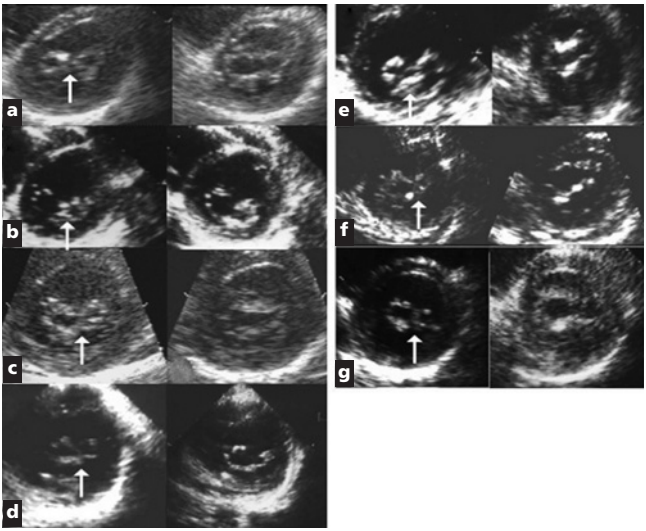


Figure 24-12 Transthoracic 2-dimensional echocardiograms of mitral valves before (left panels) and after (right panels) balloon mitral valvuloplasty in eight cases. In each case, two stenotic orifices are separated by a fibrous bridge tissue (arrow) (left panels). Balloon splitting of the fibrous septation results in a single, enlarged mitral orifice (right panels). (From [46].)

However, one may elect to administer long-term (3 to 12 months) warfarin therapy in patients with non-mobile thrombi in the left atrial cavity, if their clinical and hemodynamic status does not warrant immediate surgery, and the mitral valves are deemed suitable for BMV. Transesophageal echocardiography is deferred until thrombi resolution is observed by transthoracic echocardiography performed at 3-month intervals [34]. When transesophageal echocardiography confirms the absence of left atrial cavity thrombus, BMV can then be performed safely [23,22,24,40]. In our centers, the presence of thrombi confined to the left atrial appendage (without protruding into the left atrial cavity) is not a contraindication. BMV can be performed safely in this setting when performed with extra care using the Inoue balloon technique [23,33]. The risk of cardioembolism is low in this setting when Inoue BMV is performed by experienced operators [4].

Patients with lytic-resistant thrombi after 12 months of warfarin treatment should be considered for open surgical commissurotomy with direct visual clot removal.

TECHNICAL TIPS

*****Inoue Balloon Mitral Valvuloplasty in Double-Orifice Mitral Stenosis (Incomplete Bridge-Type):** Double-orifice mitral valve (DOMV) is a rare congenital anomaly characterized by

the presence of two mitral orifices, each possessing an independent chordal attachment of a papillary muscle [41,42]. DOMV may occur as an isolated anomaly or, more often, in association with other congenital anomalies like endocardial cushion defect, bicuspid aortic valve and coarctation of the aorta [42,43]. Echocardiographically, DOMV is classified into three types: complete bridge-, incomplete bridge-and hole-type [44]. The complete bridge-type is characterized by the presence of a fibrous tissue visible from the leaflet edge through the valve ring. In the incomplete form, however, the fibrous connection occurs only at the leaflet edge (Figure 24-12). In the hole-type, the secondary orifice with its subvalvular apparatus occurs in the lateral commissure and is visible only at the mid-leaflet level.

The isolated form of DOMV is more frequently observed in the bridge-type, and is usually associated with no significant hemodynamic abnormality. However, we have encountered 14 moderately symptomatic, middle-aged patients with stenotic DOMV of incomplete bridge-type. Their clinical presentations and physical findings were indistinguishable from those in rheumatic mitral stenosis. However, vigilance acquired from the experience in the first case contributed to expeditious echocardiographic identification of the incomplete bridge-type DOMV in the subsequent patients. All 14 patients, including seven previously reported patients [45,46], underwent Inoue balloon mitral valvuloplasty successfully.

Based on the experience in the 14 patients, several important technical tips and guidelines have evolved as follows.

1. Transseptal catheterization and left atrial placement of the balloon catheter can be performed in the usual manner.
2. Balloon crossing of posterpmedial orifice is simple and stepwise dilations of the latter orifice alone are sufficient to split the fibrous bridge between the two leaflets. Crossing attempts of the anterolateral orifice are futile because the orifice is located more cranially, making it impossible to align the distal balloon catheter with the orifice/apex axis.
3. Balloon catheter selection can be based on the height-derived reference size as in ordinary cases of mitral stenosis.
4. Stepwise dilations can be initiated at a balloon diameter of 4 mm less than the reference size. The procedure is terminated when the waist of the inflated balloon suddenly disappears, and echocardiography confirmed separation of the mitral valve septation resulting in a single enlarged orifice (Figure 24-12).
5. Application of the described Inoue balloon valvuloplasty should only be limited to patients with the incomplete bridge-type of DOMV.

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Chapter 25
Retrograde Percutaneous Aortic Valvuloplasty

Ted Feldman

GENERAL OVERVIEW

Although not commonly performed in many catheterization laboratories, balloon aortic valvuloplasty (BAV) has an important role in the management of patients who do not have an option for surgery with aortic valve replacement. BAV is a palliative procedure, and can be applied in appropriately selected patients with excellent relief from the symptoms of congestive heart failure associated with aortic valve stenosis. The AHA-ACC guidelines [1] recognize BAV as a Class 1 treatment for

*Basic; **Advanced; ***Rare, exotic, or investigational.
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Table 25-1 Indications for Diagnostic and Therapeutic Procedures

Definition	Class
General agreement procedure is useful/effective	I
Conflict evidence/divergent opinion	II
Weight of evidence/opinion in favor	IIa
Less well established	IIb
Evidence/agreement that procedure is not useful/harmful	III

Table 25-2 Balloon Valvuloplasty in the Young Adult (<21 years) with Aortic Stenosis and Normal Cardiac Output

Indication	Class
Angina, syncope, DOE with peak gradient >50 mmHg	I
Cath peak gradient >60 mmHg	I
New onset ECG changes at rest or with exercise with gradient >50 mm	I
Gradient >50 mm, patient desires competitive sports or pregnancy	IIa
Cath gradient <50 mm, no symptoms or ECG changes	III

Table 25-3 Balloon Valvuloplasty Adults with Aortic Stenosis

Indication	Class
Bridge to surgery in hemodynamically unstable high risk patients for AVR	IIa
Palliation in patients with serious comorbid conditions	IIb
Prior to urgent non-cardiac surgery	IIb
Alternative to AVR	III

children and young adults with aortic stenosis under the age of 21 years (Tables 25-1 and 25-2), and as a Class 2B indication among older patients with multiple comorbid conditions that preclude aortic valve replacement surgery (Table 25-3). In this author's own practice, one-third of these patients are nonagenarians, and half are octogenarians. Many have had prior coronary bypass or mitral valve replacement, or have comorbid conditions such as chronic lung disease or multi-organ compromise. These patients typically obtain about 1 year of improved symptoms, with diminished need for re-hospitalization for their symptoms [2]. It is clear that no overall survival benefit is conferred by this procedure in studies of groups of patients, though for individual patients it seems likely that some may derive this benefit [3–5].

STANDARD TECHNIQUE

The basic technique of retrograde BAV involves passing a catheter via the femoral arterial route retrograde across the aortic valve, placing a wire in the left ventricular (LV) apex, and then via the femoral sheath

passing a balloon into the aortic valve. Numerous special tips and tricks are critical to make this procedure successful.

TECHNICAL TIPS

****Necessity of Temporary Pacemaker:** Rapid RV pacing has been used to diminish LV stroke volume during balloon inflations, making ejection of the balloon by the LV much less of a problem than it has been in the past. Burst pacing at rates of 160–220bpm are used just before balloon inflation to produce an aortic BP of 50–60mmHg. Pacing is terminated as soon as the balloon reaches peak inflation, at which point the balloon is either withdrawn or ejected form the LV. A coordinated approach among the operator, assistant & catheterization lab staff is needed to utilize this approach. The pre-procedural electrocardiogram has great bearing on planning for the procedure. Patients with pre-existing bundle branch block or Inter-Ventricular Conduction Delay (IVCD) should have a temporary pacemaker placed for the procedure, or at very least have a venous sheath for pacemaker access. Complete heart block occurs infrequently but can be difficult to manage when it does occur in this group of patients. Since a right heart catheter is used, and the left ventricle is instrumented significantly by the balloon, both sides of the septum may be abraded with resultant loss of atrio-ventricular (AV) conduction. In addition, among patients with pre-existing conduction abnormalities, the displacement of aortic annular calcification by the balloon may impinge on the atrioventricular conducting system with exacerbation of heart block or pre-existing conduction delays. When complete heart block does occur, it usually resolves within 12 to 24 hours, but may be permanent. Infrequently, these patients need permanent pacemakers following the procedure, and I find it useful to warn most patients and families pre-procedure that permanent pacing in an occasional consequence of the effort.

****Vascular Access:** One of the most critical elements of the BAV procedure is assessment of the femoral artery. Fluoroscopic guidance of the initial puncture is critical, so that the common femoral artery is entered rather than the superficial femoral or the profunda femoris. Large sheaths needed for the balloons require the puncture to be above the femoral bifurcation. In between two-thirds and three-quarters of patients, the common femoral artery will be entered if the puncture is made at the level of the mid femoral head. Since the procedure is used principally in elderly patients, the location of the femoral crease is an unreliable landmark to guide the femoral puncture. Heavier patients may have two creases, and many thin elderly patients have lost the battle with gravity, and the crease has moved substantially caudal to the femoral head. Angiographic assessment of the femoral artery after the sheath is placed is also critical. I like to start with a 6Fr long sheath. If femoral angiography demonstrates too much atherosclerotic disease, a left internal mammary diagnostic catheter can be used to shoot over the top of the iliac bifurcation into the left iliac and femoral system to

see if they are suitable for the large valvuloplasty sheath. In addition, if the puncture is below the femoral bifurcation a sheath may be placed above the existing sheath on the right, or the left side might be used with angiographic guidance for the puncture.

****Prepatory Installation of Closure Device Suture:** Pre-closure of the puncture is important at this juncture within the order of the procedure [6,7]. The 6Fr sheath can be exchanged for a 10Fr Perclose device and sutures delivered into the artery. The sutures are of course not tied at this point. A wire is reintroduced into the Perclose delivery device, the delivery device backed out of the artery, and a 12Fr or 14Fr sheath passed back over the wire. It is especially helpful to use an extra stiff wire to allow passage of the large arterial sheath. It is my own preference to use a 30-centimeter long sheath. Sometimes this is not possible due to calcification or tortuosity of the iliac vessels, in which case a shorter sheath will suffice.

****Local Pain Management With Lidocaine:** The liberal use of lidocaine for local anesthesia is important to make passage of these large sheaths tolerable for the patient. At the same time, in the very elderly, especially among those with a history of prior stroke or seizure disorder, care must be taken not to create lidocaine toxicity. Changes in consciousness during the procedure may represent a variety of complications, but it is important to remember that lidocaine toxicity is among them and that lidocaine levels should be obtained any time there is a change in consciousness during a valvuloplasty procedure [8].

****Dobutamine for Low Cardiac Output:** After right heart catheterization and baseline pressure measurements, special consideration should be given for the cardiac output. Among patients with cardiac output less than 3 liters per minute, and certainly less than 2.5 liters per minute, it is useful to use dobutamine support. The decrease in blood pressure associated with balloon inflation may not be tolerated by patients with a low baseline cardiac output. It is my own practice to use a dobutamine infusion to improve cardiac output in those patients with low baseline pre-procedure, and to reassess valve area after the dobutamine infusion is started, for a new baseline measure [9].

CROSSING THE AORTIC VALVE

Crossing the aortic valve is an important challenge in this procedure. My preference is to use a catheter I have designed specially for this purpose [10]. There are two catheter shapes: the first with an angled design [type B] and the second a curve design [type A]. Each is constructed in small (A, B), medium (A1, B2) curve lengths. The displacement from the shaft to the catheter tip, or the "reaching distance " of the catheter, measure 4 cm, 5 cm, and 6 cm for the small, medium, and large curves, respectively. A moveable core straight wire can be used to change the angle of the catheter. When the aortic root is very small in diameter, the catheter can be straightened with the wire and

formed into shape similar to a right Judkins curve. The angled design catheter can reach the center of the aortic root in most patients unless the left ventricle and aortic meet at an extremely acute angle. The curve catheter is designed to reach a more acutely angled left ventricular chamber but is slightly more difficult to maneuver into the left ventricle in some patients [10].

TECHNICAL TIPS

****Manipulating the Catheter:** The catheter is selected based on the fluoroscopic appearance of the width of the aortic root. It is inserted into the aortic root with a straight wire and rotated clockwise to direct the tip of the catheter toward the center of the aortic root. A moveable core straight wire can be used to change the angle of the catheter, allowing the operator to scan the surface of the aortic valve. Wires with a tapered moveable core are not stiff enough for this purpose. The wire is initially made extremely soft by withdrawal of 3–4 inches of moveable core, which allows the tip to assume its formed curve completely. The catheter tip was positioned over the center of the aortic valve as determined from the appearance of the heavily calcified leaflets on fluoroscopy. The straight wire is passed back and forth until it crossed into the aortic valve. Occasionally, a hand injection above the valve will help define the central area of the commissures. Of course, the wire may cross at some distance away from the commissures, but the central point represents the best chance for success. The catheter is advanced over the wire into the left ventricle and the wire withdrawn. In all patients, a small or medium length catheter is selected initially. Based on the direction of the wire, subsequent catheter choices were made. It should be noted that both catheters can be passed into the left ventricle with much greater ease than most coronary artery catheter.

****Wiring:** After the left ventricle has been entered and hemodynamic measurements confirm the severity of aortic stenosis [11], a 260-centimeter long 0.038-inch exchange wire must be used to allow exchange or a valvuloplasty balloon. The wire must be as stiff as possible, but with the tip curled to make it less dangerous for left ventricular apical perforation. It is helpful to grasp the wire over the end of a hemostat and "Christmas ribbon" the end into a ram's horn shape with multiple concentric coils to protect the LV apex from wire trauma or perforations. The stiffest possible wire available is the best wire. It is critical to have a firm rail to allow the balloon to traverse tortuous anatomy in the aorta, and to have the support to keep the balloon in position in the aortic valve during balloon inflations. The assistant helping maintain wire position is as critical to the success of the procedure as the principal operator is.

BALLOON MANIPULATION

Once a balloon has been passed into the aortic valve orifice, maintaining its position is challenging. In patients with poor left ventricular

function there is less of a tendency for the balloon to be ejected by the ventricle. When left ventricular systolic performance is preserved, the balloon "watermelon seeds" back and forth during inflations.

TECHNICAL TIPS

****Balloon Inflation:** It is useful to partially inflate the balloon in the ascending aorta above the valve prior to trying to engage the valve, so that less inflation is needed to achieve adequate inflation within the valve orifice. If the balloon is fully inflated in the valve orifice and continues to move back and forth, it may be undersized. The balloon "locks" in the valve when it is fully inflated and delivery adequate dilating force to displace the leaflets. If a first balloon is too small, it is often necessary to size up the sheath. A 20-millimeter diameter balloon catheter will require a 12 or 13Fr sheath. It is my own practice to use a 12.5Fr sheath for this purpose. A 23-millimeter balloon requires a 14Fr sheath.

****Balloon Preparation:** Careful preparation of the balloon is necessary, because it is common for the balloon to rupture during inflations in the calcified aortic valve. Great care to remove all the air during the preparation process is essential. Preparation and balloon inflation is easiest if the contrast is diluted as much as possible. A ratio of 7:1 will allow the balloon to be visualized fluoroscopically, but inflated and deflated with the least difficulty. The contrast is ideally an old-fashioned ionic contrast, since these agents are less viscous than low osmolarity contrast. It is my practice to use a 50cc bottle of contrast, diluted with an additional 350 cc of saline to a total volume of 400 cc. Many of the basins used on the cardiac catheterization laboratories back table are graduated. There is thus no need to use a syringe to top off the total volume, since the graduated basin allows one simply to pour saline into the 400 cc mark after the contrast has been placed in the bowl.

****Set Up for Balloon Inflation:** The set up for balloon preparation includes a short pressure tube to the inflate lumen, connected to a high-pressure stopcock. A 60-cc syringe is attached to one arm of the stopcock, and a 10-cc to the other arm. If the 60-cc syringe is used to inflate the balloon, it is not possible to deliver adequate force to fully inflate the balloon [12]. Once the balloon has been inflated as much as possible with the 60-cc syringe, the stopcock can be switched to allow the 10-cc to finish the inflation, or "boost" the total inflation volume. If this is done on the back table, you will note that the balloon clearly increases in inflation volume when the booster syringe is used to fully inflate it. Thus, in vivo the balloon is passed across the valve and inflated as much as possible with the 60-cc syringe and then the stopcock is flipped and the 10 cc additional inflation used to maximize the balloon diameter.

****Balloon Deflation:** The strategy of balloon deflation is as important as the inflation. Once the balloon is fully inflated in the

valve there is a precipitous decrease in systemic blood pressure, and usually significant ventricular ectopy. Rather than waiting for the balloon to deflate to withdraw it from the valve, it can be pulled back from the valve orifice into the aortic root while it is still inflated, or just as the process of deflation begins. This allows a restoration of antegrade blood flow before balloon deflation is even initiated. It is easier for patients to tolerate this very brief effective cross clamping of the aorta than if the entire inflate deflate cycle were performed within the valve orifice. When the balloon is withdrawn into the aortic root it is possible for the arch vessels to be obstructed, so care must be taken to avoid covering the carotid origins.

MANAGEMENT OF HYPOTENSION

The management of hypotension during the procedure is one of the greater challenges [13,14]. The blood pressure inevitably falls during balloon inflations. In most cases there is a steady recovery of systolic pressure immediately following balloon deflation, and when the valve is successfully opened there is a rebound or increase in aortic peak systolic pressure above the baseline. Pressure can be monitored via the sidearm of the 12Fr sheath. If the pressure does not recover rapidly after a balloon inflation, it is unwise to proceed with further inflations. This represents left ventricular depression that may require support with pressors, sometimes for as long as a day or two.

TECHNICAL TIPS

****Differential Diagnoses of Hypotension:** Other causes of hypotension must be considered. Since the arterial sheath is large, femoral hematoma, retroperitoneal bleeding, or even venous bleeding from the venous access site must be considered. Patients with significant anemia prior to the procedure should be considered for transfusion so that they have a "full tank" before the procedure begins. If they are borderline, or there is some relative contraindication to transfusion consider obtaining a type-and-screen or type-and-cross match so that blood will be readily available if needed. Vagal reactions from insertion of the large sheath may occur though they are rare. This should be considered only after bleeding has been carefully evaluated and excluded. During the balloon inflations, the guidewire is forced into the left ventricular apex, and the tip of the balloon may also impact on the apex with considerable force. Ventricular perforation is another important consideration for hypotension. Echocardiography should be used liberally in the catheterization lab to exclude this possibility when hypotension is persistent. In the worst cases, the aortic annulus may be ruptured, or a valve leaflet avulsed with catastrophic results. Hypotension associated with these latter complications is usually fatal, and cannot be reversed.

****Hypotension Caused by the Wire:** In some cases, the ventricular ectopy produced by the wire in the ventricle for a prolonged

period of time is not tolerated, and is another source of hypotension. Reshaping the wire, or repositioning the wire may give some relief from persistent ventricular ectopy. In some cases the procedure cannot be performed due to ectopy. I have encountered a patient who had ventricular fibrillation requiring DC countershock each time the wire was introduced into the left ventricle. After two attempts it became clear that it was not feasible to perform aortic valvuloplasty for this patient.

SHEATH REMOVAL

Sheath removal is an important challenge in the management of these patients. The large caliber femoral artery sheath has been associated with transfusion rates in about one-quarter of patients in the past, and the need for vascular surgical repair in 5–10%. Recently, the use of percutaneous suture closure has been described as an adjunct to sheath removal. Pre-closure using a 10Fr Perclose device prior to insertion of the 12 or 14Fr sheath has been successful in almost 90% of patients with an almost complete elimination of the need for blood transfusion following this procedure. For those patients in whom preclosure is unsuccessful, or in whom femoral anatomy does not allow its use, it is critical to use a pneumatic compression device such as the RADI FemoStop. Manual compression by itself is extremely difficult, since prolonged compression for this large sheath size is necessary. The rigid clamp devices cannot be monitored adequately and may result in either inadequate hemostasis, or over compression of the vessel with the potential for thrombosis. The FemoStop device can be applied with a graded pressure, so that initially it is inflated to about the level of systolic pressure. Since the device is transparent, hemostasis can be visualized directly. The pressure can be decreased 10 mm to 20 mm every 10–30 minutes depending on the activated clotting time, until hemostasis is achieved. Another benefit of the FemoStop device is that it helps keep the patient immobile during the period of vascular compression.

POST PROCEDURE MANAGEMENT

Other than management of the punctures, the major issue is whether left ventricular depression has been engendered by the balloon inflations. Patients who develop pulmonary congestion during the valvuloplasty procedure require special monitoring, and may need inotropic support and intensive heart failure management for 1 to 2 days post procedure, until their left ventricular performance recovers.

Long-term follow-up requires no more than surveillance for recurrent symptoms, and periodic echocardiographic examinations to monitor the transaortic valve pressure gradient. An important consideration in follow-up is the status of other valve lesions. When the aortic valve is successfully opened, afterload reduction with often result in improvement in the associated mitral regurgitation that these late-stage aortic stenosis patients often have.

Among patients who have recurrence of the stenosis, repeat valvuloplasty may be accomplished with a high expectation for success [15]. I rarely offer repeat procedures to patients who re-stenose quickly, within 6 to 8 months following the initial procedure. For those who achieve a year or more of clinical benefit, repeat procedures can be performed even three or four times, though the resultant valve areas are usually no better than the first procedure.

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Chapter 26
Percutaneous Implantation of Aortic Valvular Prosthesis (Self-expanded Prosthesis)

Eberhard Grube, Lutz Buellesfeld

GENERAL OVERVIEW

Open heart surgery with mechanical or porcine bioprosthetic valve replacement is the current reference standard therapeutic approach for severe aortic valve disease, offering symptomatic relief and improving long-term survival in most patients. However, the etiology of aortic stenosis in the Western population is primarily degenerative, and patients are typically elderly with multiple co-morbid conditions which increase surgical risk and peri-procedural morbidity. In high-risk patients with baseline features such as left ventricular failure, concomitant coronary artery disease, prior bypass graft surgery, chronic obstructive pulmonary disease and/or advanced age, operative mortality ranges from 10% to 50% [1–3]. Percutaneous treatment of aortic valve disease with implantation of a stent-based valve prosthesis has been evaluated in animal models over the past decade [4–9].

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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In 2002, Cribier *et al.* [10] performed the first human implantation of a balloon-expandable aortic valve prosthesis (percutaneous valve therapies, PVT) in a patient with aortic valve stenosis considered inoperable due to severe co-morbidities. Initial reports with this new percutaneous valve have been promising [11–14], though the optimal device and procedural technique are evolving. A self-expanding aortic valve prosthesis intended for retrograde delivery across the aortic valve has been developed by CoreValve. The design of this stent may simplify the implantation procedure, theoretically reduce paravalvular leaks, and facilitate treatment of aortic insufficiency as well as stenosis. Following evaluation in animal models [15,16], this device was subsequently successfully implanted in humans [17,18], and its use was expanded.

DEVICE CHARACTERISTICS

The CoreValve aortic valve prosthesis consists of a trileaflet bioprosthetic pericardial tissue valve, which is mounted and sutured in a self-expanding nitinol stent. The inner diameter of the valve is 21 mm. The prosthetic frame (stent) is manufactured by laser cutting of a nitinol metal tube with length of 50 mm. The lower portion of the prosthesis has high radial force to expand and exclude the calcified leaflets and avoid recoil; the middle portion carries the valve and is constrained to avoid the coronary arteries; and the upper portion is flared to fixate the stent in the ascending aorta and provide longitudinal stability. Three device generations (Figure 26-1a,b) have been developed so far: the 25Fr/21Fr and18Fr devices. The first generation device utilized bovine pericardial tissue, and was constrained within a delivery sheath of 25Fr diameter. The second and third generation devices incorporated a porcine pericardial tissue valve within a 21/18Fr diameter

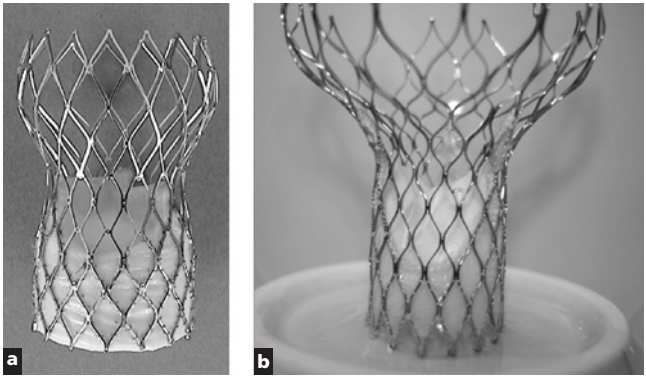

Figure 26-1 The CoreValve bioprosthesis. (a) First generation, (b) third generation.

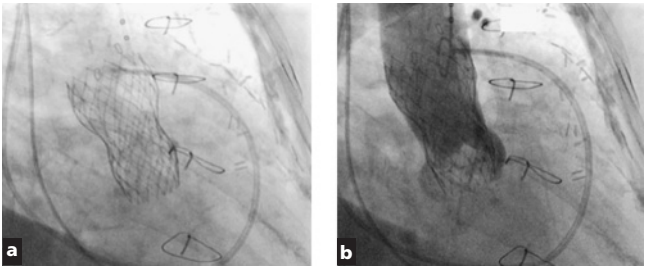

Figure 26-2 Implanted CoreValve prosthesis with and without contrast media.

sheath, the reduced profile allowing access through smaller diameter vascular beds. These devices are also characterized by a broader upper segment for more secure fixation in the ascending aorta, allowing inclusion of patients with an ascending aorta diameter up to 45 mm. Due to the small French size of the third generation device, the complexity of the procedure has been impressively reduced over the past 12 months. Nowadays, the prosthesis is implanted without hemodynamic support, anesthesia or surgical access preparations. Now, it is almost a stent-like procedure with a duration of approximately 30 minutes in experienced hands (Figure 26-2).

CLINICAL RESULTS

More than 100 patients have been treated with the CoreValve prosthesis, mainly with the second generation device. The results of the first 25 patients have been published by our group in 2006. Device and procedural success were achieved in 22 (88%) and 21 (84.0%) patients respectively. Successful device implantation resulted in a marked reduction in the peak and mean aortic valve gradients (to 16.5 $\pm$ 9.2 mmHg and 10.8 $\pm$ 4.2 mmHg respectively, both $p < 0.0001$). The mean aortic regurgitation grade was unchanged (0.92 $\pm$ 0.7 pre procedure and 0.73 $\pm$ 0.6 post procedure, $p = 0.27$). Major in-hospital cardiovascular and cerebral events occurred in 7 (28.0%) patients, including mortality in five patients (20.0%). Among 18 patients with device success surviving to discharge, no adverse events occurred within 30 days after leaving the hospital, valve function remained stable, and clinical status improved from New York Heart Association class III at baseline in all patients to class I in 5 patients (27,7%) and class II in 13 patients (72.3%).

In summary, percutaneous implantation of the self-expanding CoreValve aortic valve prosthesis in high risk patients with aortic stenosis with or without aortic regurgitation is feasible, and when successful results in marked hemodynamic and clinical improvement.

Patient Selection: The optimal patient selection is crucial for a successful percutaneous valve procedure. Particularly with regard to

the vascular access, the morphology of the ascending aorta as well as the native valve.

Vascular Access: Initially, vascular access was obtained by surgical cutdown of the common iliac artery, subclavian artery, or common femoral artery. Due to the reduction of the device dimensions, there is now no need for surgical preparations in properly selected patients. Access screening should include duplex and CT angiography to exclude heavy calcification and ensure a vessel diameter of >7 mm in the femoral/iliac artery. The device can be usually inserted in the common femoral artery using a 18Fr sheath. (Alternatively, the device can be inserted via the subclavian artery after surgical cut-down.) Prior to sheath insertion, we place two Prostar sutures which easily close the puncture site at the end of the procedure. Patients with heavy calcifications of the aorta should be excluded as this might prevent the device passage.

Ascending Aorta: Since the prosthesis is anchoring in the ascending aorta, the dimensions of this vessel segment are critical. Using the current 18Fr device, the diameter of the ascending aorta must be ,45 mm measured by CT 3 cm above the annulus. Larger aortic dimensions would lead to an unstable valve position with the risk for valve dislodgement. Larger prostheses are currently under development, which will finally allow the inclusion of patients with larger ascending aortas in the future.

Native Aortic Valve: The morphology of the native aortic valve is even more important than anything else. In order to avoid paravalvular leaks, the annulus of the native valve must be ,24 mm. Otherwise, the prosthesis does not completely fill out the valve area. Leaflet calcification is not an exclusion criterion, and usually seen in elderly patients with aortic stenosis as part of the pathogenesis. However, severest leaflet calcifications might complicate the valve positioning. Appropriate balloon predilation certainly helps to overcome this problem. In addition, we have never experienced significant residual stenosis due to an underdeployed valve prosthesis which was not able to press calcified leaflets completely to the vessel wall.

Hemodynamic Support: In the first series, the procedure was performed with the patient under general anesthesia with transesophageal echocardiographic guidance and with extracorporeal percutaneous femoro–femoral bypass. Extracorporal circulatory support was activated just prior to device placement across the native valve position and stopped immediately after withdrawal of the delivery catheter and confirmation of adequate valve function. It was requested per protocol as a safety measure establishing a hemodynamic standstill of the heart which facilitated the valve positioning. In the course of the series with growing experience, the extracorporal circulation was first replaced by rapid ventricular pacing and finally abandoned totally. Implantation of the current 18Fr device can be performed so quickly and reliably, that there is no particular hemodynamic support needed.

Wire Selection: We are using an 0.035" Amplatz super stiff guidewire with a kind of pigtail tip in the left ventricle which we manually form. This provides sufficient back-up for the procedure while minimizing the risk for ventricular perforations.

Balloon Predilation: Balloon valvuloplasty prior to device placement is generally recommended to prepare the native valve for device insertion.

Device Positioning: Correct positioning of the device should be mainly confirmed by repeated supraaortic angiograms using a pigtail catheter which we usually insert by radial or brachial access. A RAO/caudal view displays the valve area optimally in a perpendicular view. In addition, transesophageal echocardiography can be helpful for device positioning when used.

Device Release: The device handle allows a fast and slow device release. We are using usually the screw of the handle for slow release for the first few millimetres followed by a fast release of the first 2/3 of the valve prosthesis. After deployment of 2/3 of the prosthesis, the device is almost fully expanded in the distal part with establishing of a normal prosthetic valve function while the proximal tip is still anchored in the catheter; this allows a pull back of the device as a kind of fine-tuning as well as a complete bail-out retrieval of the valve by withdrawal of the prosthesis all the way back into the sheath. However, at this stage, the prosthesis cannot be introduced in the catheter by operating the handle. If the position of the device is satisfying, the prosthesis can be released completely and the catheter is withdrawn.

A final angiogram is performed to confirm the final result. If there is still a paravalvular leak, a post-dilation can be performed. However, paravalvular leaks will dissolve over time at some degree due to the self-expanding properties of the stent frame.

Adjunctive Medication: Aspirin (100 mg/day) is given pre-procedure and continued indefinitely. A 300 mg loading dose of clopidogrel is given prior to the procedure and continued for 12 months. During the intervention, the patient receives intravenous heparin to achieve an activated clotting time (ACT) .250 s.

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Chapter 27
Percutaneous Implantation of Aortic Valvular Prosthesis

John G. Webb, Lukas Altwegg

GENERAL OVERVIEW

Surgical valve replacement has a proven role in the management of severe aortic stenosis (AS) improving both symptoms and survival [1].

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Good surgical candidates have a low risk of mortality with conventional aortic valve surgery in experienced hands. However, in many patients with co-morbidities, mortality and morbidity risk may be excessive. In such patients, particularly the elderly, conventional surgery is often not offered, or is declined.

Percutaneous valve implantation was first described by Andersen in 1992 in an animal model. The first human percutaneous aortic valve implantation was described by Cribier in 2002. An antegrade transvenous approach was initially utilized due to the large profile of the stent-based prosthesis [2]. However, due to complexity and associated risk the antegrade procedure did not find favor. Subsequently we described a technique of retrograde aortic valve implantation from the femoral artery. The retrograde approach has found favor as the currently preferred method of percutaneous valve implantation. Early experience with the retrograde transarterial approach to implantation of the balloon-expandable Cribier Edwards™ (Edwards Lifesciences Inc, Irvine CA) valve is encouraging and is discussed below (Figure 27-1) [3–5].

INDICATIONS AND PATIENT SELECTION

Most patients with severe symptomatic aortic stenosis are at least potential candidates for percutaneous aortic valve implantation. However, at this time, the risks and durability of the percutaneous procedure are less well understood than those associated with conventional surgical aortic valve replacement. In absence of more experience and data it is our view that the procedure should be restricted

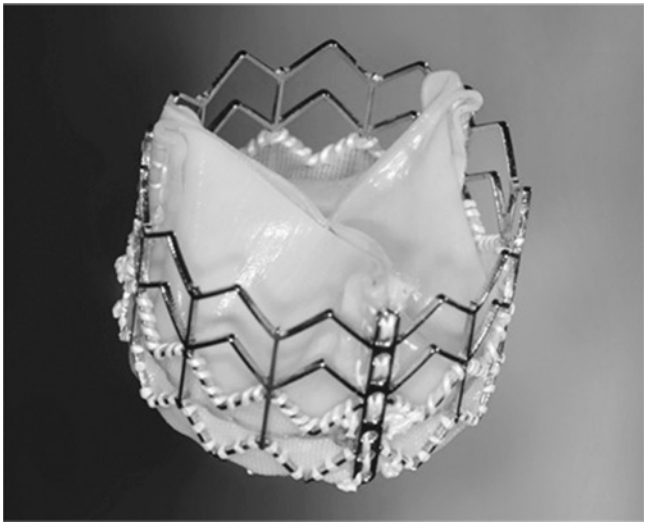

Figure 27-1 The Cribier-Edwards™ valve consists of a stainless steel stent with pericardial leaftlets and a fabric sealing cuff.

to symptomatic patients in whom the risks of mortality or morbidity with conventional surgery are excessive. Currently we consider a logistic EuroSCORE predicted surgical mortality of >20% as a reasonable level at which to consider a percutaneous procedure. What constitutes excessive morbidity, particularly in the very elderly patient, is even more controversial but must represent a consensus of the patient, cardiologist and surgeon. In addition to a careful history evaluation begins with a transthoracic echocardiogram and cardiac catheterization.

TECHNICAL TIPS

***Echocardiogram: Guessing the Occlusion of the Coronary Artery by the Bulky Valve Leaflet** Echo is invaluable in assessing the severity of aortic stenosis, hypertrophy, left ventricular function and other valvular disease. If the aortic valve is particularly bulky it is important to consider whether stenting might result in a calcific nodule being displaced such that it occludes the left coronary ostium. If this is a concern we exclude the patient or observe the leaflet during valvuloplasty prior to committing to valve implantation. The aortic annulus size is carefully measured to facilitate prosthesis sizing (Figure 27-2).

***Cardiac Catheterization: Indication for Coronary Artery Intervention Before Valve Deployment** We perform single stage left and right heart catheterization, coronary angiography, ascending aortography and descending aortography with imaging of

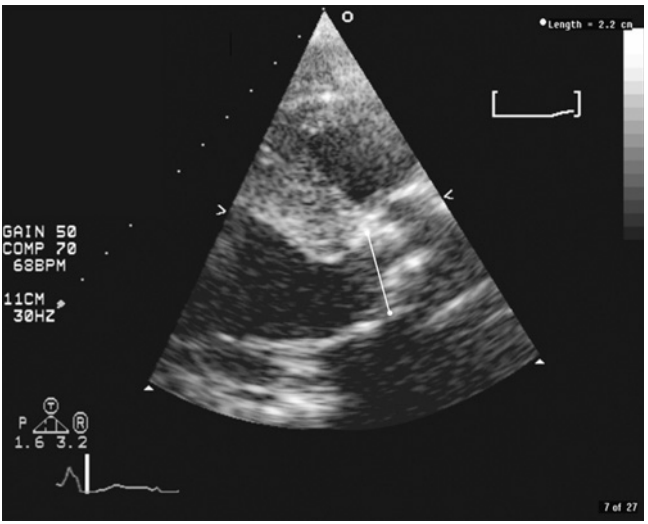

Figure 27-2 Transthoracic echocardiogram. The aortic annulus is measured at the insertion point of the valve leaftlets. This differs from measurements of the left ventricular outflow tract.

both iliac and femoral arteries. Left ventricular angiography is usually avoided. If the patient is not being considered for surgical revascularization coronary angiography is focused on assessment for the need for revascularization prior to a percutaneous procedure. In some patients with renal dysfunction two views of the left and one of the right artery may be adequate. In some patients presenting with angina angioplasty alone may be all that is needed, with valve therapy deferred. In others coronary anatomy may argue strongly that a surgical option be reconsidered. On occasion we will perform angioplasty to reduce the potential for ischemic left ventricular dysfunction during valve implantation. For the most part we have found conservative management of coronary disease reasonable in patients being considered for aortic valve implantation. We do not favor ad hoc procedures in patients with aortic stenosis.

****Measurement of the Aortic Annulus Diameter:** Unlike surgery, where the aortic annulus can be physically measured, the interventionalist is dependent upon less direct means. We start by measuring the aortic annulus diameter in the parasternal long axis view at the point of leaflet insertion (Figure 27-2). This differs from more commonly reported left ventricular outflow tract measurement. If the annulus appears too small or large for the currently available prostheses confirmatory transesophageal study may be considered. We have found angiographic measurements unreliable and neither CT nor MRI annular measurements are sufficiently standardized at this time. Our final determination is based on transesophageal echo imaging at the time of implantation. We currently consider an annulus of 18–22 mm suitable for a 23 mm diameter prosthesis and an annulus of 21–25 mm suitable for a 26 mm prosthesis.

****Angiography of the Aortic Root: Looking for Landmarks for Prosthesis Positioning and Unfavorable Aortic Features** Angiography of the ascending aorta is performed in the magnified anterior view with 20–30 mL of contrast over 2 seconds. Attention is paid to the presence and location of leaflet and annular calcification as landmarks for prosthesis positioning. The presence of very bulky aortic valve leaflets in close proximity to the left main artery is a concern should the leaflets obstruct coronary flow when stented open. An unfolded aorta with a horizontal root and vertical valve plane raises concerns about retrograde crossing of the valve coaxial valve deployment. During prosthesis positioning it is important to image the native valve in profile. If the valve cusps do not appear to be seen in the same plane then alternate fluoroscopic views should be sought.

****Angiography of the Ilio–femoral Arteries:** Attention to detail is critical in the assessment of the adequacy of vascular access when dealing with sheaths of up to 24Fr in size. We place a pigtail just below the renal arteries. If placed higher opacification of the femorals is typically inadequate. If placed too low aorto–iliac bifurcation disease may not be visualized. With an injection of 40 mL over 2 seconds

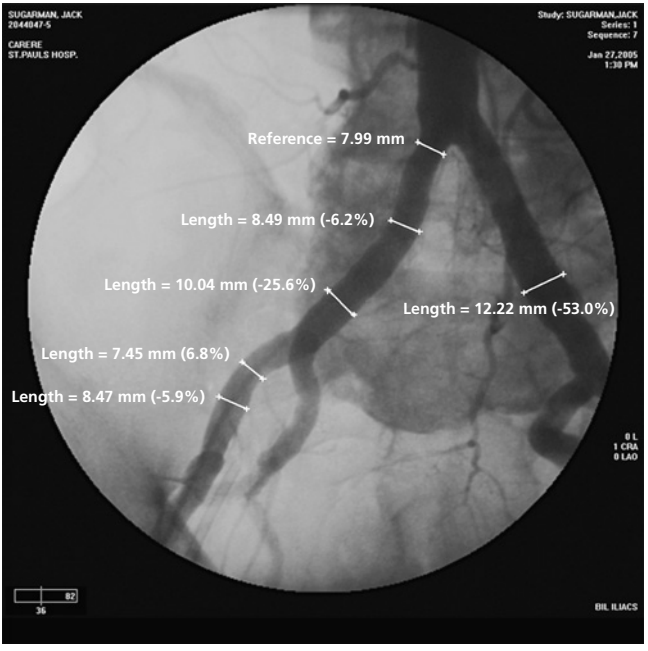

Figure 27-3 Careful examination of the iliac and femoral arteries is important for placement of the large sheath required. We image both sides from the aortic bifurcation to the femoral head.

and rapid panning adequate assessment of both iliacs and both femorals is usually possible (Figure 27-3).

We measure the diameter of both iliac and femoral arteries while paying particular attention to areas of narrowing. The outer diameter of 22 and 24Fr sheaths are approximately 8 and 9 mm respectively. Shorter segments with a diameter ~1 mm or less are adequate in the absence of excess calcification or tortuousity.

It is important to visualize the femoral artery at the level of the femoral head as this is where vascular entry will be made. Generally a puncture at the level of the middle of the femoral head is desirable and the inguinal skin crease is a poor landmark. We routinely use fluoroscopy of the femoral head to chose the site of puncture based on the earlier femoral angiogram hoping to puncture the common femoral above the profundus and below the retroperitoneal inferior hypogastric artery.

AORTIC VALVULOPLASTY

Patient Preparation: Patients should be stabilized as much as possible prior to the procedure. Frequently this means diuresis to minimize edema or pleurocentesis to remove large effusions. We routinely recheck

Table 27-1 Medications Commonly Used in Percutaneous Aortic Valve Implantation

Medication	Dose	Use
ASA	81–325 mg daily	Indefinitely
Clopidogrel	75 mg daily	Before and 3 months after implantation
Vancomycin	1 g IV	Pre-procedure
Heparin	ACT >250 seconds	During procedure
Norepinephrine	Bolus: 100 μg IV; continuous administration: 0.5–30 μg/min	To maintain coronary perfusion pressure
Phenylephrine	Bolus: 50–100 mg IV q5 min prn; continuous administration 0.1–0.18 mg/min	To maintain coronary perfusion pressure
Warfarin	INR ~ 3	Post-procedural for atrial fib or thromboembolism prophylaxis*

*The optimal anticoagulation regimen is unknown.

Table 27-2 Standard Catheterization Laboratory Set-up for Percutaneous Aortic Valve Implantation

Hats and masks
Arm boards
Defib pads on lateral chest or suprasternal to avoid obscuring the aortic valve
Overhead monitors (hemodynamic/rhythm monitor, live fluoro, review fluoro and live echo)
Imaging system and shields positioned to facilitate access to the head and radiation protection for anesthesia and TEE
General anesthesia
TEE probe placed
Jugular venous and radial arterial line for anesthetist
Sheath in the common femoral artery selected for the large sheath
Pigtail catheter in the ascending aorta from contralateral femoral artery
Pacemaker wire in the right ventricle from contralateral femoral vein
Two manifolds for gradient measurement

hemoglobin, electrolytes, creatinine and coagulation status immediately prior to the procedure. We find anesthetic pre-assessment a few days prior to the procedure helpful to identify any outstanding issues. Elderly patients with comorbidities may benefit from pre-procedural social work and nursing assessment as to their needs after the procedure. We typically premedicate patients with aspirin, clopidogrel and either 1 g of intravenous vancomycin or cefazolin. Medications commonly utilized are detailed in Table 27-1.

Equipment Set-up: Percutaneous aortic valve implantation requires excellent fluoroscopic imaging in either a cardiac catheterization laboratory or ideally a hybrid operating room setting. Operating room-like

sterile precautions are desirable to minimize the risk of infection of the implanted prosthesis or femoral access site should operative closure be required. Anesthetic, echocardiographic and vascular surgical supports are required. Our routine cardiac catheterization laboratories set up is detailed in Table 27-2. The equipment required for valve implantation should be assembled as it may not be readily available in the routine cardiac catheterization laboratory setting (Table 27-3).

Anesthetic Management: Although percutaneous aortic valve implantation can be performed with conscious sedation and local anesthesia, we prefer general anesthesia with endotracheal intubation, as it facilitates sheath placement and removal, airway control, transesophageal echocardiography, burst pacing, defibrillation when necessary and reduces patient discomfort. The use of volatile anesthetic agents and non-depolarizing muscle-relaxants typically allow early extubation already in the catheterization laboratory. Patients should be treated with a level of intensity as if they were undergoing surgery. Monitoring should include a peripheral and central venous line, a radial arterial line, oximetry, and capnography.

TECHNICAL TIPS

***Arterial Cannulation:** The screening angiograms are reviewed. We select the side with the larger, less tortuous, less diseased femoral and iliac artery. We note the relationship of the common femoral head. A desirable puncture site is identified that is free of disease, below the peritoneal space as demarcated by the inferior hypogastric artery and above the bifurcation into superficial and deep femoral

Table 27-3 Standard Equipment Needed for Percutaneous Aortic Valve Implantation

Manifolds
Two, for gradient measurement
Sheaths
7F × 3 (femoral artery × 2, femoral vein × 1)
14–16F for valvuloplasty
Wires
J wire, 0.035
Straight wire, 0.035
Straight wire, hydrophilic, 0.035 (Glidewire™, Terumo Inc.)
Exchange wires, 0.035 (Amplatz Extrastiff™, Cook Inc and Meier™, Boston Scientific Inc)
Catheters
Amplatz Left 1 diagnostic (for crossing the aortic valve)
Pigtail (for aortography and valve positioning)
Valvuloplasty balloon
20 and 23 mm diameter × 3 cm long (Z-Med™, Numed Inc)
Contrast 10% for valvuloplasty balloon
Pacemaker
Generator capable of rate of >200 per minute
6F lead and sterile cable

arteries. The relationship of the ideal puncture site to the bony femoral head is noted. Usually this corresponds to the middle or upper third of the femoral head.

A radio-opaque hemostat is placed over the proposed puncture site. Fluoroscopy is utilized to assure the hemostat overlies the bony landmark previously identified. When the inguinal skin crease is used as a landmark fluoroscopy will usually show that the puncture would have been too low, particularly in obese or elderly patients. Given the large sheath size subsequently required a good puncture is important.

****Burst Pacing:** A right ventricular pacemaker is placed via the femoral vein opposite the site selected for placement of the large valvuloplasty sheath. We prefer to avoid heparin until well after pacemaker placement due to the risk of pericardial bleeding. We assure a good threshold of <1 mV and set the output at 10 mV with a back up rate of 50 per minute. Burst pacing is commonly initiated at 220 per minute for 10 seconds (Figure 27-4). Incomplete capture is common and generally associated with a mild reduction in blood pressure. After a period of recovery we decrease the rate incrementally by 20 per minute until 1:1 capture is achieved. In most patients 1:1 capture is achieved at a rate of 160–220 which invariably results in profound hypotension in the setting of aortic stenosis. Hypotension and reduced pulse pressure are associated with reduced transvalvular flow and cardiac motion. This reduces flow directed movement of the valvuloplasty balloon and facilitates accurate prosthesis deployment. We have described burst pacing for valve implantation in detail elsewhere [6].

***Crossing the Valve:** We typically place an AL1 diagnostic catheter just above the valve utilizing a standard J wire. The J wire is replaced with a straight tip wire. We generally use a coiled wire although a hydrophilic coated wire may be useful at times. A straight wire is carefully aimed at

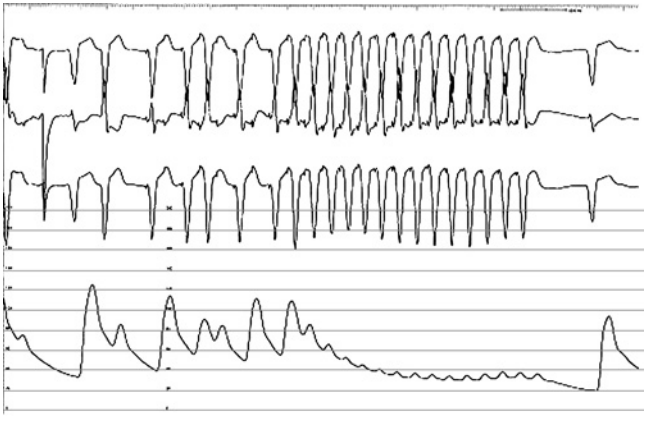

Figure 27-4 Burst pacing is used to reduce transvalvular flow and cardiac motion. We aim for a reduction in systolic arterial pressure to below 50 mmHg.

the apex of the fluoroscopically visible calcified valve. The wire is gently advanced until it slides off down the surface of one valve leaflet at which point the wire is withdrawn. The catheter advanced or withdrawn slightly to reorient the wire which is then readvanced. Forceful probing contributes nothing to the procedure. At some point the wire will pass over the apex of the valve and be redirected into the contralateral cusp. At this point the process is reversed to again direct the catheter and probing wire towards the apex of the valve. This approach typically results in relatively quick passage through the larger central orifice of the valve after which the wire is typically observed to settle into the commissure towards the patient's right.

****Balloon Valvuloplasty:** A large curve is formed on an exchange wire which is then placed through the diagnostic catheter into the left ventricle. We typically select a valvuloplasty balloon slightly smaller in diameter than the intended prosthetic valve to avoid excessive dilation. We typically use a 20 or 23 mm diameter balloon. We prefer a 3 cm length to simulate the behavior of the balloon utilized later for stent deployment. Longer balloons take longer to inflate and deflate and are unnecessary with proper attention to positioning and burst pacing. To facilitate balloon passage through a 14Fr sheath we evacuate air and test the balloon with repeated low volume inflation in the descending aorta. We utilize dilute 10–20% contrast to reduce viscosity and reduce inflation-deflation time. Balloon sizes of 20 mm × 3 cm and 23 mm × 3 cm respectively require approximately 15 and 18 mL to reach nominal diameter. We use a 20 mL syringe or inflation device and inflate to a predetermined volume to achieve a predicted diameter. It is important to practice the sequence of events to assure that burst pacing and the inflation–deflation cycle is performed flawlessly during later valve deployment.

The balloon is positioned across the valve. Burst pacing is initiated, the balloon is rapidly inflated and deflated, pacing terminated and the balloon withdrawn from the valve to allow hemodynamic recovery. We observe the motion of the valve leaflets during inflation to aid in later prosthesis positioning and assess the risk of coronary obstruction.

Management of Hypotension: Most patients develop a progressive fall in arterial pressure during percutaneous valve implantation. Burst pacing, tachycardia, ventricular ectopy due to the left ventricular manipulation, radiographic contrast, balloon valvuloplasty, pre-existing left ventricular dysfunction, untreated coronary artery disease and general anesthesia predispose to myocardial ischemia and reduced contractility.

Systemic arterial pressure should be continuously monitored. Transient hypotension can rapidly deteriorate into hemodynamic collapse. Hypotension should be managed aggressively in a patient with aortic stenosis to avoid a downward spiral of myocardial ischemia, left ventricular depression and worsening hypotension. We aim for a systolic blood pressure of above 100 mmHg or a mean aortic pressure over 70 mmHg. In most patients this requires intravenous vasopressor

support (Table 27-1). We favor low dose norepinephrine to maintain systolic pressure above 100 mmHg. Phenylephrine boluses are commonly utilized if systemic pressure falls precipitously or a fall is anticipated due to impending burst pacing or valvuloplasty.

When possible we avoid inotropic and chronotropic agents like epinephrine, dopamine or dobutamine. These agents tend to exacerbate ischemia in the presence of aortic stenosis, although they may be useful if myocardial depression persist following aortic valve implantation. Most importantly the procedure must be judiciously paced, allowing appropriate time for recovery between pacing, angiography, and balloon valvuloplasty to allow myocardial recovery.

Large Sheath Insertion: Sequential dilators are used prior to insertion of the large (currently 22–24Fr) sheath required for valve insertion. Fluoroscopy is used to alternately ensure that the wire is not pulled out of the ventricle while the dilators and sheaths do not perforate the pelvic arteries. The sheath must be advanced past the aortic bifurcation to allow free passage of the prosthetic valve into the aorta. We routinely suture the sheath in place to avoid movement and trauma to the aorta by the stiff tip.

TECHNICAL TIPS

****Management of Arterial Injury:** Vascular dissection or perforation is a concern when utilizing large sheaths and catheters. It is important to utilize fluoroscopy during large sheath insertion and any manipulation of the valve delivery catheter. A hematoma or unexplained hypotension may be the initial clue to vascular perforation. Aortic or iliac angiography with a pigtail inserted through the large sheath or contralateral sheath should be considered. We maintain contralateral arterial access until after large sheath removal is successfully accomplished. Should pelvic arterial perforation occur a compliant occlusion balloon rapidly advanced into the aorta and inflated followed by implantation of a covered stent or surgical repair can be lifesaving. When utilizing large sheath access it is important to have ready access to equipment suitable for the management of large vessel dissection or perforation.

****Wire Selection: Why Difficulty on Advancing the Prosthetic Valve on a Very Stiff Wire?** A wire diameter of 0.035″ is required for the currently available delivery system. A 0.038″ wire is incompatible with several other procedural components (valvuloplasty balloon, large diameter occlusion balloon, covered stents, etc). A relatively soft atraumatic distal left ventricular segment is desirable. We typically utilize an Amplatz extra stiff exchange J wire (Cook Inc, Bloomington IN) and hand form the distal portion by pulling a hemostat along the wire to form a gentle left ventricular curve. If ilio–femoral tortuousity is a problem we may utilize the much stiffer Meier wire (Boston Scientific Inc, Natick MA) during sheath placement. However a wire that is too stiff typically follows the outer curvature of the aortic arch and settles in a lateral commissure. It may be extremely difficult to

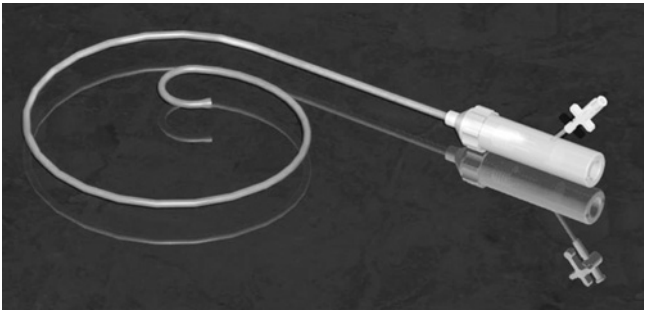

Figure 27-5 A steerable pusher catheter (Retroflex™ catheter) is used to manipulate the prosthesis through the aorta and native valve.

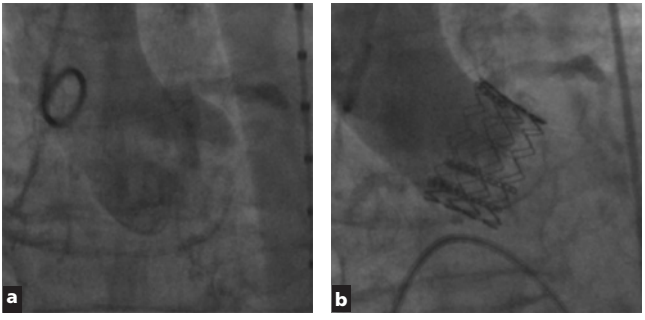

Figure 27-6 (a) Aortogram in a patient with aortic stenosis. (b) Following valve implantation.

displace a too stiff wire from its position within a commissure to allow passage of a bulky prosthesis through the central orifice of the valve.

TECHNIQUE Valve Implantation: The prosthetic valve is introduced through the sheath and advanced to the aortic arch under fluoroscopy. The prosthesis is advanced around the arch while imaging in the LAO view utilizing the steerable delivery RetroFlex catheter to hug the inner curvature of the aorta and avoid scraping the aortic wall (Figure 27-5). Once positioned in the ascending aorta we move to the radiographic view previously determined to show the valve in profile. Utilizing a combination of force and careful manipulation the prosthesis is passed into the left ventricle and the delivery catheter withdrawn to the aorta. The prosthesis is positioned in the annulus. Correct positioning is confirmed with fluoroscopic imaging of valvular calcium, ascending aortography and transesophageal echocardiography. Burst pacing is initiated and the prosthesis delivery balloon inflated (Figure 27–6).

We usually utilize surgical open closure to achieve reliable hemostasis. However in selected patients currently available percutaneous closure

devices can be utilized successfully. Patients are extubated in the cardiac cath lab and transferred to the coronary care unit. Mobilization occurs the next day with hospital discharge occurring at a median of 4 days in these, mostly elderly, patients.

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Chapter 28
Intervention in Intracranial Arteries

Sundeep Mangla

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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GENERAL OVERVIEW

Ischemic stroke and intracranial atherosclerotic disease remain as leading causes of morbidity and mortality throughout our global population, ranking second as a cause of death worldwide [1]. In the United States, 700,000 people suffer a primary or recurrent stroke annually. Stroke remains the leading cause of serious, long-term disability, the third leading cause of death accounting for 1 of every 15 deaths (approximately 273,000 stroke-related deaths annually). In 2006, the estimated direct and indirect cost of stroke in the United States is about $58 billion [2]. Atherosclerotic intracranial stenosis of the major intracranial arteries is a major cause of ischemic stroke, especially in certain high risk ethnic populations including blacks, Asians, and Hispanics where the risk may be as high as 29% [3,4]. In the general population, intracranial stenosis accounts for 8–10% of all ischemic strokes in the United States. The risk of recurrent stroke in these patients may be as high as 15% annually, higher within those patients with greater than a 70% symptomatic stenosis [5–11]. The purpose of this chapter is to review the current evidence concerning natural history risks of intracranial atherosclerotic disease (IAD) with medical therapy, and the evolving role of endovascular intracranial intervention in this high-risk disease population, as well as to provide a basic primer on clinical evaluation, management, and performance of these complex neurointerventions.

Current Evidence and Experience: The evolution of intracranial revascularization of intracranial arteries using percutaneous transluminal angioplasty (PTA) and eventually stent-assisted angioplasty (SAA) was initiated soon after successful application within the cardiac circulation [12] first reported in the basilar artery by Sundt *et al.* over 25 years ago [13]. In the interim, coronary intervention has advanced rapidly becoming standard care in a growing population of cardiac disease patients. Intracranial intervention has progressed but not at the same rate, secondary to the inherent complexities of the end organ, the brain, including but not limited to highly eloquent tissue (i.e. functionality), complex technical geometry, and the delicate nature of the cerebral circulation (subarachnoid location, thinner media, absence of a significant adventitia). Since then, there have been numerous, primarily retrospective, single center reports by neurointerventional pioneers of the application of these techniques [14–31]. Until recently, these procedures were performed with compassionate use of "off-label" coronary devices in primarily high risk symptomatic patients who experienced recurrent events despite optimal medical therapy. Some reports have included treatment of asymptomatic patients with

high-grade, high-risk lesions of isolated cerebral circulations. Their collective experience reflects a very diverse poorly defined primarily high-risk patient population, with correspondingly diverse techniques, perioperative management regimens, adverse event rates, and clinical outcomes. These reports highlight the need for prospective, controlled studies to test many of the concepts and hypothesis developed from these experiences. Outside of an approved clinical trial or institutional board review (IRB) protocol, intracranial PTA or SAA should be considered primarily for high-risk, high-grade stenosis, symptomatic patients failing optimal medical therapy, or in rare instances, perhaps asymptomatic patients with high-risk territory lesions [32].

Currently, only two prospective, non-randomized, uncontrolled studies evaluating SAA have been reported. Both of these studies, SSYLVIA and WingSpan Trials, were performed as prospective, multicenter, non-randomized, feasibility studies without control limbs to demonstrate preliminary safety in treating high risk groups failing medical therapy [10,33,34]. These studies were performed in symptomatic patients with symptoms attributable to intracranial cerebral vessel stenosis of greater than 50%, similar to the WASID study group. In the SSYLVIA trial, results from 61 patients were reported with successful stent placement in 58/61 (95%) and 30 day stroke and mortality rates of 6.6% and 0% respectively. A high rate of recurrent stenosis was observed at 6 months (intracranial arteries – 32.4%, extracranial vertebral arteries – 42.9%), as were recurrent symptoms in this group (39%). In addition, 4 of 55 patients (7.3%) followed experienced strokes between 30 days and 1 year. The combined 1-year stroke and death rate death rate exceeded 10%, approaching the natural history risks associated with mid risk WASID lesions cited above. The United States Food and Drug Administration (FDA) did ultimately grant Guidant (Santa Clara, CA) a Humanitarian Device Exemption (HDE) for their balloon expandible Neurolink intracranial stent. This approval was limited to an orphan indication (anticipated less than 4000 patients/year, as defined by HDE guidelines) of high risk, high grade stenosis within the intracranial and extracranial circulation for patients failing medical therapy for their disease [10]. Although FDA approved and not technically representing research, institutions performing these HDE approved therapies must obtain IRB approval and FDA prescribed training before being permitted to purchase these devices.

Boston Scientific (Freemont, CA) sponsored a similar study to evaluate a new therapeutic strategy of intracranial balloon angioplasty followed by placement of a self-expanding nitinol stent system (WingSpan). Similar technical success was reported (44/45, 98%) in 45 patients, however, with composite 30 day ipsilateral stroke and death rates of 4.5% (2/44) and a 6-month ipsilateral stroke and death rate of 7.1% (3/42). Based on these results, the FDA also granted HDE approval for the Gateway/WingSpan PTA/Stent System (Boston Scientific, Freemont, CA) for high-risk symptomatic patients failing medical therapy [33].

These trials highlight the continued high-risk nature of these procedures and currently approved limited role. These early results are promising and may represent progress in improving outcomes over the natural history risks associated with this disease. Nevertheless, many questions about general application of current techniques to all symptomatic patients persist. The results support the development and initiation of independent publicly funded prospective controlled trials to objectively evaluate these new therapeutic strategies and develop benchmarks for future innovations in attempts to improve outcomes in high-risk subsets of cerebrovascular atherosclerotic disease patients.

PRE-PROCEDURAL EVALUATION

If an anatomic intracranial vascular stenosis is identified, numerous steps need to be undertaken before one can proceed with the therapeutic decision to attempt revacularization. Understanding the patient's symptoms and clinical findings, and correlating with the anatomically identified lesion is of paramount importance. In the current era of advanced anatomic imaging, many incidental lesions are discovered, most of which are asymptomatic, low risk stenosis that may be best managed medically.

CLINICAL EVALUATION

All patients being considered for revascularization should undergo a comprehensive clinical evaluation by an experienced stroke neurologist. Anterior circulation lesions tend to cause contralateral motor and sensory symptomotalogy as well as expressive and receptive language deficits (especially if involving the language dominant hemisphere, the left side in most patients, even left handed ones). Posterior circulation ischemic syndromes are more likely to present with cranial neuropathies, bilateral motor or sensory long tract syndromes, dysarthria, or syncope. Visual symptoms are usually related to thrombo-emboli to ophthalmic artery from a proximal source (i.e. cardiac, carotid bifurcation), but may be caused by ischemia involving the visual cortex, within the occipital lobe supplied via the posterior cerebral arteries (PCAs) of the posterior circulation. Common anatomic variations (i.e. fetal origin of the PCA from the internal carotid artery) need to be identified, as they may confound traditional presentations. Detailed evaluation of the clinical syndrome to piece together a likely etiology is critical.

Alternative diagnosis that may potentially mimic a vascular syndrome including seizures, neoplasms, and degenerative neurologic disorders need to be excluded. In addition, cardiogenic emboli, small vessel perforator occlusive disease, intracranial hemorrhage, or cerebral vasculitis may result in a stroke syndrome, independent of an incidentally identified asymptomatic stenosis.

Intracranial atherosclerosis, itself, may cause an ischemic injury by a variety of potential mechanisms, including hypoperfusion, acute thrombosis, thromboembolism, and penetrating branch occlusions.

Not all of these are amenable or likely to benefit from interventional revascularization techniques. The most commonly accepted indication for revascularization is currently hypoperfusion secondary to inadequate cerebrovascular reserve related to poor collateral circulation, often termed "perfusion failure". These patients may present with intermittent, transient symptoms of variable severity attributable to the identified vascular stenotic distribution. These symptoms may be unstable and exacerbated with orthostatic positioning or hemodynamic instability (i.e. hypotension, dehydration, anemia). Cross sectional MR or CT imaging studies may demonstrate evidence of non-confluent or confluent subcortical or periventricular white matter ischemic injury within the borderzone "watershed" territories of the major cerebral arteries. These patients should undergo a functional evaluation to assess their cerebrovascular reserve as will be described below.

Patients experiencing arterial-to-arterial thromboembolism from an unstable plaque or high-grade stenosis may also experience a similar clinical presentation and may be higher risk for attempted revascularization. Modern medical management to minimize thromboembolic events and stabilize plaque utilizing antiplatelet agents and statins may offer an opportunity for clinical stabilization. In addition, aggressive control of vascular risk factors such as hypertension, diabetes, smoking, and obesity may slow or in rare cases reverse the progression of these lesions. If medical therapy fails, the treating physicians and affected patients need to be informed of high-risk natural history of their lesions, as well as the high-risk nature of attempts to treat them.

Acute thrombosis of a high-grade initially asymptomatic stenosis remains as a major concern in the small vessels of the cerebral circulation. If this occurs within the proximal branches of the Circle of Willis (i.e. M1 segment of the middle cerebral artery (MCA)) beyond the direct conducting collaterals of the anterior or posterior communicating artery, a catastrophic stroke may be a patient's initial and only clinical presentation. Indirect pial-to-pial collaterals are often inadequate in these situations. Unfortunately, high-level prospective evidence for these specific asymptomatic lesions has not yet been obtained, and a team of experienced neurointerventionalists and stroke neurologists may consider judicious application of revascularization in these select cases.

Small vessel arterial perforator occlusion may result in strokes in the basal ganglia, thalamus, and brainstem at the site of a larger conducting vessel stenosis. These types of stroke syndromes are unlikely to benefit from large vessel revascularization, and in fact, may be exacerbated if revascularization is attempted.

NON-INVASIVE IMAGING

The initial evaluation of potential stroke patients includes standard non-invasive cross-sectional imaging techniques including computed tomography (CT), magnetic resonance (MR), contrast enhanced CT angiography (CE CTA), or MR angiography (MRA). These initial screening studies will often establish a preliminary differential diagnosis of potential stroke syndromes, identifying evidence of ischemic injury

(Diffusion MR, FLAIR) as well as potential vascular stenosis (MRA, CECTA). In addition, they can often help differentiate non-vascular etiologies of the presenting neurologic syndrome.

After a potentially symptomatic vascular stenosis is identified, additional techniques to determine the potential mechanisms of cerebral injury may need to be performed. These techniques may help differentiate patients experiencing symptomatic "perfusion failure" from other mechanisms of ischemic injury (i.e. arterial–arterial thromboembolism). A variety of different modalities and techniques are currently to assess cerebral blood flow (CBF) and cerebrovascular reserve, including transcranial Doppler ultrasound (TCD), single photon emission CT (SPECT), stable xenon CT, CT Perfusion, MR Perfusion, and Positron Emission Tomography (PET). Many of these studies are performed with a provocative challenge to promote a vasodilatory response. Vascular territories experiencing ischemia will normally autoregulate their vasculature by dilating affected vessels to enhance CBF. If CBF reduction is advanced, autoregulatory vasodilatation may reach maximum limits in the absence of inadequate collateral perfusion. Pharmacologic vasodilatory agents (i.e. acetazolamide) or a physiologic vasodilatory stimulus (hypercapnia by inhalation of carbon dioxide (CO_2)) are administered to determine post-stenotic vasoreactivity by measuring reductions in CBF (i.e. SPECT, Xe CT) or changes in flow velocities (TCDs) [35–41]. In addition, PET can be used to measure oxygen extraction fraction (OEF), which is elevated in perfusion failure as brain tissue attempts to maintain cerebral oxygen metabolism and preserve neuronal function. Many preliminary studies have been performed to suggest that patients experiencing advanced perfusion failure as identified with reduced vasoreactivity or elevated OEF may be at significantly higher risk for future cerebral ischemic events. Previous studies have been performed in a variety of different types of at risk ischemic patients (internal carotid artery (ICA) occlusion, intracranial stenosis, vasospasm), however, further systematic prospective evaluation of these techniques needs to performed to determine their prognostic utility in intracranial atherosclerotic stenosis. Nevertheless, the current evidence supports a hypothesis that impaired cerebrovascular reserve may identify patients likely to benefit from revascularization, while those with adequate reserve may experience a lower risk of future ischemic events [38,40–47]. Ultimately, the decision to attempt revascularization in any specific patient is complex and all of the clinical and imaging data need to be considered carefully, presented to the patient, with appropriate counseling of their risks and potential therapeutic options.

PERI-OPERATIVE MANAGEMENT

As in cardiac intervention, the risk for acute thrombotic occlusion following PTA or SAA remains significant. Although no prospective trials comparing perioperative medical regimens are currently available, general feeling and practice has been extrapolated from coronary artery stenting trials. Most neuro-interventionalists place patients on double antiplatelet therapy with Aspirin (ASA) (325 mg po qd) and Plavix

(75 mg po qd) for 3 to 5 days prior to an elective procedure and maintain them on double antiplatelet therapy for 4 to 6 weeks to allow the stent to endothelialize. Subsequently, one antiplatelet agent is usually maintained for life. In the event of urgent or emergent case, bolus dose of Plavix (375 mg) or trans-rectal ASA can be given to accelerate platelet inhibition.

Glycoprotein 2b3a inhibitors (GPI) have also been used in select cases, but their routine role remains undefined. As one might surmise, ischemic complications have been averted using GPI both with bolus infusions administered intra-arterially at the site of platelet aggregation or with standard cardiac intravenous bolus and continuous infusions regimens. Caution is warranted with regards to their routine use, as ischemic brain tissue is particularly vulnerable to hemorrhagic complications, which frequently are fatal or devastating. A variety of different inhibitors have been utilized with the full spectrum of anecdotal reports of their effectiveness [48–52]. In addition, active intraoperative monitoring of platelet inhibition is recommended, although no evidence-based guidelines or recommendations for neurovascular intervention are currently available.

Anticoagulation with intravenous heparin is routinely performed with frequent ACT monitoring. Generally, ACT goals are generally twice baseline (200–250) for these types of cases, as aggressive anticoagulation in conjunction with antiplatelet agents may increase the risk of intracranial hemorrhage.

Hemodynamic management also plays a critical role, targeting blood pressure to each patient's unique cerebral perfusion needs and risks. For example, a patient with a high-grade intracranial stenosis may require an elevated blood pressure to maintain cerebral perfusion in a maximally dilated (autoregulatory dilation) cerebral vessel. Following revascularization, the patient may be at significant risk for reperfusion hemorrhage or injury, and the blood pressure may need to be significantly reduced and closely monitored until the patient's autoregulatory mechanisms are restored. Intensive care monitoring of hemodynamic and neurologic status is strongly recommended to avert potentially devastating delayed complications. As described earlier, these procedures are best performed with the collaboration of a multidisciplinary team of neuroclinicians.

STANDARD TECHNIQUES

General Preparation: As the field of neurointervention continues to evolve and a growing number of tools are becoming available, the techniques, protocols, and tricks utilized vary widely among interventionalists, the discussion to follow is a general guideline of how these procedures may be performed.

General preparation includes arterial line, large bore IV's, and Foley catheterization. Most intracranial PTA and SAA cases for IAD are performed under general anesthesia to minimize the risk of perforating the delicate cerebral vessels secondary to patient movement. In addition,

stricter hemodynamic monitoring and management is facilitated by utilization of general anesthesia. Some interventionalists may perform their procedures under monitored anesthesia care or conscious sedation in favor of neurological monitoring, but the operating physician should carefully weigh benefits of each.

Careful preparation of several pressurized continuous arterial flush lines (3 to 5) should be performed, one each for the femoral line and large vessel cerebral guide, as well as one or two lines potentially for the microcatheter/PTA balloon/stent devices.

CAVEAT

Safety Measures: Great care should be taken to avoid microbubbles (and definitely macrobubbles!), as the brain is particularly vulnerable to air/thrombus microemboli. A small vessel air embolus in the heart muscle may not be clinically relevant (perhaps a small reduction in contractility or ejection fraction), however, in the brain, this could result in irreversible injury to critical functional (eloquent) regions of the brain (i.e. speech, consciousness, memory, strength, balance, vision, respiration, etc.). The location or eloquence of damaged tissue is as critical if not more critical than the volume of injury.

Access: Arterial access is usually performed transfemorally, although alternative access is occasionally preferred or necessary (brachial, radial, direct carotid). A comprehensive cerebral angiogram should be performed to assess the cerebral vasculature for tandem or additional lesions, as well as to assess collateral circulation and cerebrovascular reserve anatomically. Isolated cerebral circulations and distal stenosis may be particularly vulnerable to temporary occlusion during balloon inflation or stent deployment, and accommodations in techniques may be required. All procedures should generally be performed with anticoagulation as detailed above.

Guide: The choice of access sheath size and guide size will be based on the devices that may be used. Most currently available balloon catheters and stents may be placed through a 6Fr guide, however, some devices or if necessary, multiple devices may require a larger 7Fr guide. These guides should be connected to a pressurized continuous flush to prevent thrombus accumulation and distal nontarget embolization. The position of the guide should be within the most distal cervical segment of the target vasculature (i.e. cervical carotid or vertebral artery). These guides are generally too rigid to be placed within intracranial segments, but placement as distal as is safe is generally preferred to provide adequate support for distal navigation of devices. Guide placement is a particularly dangerous step in neurovascular interventions, as the additional guide rigidity required for distal

navigation makes the guide more likely to injure the endothelium/vessel. Several steps and signs may be utilized to help obtain safe distal guide placement.

TECHNICAL TIPS

****Guide Position:** First, the guide may be placed over an exchange length wire (0.035″ or 0.038″) or primarily (in less tortuous vasculature). When using a large diameter guide, there is often a significant step-off/transition between the inner diameter of the guide and the outer diameter of the wire. This transition may act like a wood-plane or cheese grater damaging endothelium as the guide is advanced. A smaller diagnostic catheter within the guide may smooth the transition from wire/diagnostic catheter/guide for advancement (4Fr within a 6Fr Guide, or a 5Fr within a 7Fr Guide). The diagnostic catheter and wire may then be removed and the guide connected to pressurized continuous flush.

****Guide Induced Vasospasm:** Guide induced vasospasm is very common during guide placement. It should be recognized immediately as flow-arrest develops quickly placing patient at risk for thrombotic occlusion and distal emboli. Vasospasm can often be remedied by withdrawing the guide and delayed observation. Alternatively, small doses of intra-arterial verapamil (1–3 mg) or nitroglycerin may be administered to facilitate resolution or prevent catheter-induced vasospasm.

CAVEAT

Continous Position Monitoring: Finally, once a safe position is obtained, continuous monitoring of this distal position is recommended for several reasons. First, vasospasm may recur secondary to movement of the guide and should be recognized and managed quickly. Delayed contrast washout within the guide vessel is usually observed and is a warning sign of proximal periguide occlusion as well as potentially distal embolic occlusion. Second, the guide may be "pushed" back proximally as rigid distal device navigation is performed risking loss of distal access and control.

Wire: After guide placement is performed, standard over the wire techniques are recommended to approach the stenotic lesion. All intracranial navigation should be performed with the assistance of digital roadmapping to minimize vascular trauma during the intervention. A non-exchange (205 cm) 0.014″ wire/microcatheter system is generally utilized to gain primary access across the stenosis. The shorter microwire is then exchanged for a variably firmer (usually medium stiffness)

exchange length (300 cm) 0.014-inch microwire in preparation for device deployment.

TECHNICAL TIPS

****Wire Manipulations:** When approaching the stenotic lesion, care must be taken to minimize wire manipulation within the intracranial sidebranches. Excessive manipulation or twisting may result in prolonged vasospasm in an important branch (i.e. anterior choroidal artery – dense hemiparesis face/arm/leg), or branch avulsion with subsequent intracranial hemorrhage.

Angioplasty and stenting: Primary angioplasty (without stent assistance) remains a viable option and may often be adequate in restoring adequate cerebral perfusion (Figure 28–1). The role of stenting remains poorly defined, however, may be performed safely primarily (Figure 28–2) and to manage PTA related dissections or vascular

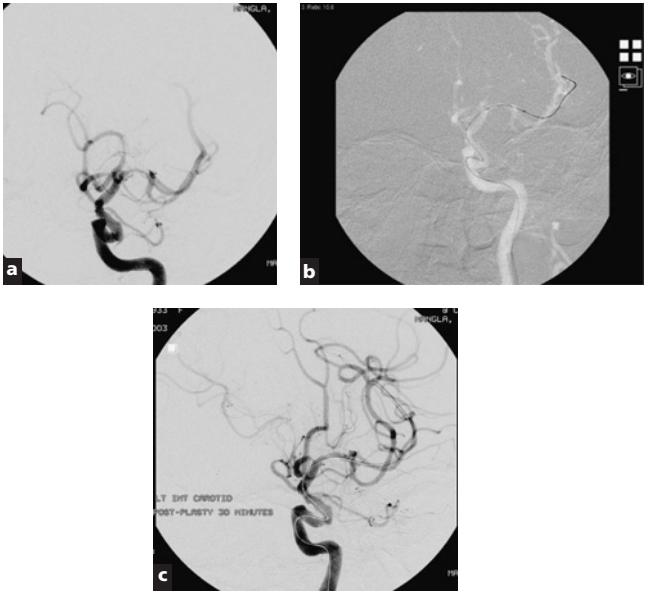

Figure 28-1 A 70-year-old right-handed woman presented with an acute infarct and progressive crescendo TIAs resulting in worsening expressive aphasia and right hemiparesis. (a) AP Left ICA angiography demonstrates a 70–80% Left MCA stenosis. (b) AP Left ICA digital roadmap of a compassionate off label coronary balloon position, note the slight straightening of the vessels following balloon placement. (c) AP Left ICA angiography following two serial inflations (nominal pressures 4–6 atm) demonstrates less than a 20% residual stenosis with subsequent stabilization of neurological syndrome.

injury. In addition, the hypothesis that stent-assisted PTA should be utilized primarily to reduce restenosis and prolong disease free survival has been proposed, awaiting future trials.

TECHNICAL TIPS

****Balloon Angioplasty:** The PTA balloon should be slightly smaller than the target vessel (0.5–1.5 mm) (and never larger!) to minimize the risk of vascular perforation. Gradual serial dilatation may be performed to maximize revascularization while attempting to minimize traumatic dissections. Pre-dilatation is often required before stent placement and deployment can be achieved. Post dilatation may also be required, however, should be performed with caution.

Slow gradual inflation to the nomimal pressure is generally recommended. The goal is, if possible, to stretch the vessel rather than dissect. Slow gradual low-pressure inflations are generally recommended, lasting 30–60 s per inflation, 1–2 minutes apart to allow interim reperfusion.

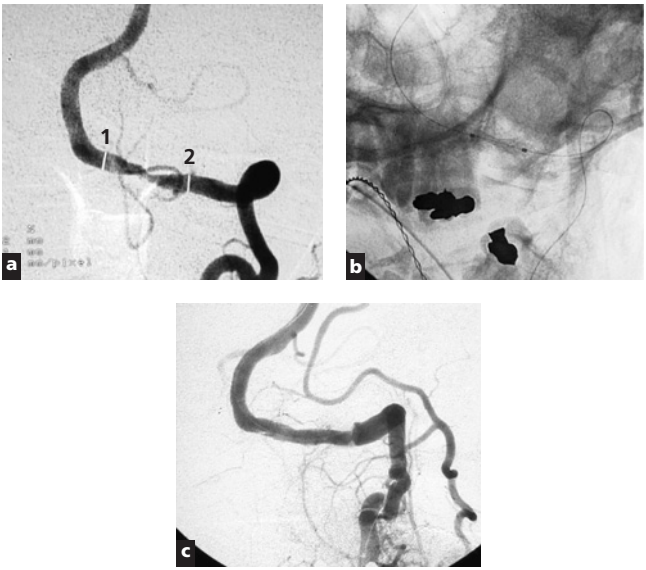

Figure 28-2 A 75-year-old male presented with rapidly progressive ataxia and vertebrobasilar insufficiency. (a) AP left vertebral angiography demonstrates a 70–80% stenosis of the only remaining dominant left vertebral artery (and absent posterior communicating artery collaterals isolating this vertebrobasilar circulation). (b) AP radiograph during deployment of a compassionate off-label balloon expandable coronary stent (nominal pressures 6–10 atm). (c) AP left vertebral angiography confirms restoration of adequate cerebral blood flow with resolution of neurological syndrome.

At present, both balloon expandable (Neurolink, Guidant, Santa Clara, CA) and self-expanding (WingSpan, Boston Scientific, Freemont, CA) stents are currently approved for intracranial use in IAD. When employing balloon expandable stents, sizing larger than the target vessel is discouraged secondary to risk of perforation. Self-expanding stents, however, should be sized slightly larger than the target vessel to facilitate vessel wall approximation and radial stabilization.

Prior to concluding the intervention, control angiography of the distal cerebral circulation should be performed to identify distal embolic complications. If necessary, pharmacologic/mechanical thrombolysis and modification of the perioperative medical regimen may be required.

Postoperative Management: Double antiplatelet therapy for 4–6 weeks is generally recommended (especially for stents), with at least one medication maintained indefinitely to facilitate endothelialization and minimize acute thrombosis, thromboemboli, and neointimal hyperplasia. Non-invasive imaging and cerebral angiography (as necessary) should be performed for short and long-term surveillance, in conjunction with routine clinical monitoring.

NEURO-INTERVENTION DURING STROKE

The present options of treatment for ischemic stroke include systemic intravenous infusion of fibrinolytic agents, localized intra-arterial infusion of fibrinolytic agents, angioplasty or stenting.

Local Intra-arterial Thrombolysis: The concept of intravenous thrombolysis represented a significant advance over traditional conservative medical management of stroke patients, however, patient eligibility and benefits remain limited to a small subpopulation of the overall afflicted population. The therapeutic strategy to administer a smaller dose of thrombolytics intra-arterially directly within the intraluminal thrombus represented a significant potential advance over intravenous therapy.

Despite the modest rate of recanalization (complete recanalization ~19%) and the increased risk of hemorrhagic conversion (10%) [53], this represented a significant advance over intravenous thrombolytic therapy (TT) with improved clinical outcomes in a prolonged therapeutic window in more critically affected patients. Unfortunately, a reported manufacturing quality assurance deficiency of urokinase resulted in suspension of urokinase production, and FDA approval for Prourokinase was never granted (http://www2.kumc.edu/druginfo/drugsafety/drgsafe99-2.html). The therapeutic benefit over intravenous and conservative medical therapy remains significant, and compassionate off-label use of similar thrombolytics (tPA, urokinase) is performed throughout the world in clinical stroke treatment centers where the resources and expertise have been adequately developed. The inclusion criteria for direct intracranial intra-arterial thrombolysis are listed in Table 28-1.

Table 28-1 Indications and Exclusion Criteria of Intra-arterial Thrombolysis

Indications
Fibrinolytic therapy within 6 hours from onset of symptoms
Symptoms appropriate to the location of the occlusion
No history of recent infarct
Exclusion criteria
Rapidly improving clinical symptoms
Very severe onset symptoms (e.g. seizures)
Recent stroke (<6 weeks)
Bleeding disorder
Certain types of recent surgery
CT scan evidence of intracranial bleed, large area of edema, or mass effect

Arterial Infusion Techniques: If a patient meets the criteria for emergency intracranial fibrinolysis, a diagnostic angiogram is performed in the anteroposterior and lateral projections [54]. If possible, angiography of the opposite carotid artery and one vertebral artery should be done to determine the status of anterior and posterior communicating arteries and leptomeningeal anastomoses. All intracranial vasculature is evaluated, beginning with the uninvolved vessels to assess the collateral supply to the ischemic territory. The target vessel is studied last for direct evidence of vessel occlusion. If the study is positive, heparin is given intravenously, generally recommended at a relatively low dose as used in PROACT II trial, 2000 units initially [53,55].

Isotonic contrast is very slowly injected because the capillary endothelium is ischemic and there is no significant run-off or reflux if the vessel is occluded. This may theoretically increase the risk for contrast related endothelial injury, and more concerning, acute vessel rupture and sudden death secondary to high intracranial pressure. The thrombus is laced with rt-PA, injected as the catheter is withdrawn; the catheter tip is left at the thrombus. A continuous infusion is begun at a variety of different rates depending on the concentration and thrombolytic agent selected [53,55].

The progress is checked every 15 min by contrast injection through the guide around the microcatheter. The microcatheter position is adjusted as thrombolysis progresses to maintain the tip of the catheter as close to the thrombus as possible. If an underlying stenosis is encountered, angioplasty may be required to prevent acute rethrombosis. The decision to stop therapy is based on the response, the duration and depth of ischemia, and the location of the occlusion. For most standard acute stroke intervention protocols, attempts to revascularize an occluded vessel in the anterior circulation are terminated by 8 hours from onset of symptoms. The posterior circulation (i.e. the basilar artery) may be attempted in a prolonged therapeutic window but must be considered on a case by case basis. The heparin infusion is stopped at the end of the procedure [55].

Table 28-2 Factors Influencing the Incidence of Bleeding Conversion

The length of time of occlusion
The status of the lenticulostriate arteries
When and if reperfusion was accomplished
The systemic blood pressure
The presence or absence of very early ischemic changes on CT scan

Postprocedural management includes control of blood pressure if elevated, neurological monitoring, and routine supportive care. Control of hypertension (intravenous labetalol, intravenous nicardipine, and/or nitroprusside if needed) is indicated if reperfusion of a large ischemic area has been achieved and the blood pressure is above 180/110. Otherwise, a modest physiological increase in blood pressure is therapeutic for the ischemic area and should not be aggressively treated. Neurological progression should be monitored closely and other co-morbidity appropriately treated [55].

Complications: The major complication of this procedure is symptomatic intraparenchymal hemorrhage. In addition, reperfusion edema can worsen an already bad situation. Factors that affect the incidence of bleeding conversion are included in Table 28-2 [55].

Intra-arterial Mechanical Thrombolysis: The utilization of thrombolytics for acute stroke (intravenous and intra-arterial), have resulted in a significant benefit for patients over traditional conservative medical therapy, however the hemorrhagic conversion rates remained relatively high and the number of patients excluded from eligibility secondary to the numerous high-risk exclusion criteria limits applicability. This has led to a search for a mechanical method of clot retrieval that may increase the number of patients eligible for therapy, enhance recanalization efficacy, minimize the potential for hemorrhagic conversion, and further improve the rate of independent survival for stroke victims.

Wire: The embolus can be fragmented with several passages of wire and catheter through its length; by twisting the curved end of the wire within the thrombus while the wire is pulled back; and by saline injection into the thrombus through the micro catheter [56]. Vessel perforation with excessive manipulation can be a concern.

Balloon Angioplasty: When anatomically possible or with a failure of pharmacologic thrombolysis, mechanical thrombolysis using a soft high compliance neurovascular balloon may be performed in an attempt to macerate the thrombus and enhance low-dose pharmacologic therapy. Balloon inflations should be limited to less than 20–30 seconds and should be performed at very low pressures (less than 1 atmosphere) when attempting to macerate intraluminal thrombus. If an underlying stenosis is observed following successful thrombolysis, angioplasty of the stenotic segment may be required acutely

to maintain vascular patency. This can be performed using a low-compliance atherosclerotic PTA balloon, with short duration (10–30 secs) and low pressure (1–5 atm) inflations in an attempt to stretch the stenotic intracranial vascular segment [56–59]. The short balloon inflation and low pressure are to avoid compromising the perforating arteries, flow limiting dissections, and vessel rupture [60]. Post-angioplasty angiogram should be done to assess for success, distal embolization, and intimal or medial damage to the artery. The combination of TT and mechanical disruption usually helps to achieve vessel recanalization faster than thrombolysis alone. It also decreases the need for a large amount of thrombolytic agent, thus decreasing the possibility of reperfusion intracranial hemorrhage [56].

Intra-arterial Clot Retrieval or Mechanical Thrombolysis: The FDA has approved the Mechanical Embolus Removal in Cerebral Ischemia (MERCI) retrieval system, a corkscrew-like apparatus, to remove blood clots from the brain in patients experiencing an ischemic stroke within 8-hour of onset of stroke symptoms.

The initial safety and efficacy studies demonstrated promising early results in a very critically ill population of patients in a further prolonged therapeutic window of 8 hours. Recanalized patients experienced a higher rate of functional neurologic recovery (46%) and decreased mortality (32%) compared to persistently occluded patients (10% functional recovery, 54% mortality). Nevertheless, the rate of recanalization remained modest (46%) and procedural complications occurred at a significant rate (7.1%). In spite of the prolonged therapeutic window, the symptomatic hemorrhagic conversion rate remained relatively low (7.8%) compared with trials utilizing pharmacologic thrombolytics within a similar therapeutic window [61], further supporting the hypothesis that minimizing thrombolytic administration during attempted recanalization may improve clinical outcomes [62].

Adjunct Pharmacotherapy: Nicardepine and/or verapamil may be very useful for treating distal spasm. Glycoprotein IIb-IIIa inhibitors are promising drugs. These medications are often given intra-arterially through the guide or microcatheter. When good flow has been rapidly reestablished, protamine may be given intravenously to reverse anticoagulation or intravenous heparin can be discontinued to hopefully lessen the risk of hemorrhagic conversion [56].

Complications: The most feared post-embolic recanalization complication is intracranial hemorrhage [56]. It may be part of a natural pathological process that degrades the integrity of the vascular system and the exudation of blood components into the cerebral environment. Spontaneous or facilitated reperfused ischemic brain parenchyma is very prone to hemorrhagic conversion. Ischemically damaged terminal branches, especially the lenticulostriate basal ganglionic arteries, are also very prone to rupture, without recourse from collaterals [56]. When the horizontal M1 segment of the MCA is recanalized and reperfused, it is the combination of ischemic brain parenchyma and

ischemic damage to the small vessels that causes the highest post-recanalization hemorrhage into the basal ganglia [56].

NEUROVASCULAR RESCUE

Treatment of Iatrogenic Peri-procedural Acute Thromboembolic Stroke: Embolization of thrombotic or atherosclerotic material can be caused by instrumentation through the aortic arch, in the ventricle, or in the supra-aortic vessels, including the carotid arteries. It is an uncommon but important complication from endovascular intervention. Experienced operators with compulsive attention to meticulous techniques coupled with careful patient selection should encounter this problem in less than 1% of coronary angioplasty or 13% of carotid interventions [63]. It is important to assess the neurological status of the patient after every step of the procedure. Even minute changes in the level of consciousness, minimal confusion, slurred speech, or other changes in the neurological status can indicate distal embolization [54]. During endovascular interventions, especially in the carotid territory, a change in neurological status should initiate the actions listed in Table 28-3 [54].

A diagnostic angiogram is done in the the anteroposterior and lateral projections. The angiograms are carefully examined to determine the site and extent of intracranial vessel embolism. Because of anatomical arrangements and flow pattern, the most likely site of intracranial embolism is the distal internal carotid artery and the middle cerebral artery and its branches. Large vessel occlusion is usually obvious (especially in lateral projection), but embolism in the smaller branches requires careful scrutiny. Acute small branch vessel occlusion may be noted only in comparison with preprocedural angiography. The availability of a good preprocedural intracranial angiogram is therefore essential in all patients undergoing supra-aortic vessel stenting. Change of neurological status can ensue not only from occlusive phenomena such as embolus, but also from intracerebral hemorrhage or hyperperfusion syndrome (especially if very tight stenosis or an occluded artery is opened). If on the diagnostic angiogram there are no signs of embolism, a CT scan should be performed forthwith. If there are signs of localized expanding phenomenon indicating intracerebral hemorrhage, heparin should be reversed and a CT scan performed [64].

Table 28-3 General Care of Patients with Periprocedural Ischemic Stroke

General care of the patient should be instituted
Maintaining normal blood pressure, heart rate
Maintaining airways and administration of oxygen
In case of carotid procedure, the stenting should be quickly and efficiently completed, including stent deployment. Additional maneuvers will involve working through the stented segment to access distal vasculature
If the patient becomes uncooperative or agitated, and especially if there are compromised airways, the intervention of an anesthesiologist is required

Equipment and Adjunctive Medications Needed for Neurovascular Rescue: The sheath within the common carotid artery is the basic support for advancement of the variable-stiffness microcatheter into the intracranial circulation. Through this sheath, a guide catheter is advanced into the ICA over a wire. This guide should not be advanced deeply into the internal carotid artery if there are tortuosities in this vessel. This would produce spasm or even dissection. Through this guide (5 or 6F), a variable-stiffness microcatheter (2.3–3.0French) is advanced. All of these catheters should be continuously perfused with heparinized saline, which should be appropriately prepared, dripped, and filtered without air to avoid stroke producing air emboli. The variable-stiffness microcatheter is advanced over the wire into the intracranial segment of the internal carotid artery. There are a variety of neurovascular wires that are now commercially available from a variety of vendors. Choice of wire is based an individual patient's vascular anatomy, favoring wires with good torqability, adequate stiffness, and atraumatic malleable tips. If the position of the embolus is obvious (in the internal carotid artery or horizontal M1 segment of the middle cerebral artery), no additional arteriography is needed. If the occlusive lesion is within the trifurcation of the middle cerebral artery or distally, an angiogram through the variable-stiffness microcatheter is performed (using 1–3 cc syringe) in both projections, so the occluded branch of the middle cerebral artery is identified and subsequently entered. Road mapping is useful. Before the recanalization is attempted, the length of the occluded vessel segment should be determined. This can be done by careful analysis of the diagnostic angiogram by looking for retrograde flow into the occluded artery. If there is no retrograde flow, the variable-stiffness microcatheter is passed through the embolus over the wire distally. Rotating movement of the distal curved end of the wire usually shows a patent vessel. The wire is withdrawn and slow injection of contrast through the microcatheter is undertaken, while the catheter is slowly pulled back simultaneously. This determines the distal end of the embolus, which can be both "soft" and "hard." The soft one consists of blood coagulation and is prone to lysis. The hard one consists of stenotic debris (part of the plaque with cholesterol and calcium) and cannot be lysed, but it can be mechanically disrupted. There are three techniques to reopen an acutely occluded intracranial artery: thrombolysis, mechanical disruption, and removal of the embolus [54].

THE FUTURE

Endovascular intracranial intervention with PTA/SAA is an FDA approved therapy for appropriately selected high-risk IAD patients who have failed medical therapy. Careful patient selection and highly experienced teams are required to achieve a clinical benefit. Prospective multi-center trials need to be performed to more precisely define indications, therapeutic strategies, and perioperative medical regimens. Technological and strategic innovations remain ongoing in

efforts to advance clinical efficacy and establish a role for this promising new alternative for these high-risk patients.

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Chapter 29
Percutaneous Interventions in Adults with Congenital Heart Disease

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*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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Technical tips
**How to position the sheath perpendicular to the atrial septum
**Final check of device position
Technique: Post placement assessment
Indications
Contraindications

Patent Ductus Arteriosus
Pre-procedure evaluation/management
Technical tips
**Misleading systolic flow mimicking PDA
Technique: Defining the anatomy
Technical tips
**Locating the point of minimal diameter of the PDA
**Crossing the PDA from the aorta
Technique: Choosing device size
Technical tips
**Use fewer coils in PDA closure
Technique: Sheath placement
Technical tips
**Inserting the sheath into the RVOT
Technique: Device positioning
Technical tips
**Avoiding first coil embolism while deploying the second coil
**Avoiding entangling the already deployed coil in the directional wire
**Checking the position of device prior to deployment
**Closing the PDA with occluder
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Contraindications

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Technique: Choosing balloon and stent size
Technical tips
**Sequential dilation of the coarctation
**Advantage of MRI compatible stents
Technique: Sheath and wire placement
Technical tips
**Optimal wire position
Technique: Balloon and stent positioning
Technical tips
**No problem with subclavian jailing
**Post-dilation with high pressure balloon
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Technical tips
**Accurate pressure gradient measurement

Indications
Contraindications

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Technique: Sheath and wire placement
Technical tips
**How to track the balloon across the valve
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Post-dilation assessment
Indications
Contraindications

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Technique: Choosing balloon and stent size
Technique: Sheath and wire placement
Technical tips
**Stabilizing wire position in the pulmonary artery branch
Technique: Stent position and implantation
Technical tips
**Using a stiffer wire to track a stent
Technique: Post dilation assessment
Technical tips
**Reshaping the pigtail catheter
Indications
Contraindications

Pulmonary Insufficiency – Percutaneous Valve Implant
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Technique: Defining the anatomy
Technique: Choosing valve stent size
Technique: Sheath and wire placement
Technical tips
**Crossing the RVOT
Technique: Stent position and implantation
Technique: Post-dilation assessment
Indications
Technical tips
*Be careful of compression on proximal organs
Contraindications

GENERAL OVERVIEW

Adult patients with congenital heart disease are an exponentially increasing population due to improved treatment strategies for children resulting in excellent long term survival. In many countries around the world 2007 will usher in a new era in which adults with congenital

heart disease outnumber children [1]. Newer interventional techniques and tools developed over the last 20 years are now able to treat the majority of common congenital lesions in the catheterization laboratory instead of the operating suite. This chapter will detail percutaneous interventional techniques for treating the most common congenital cardiac lesions seen in adults including patent foramen ovale (PFO), atrial septal defect (ASD), patent ducts arteriosus (PDA), coarctation of the aorta, valvar pulmonary stensosis, pulmonary insufficiency (PI), and branch pulmonary artery stenosis.

PATENT FORAMEN OVALE

Device closure of PFO was first described in 1987 [2] for the prevention of recurrent stroke associated with paradoxical embolus [3]. It has also been used to prevent right to left shunting causing desaturation in patients with orthodeoxea – platypnea syndrome [4], as well as the bends in deep sea divers [5]. The foramen ovale is a flap valve in the atrial septum created by overlap of the superior anterior septum secundum on the inferior posterior septum primum (Figure 29-1). It is present in all fetuses during development to direct oxygenated venous

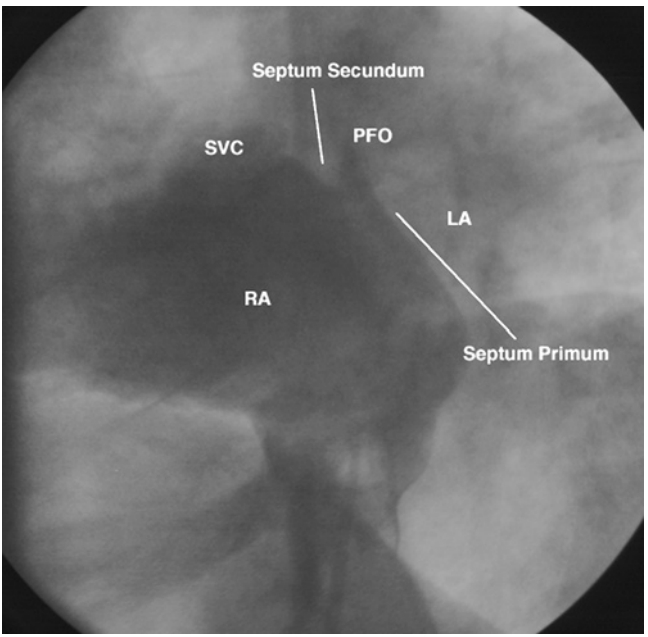

Figure 29-1 Lateral right atrial angiogram showing typical patent foramen ovale anatomy with a thin septum primum and thick septum secundum. LA, left atrium, PFO, patent foramen ovale, RA, right atrium, SVC, superior vena cava.

return from the placenta through the interior vena cava (IVC) across the atrial septum, bypassing the right ventricle (RV) and unexpanded lungs, to fill the left ventricle allowing optimal cerebral perfusion. After birth, with redistribution of flow due to lung expansion resulting in an increased left atrial pressure, the PFO closes and seals permanently in 65–80% of people, age dependent [6]. However, in 20–35% of the normal population the foramen ovale does not fibrous closed and remains patent allowing unidirectional flow from right to left if right atrial (RA) pressure exceeds left atrial (LA) pressure. This is physiologically insignificant for most people unless the amount of right to left shunting is significant causing orhtodeoxia–platypnea syndrome or an embolus crosses right to left resulting in a cryptogenic transient ischemic attack (TIA) or stroke. Approximately 55% of patients who have had a stroke have a PFO [7] suggesting it plays an important role in many of these patients. There is recent data suggesting at least some severe migraine sufferers with aura may have right to left shunting of microemboli through the PFO as a cause of their headaches [8].

Over the last 14 years interventional device closure of patent foramen ovale has become an attractive alternative to surgical PFO closure or lifelong anticoagulation for stroke prevention. No large prospective comparative studies between treatment strategies exist for PFO closure although several prospective multi-center studies in stroke and migraine patients are underway. Many retrospective studies and several small prospective comparison studies suggest comparable results between medication and device closure strategies for recurrent stroke prevention [9]. Retrospective data and early results from prospective trials suggest a small but significant reduction in migraine headaches with PFO closure in select migraine sufferers [10]. Procedural success with PFO device closure is 98 to 100% with complete closure rates of 51% to 96% at 6 months if evaluated by saline contrast transesophageal echocardiography (TEE) [11–14]. Recurrent neurologic event risk following PFO device closure is 1 to 2% annually with a 96% 1-year, and 90 to 94% 5-year, event free rate [11–14]. These results are significantly influenced by patient selection since some patients who undergo device closure may have recurrent strokes unrelated to either the PFO or device. More definitive information regarding recurrent stroke risk will be available from controlled randomized trials underway. Procedural complications are uncommon occurring in less than 2% and include stroke, TIA, transient myocardial ischemia (the latter three due to air or clot embolism with the large delivery sheaths in the left atrium), device malposition or embolization, cardiac perforation with tamponade, and local femoral vein injury [11]. Late complications include atrial arrhythmias in 4%, although most are mild requiring no treatment [15], and thrombus formation on the device.

There are currently six devices in use worldwide for PFO closure: the Amplatzer PFO Occluder, the Buttoned device, the CardioSEAL STARFlex septal occluder, the Gaurdian Angel device, the Helix septal occluder, and the Cardia Star (Cardia Inc., Burnsville MN) (Table 29-1). Common features in all include a metal frame supporting two patches, left and

Table 29-1 Comparison of PFO and ASD devices

Device		Frame	Material	Sizes	Delivery sheath
Amplatzer PFO		Nitinol wire	Polyester fabric	18, 25, 35	9
□ ASD				4–40	6–12
Button PFO		Teflon coated stainless steel wire	Polyurethane foam	25–30	7–9
□ ASD				25–60	9
CardioSEAL		MPN35	Polyester fabric	17–40	10
Guardian Angel		Nitinol wire	Polyester fabric	18–30	10
Helix		Nitinol wire	PTFE	15–35	9
PFO Star		Nitinol wire	Ivalon plug	15–35	10

right atrial patches, which are connected by a central core. These devices are folded or stretched into a loader to minimize their diameter for delivery through a 9 or 10 French sheath positioned across the PFO. Once delivered from the sheath the devices expand into position and immediately obstruct flow mechanically by covering the flap valve. The final and complete seal comes from endocardial in-growth covering the patches completely within 8 to 12 weeks. Device implantation is most typically guided by both fluoroscopy, and echocardiography (either transesophageal or intracardiac), although either alone will suffice.

New techniques are in development to close the PFO without residual implants. The two most promising techniques currently are suture closure and radio-frequency ablation fusion. The latter technique uses a catheter with a flat plate electrode positioned against the right atrial side of the foramenal flap through which radio-frequency energy is applied. The energy heats the septum primum and secundum which overlap, melting collagen, and causing closure. Preliminary trials in stroke and migraine patients show over 95% complete closure rates in PFOs with minimal complications [16,17].

Pre-procedure Evaluation/Management: Because most patients undergo PFO device closure for prevention of stroke, recurrence it is essential to evaluate the patient's prior neurologic events and assure they were crytogenic associated with the PFO. Stroke associated with paradoxical embolism is a diagnosis of exclusion so it is imperative to rule out other potential causes of stroke, including cerebral aneurysm, carotid or vertebral vessel abnormalities, atrial arrhythmias, left atrial appendage thrombus, cardiomyopathy or a hypercoagulable state. Standard pre device closure evaluation includes head and neck MRI/MRA, carotid ultrasound, transesophageal echocardiogram with

saline contrast, and hyper-coagulable screen including protein C and S, antithrombin III, factor V Leiden, prothrombin 20210, MTHFR, anticardiolipin antibody, and homocysteine. This latter work up is essential to help guide decisions regarding the appropriateness of implanting a device and the optimal medical strategy during the endocardialization process. Because of a small incidence of atrial arrhythmias after device placement a baseline EKG should also be obtained. If patients are on Coumadin before the procedure they should hold the dose two days before and begin daily aspirin to minimize both bleeding risk associated with the procedure and clot formation on the implant immediately after the procedure. ACT is maintained at >250 seconds during the procedure with IV heparin. Local anesthesia and mild to moderate sedation are used to maintain patient comfort. A dose of antibiotics (Kefsol or Clindamycin) is given IV prior to device implantation to prophylax against procedural related sepsis / endocarditis.

TECHNIQUE Defining the Anatomy: A 9 or 10 French sheath is placed in the femoral vein and right heart catheterization is performed using a Berman balloon tipped or multipurpose catheter with measurement of pressures and saturations in the superior vena cava (SVC), RA, RV and pulmonary arterial systolic pressure (PAS) to assure normal physiology and no evidence for significant left to right intracardiac shunt (to exclude additional pathology, especially an additional ASD or anomalous pulmonary vein). An angiogram is then performed in the low RA with the anterior-posterior (AP) camera angled 20 degrees right anterior oblique (RAO) and 20 degrees cranial, and the lateral camera 70 degrees left anterior oblique (LAO) and 10 degrees caudal. 24 cc of contrast is injected at a rate of 24 cc/sec. The lateral projection will profile the PFO nicely (Figure 29-1) and can be used as a roadmap for device delivery.

TECHNICAL TIPS

****Shaping the Tip of the Catheter in Order to Enter the PFO:** If the anatomy or size of the defect is in question cross the PFO with the Berman catheter by inserting the stiff end of a 0.035" straight wire shaped with a 45 degree angle at the distal 4 cm. This will give the end of the Berman catheter a "hockey stick" shape that can be easily directed slightly leftward and posterior to slip through the tunnel PFO. The balloon on the Berman catheter can then be inflated and the catheter withdrawn to the septum against the foramenal flap pulling it closed (Figure 29-2). Record an image of the balloon against the septum to create an additional roadmap for placement of the LA side of the device when appropriately positioned against the foramenal flap. If the inflated Berman balloon easily pulls through the defect reassess the anatomy and consider a larger device.

Occasionally a pre procedural echo will suggest a PFO with right to left shunting seen during saline contrast yet no PFO can be demonstrated by angiography or with catheter probing of the atrial septum.

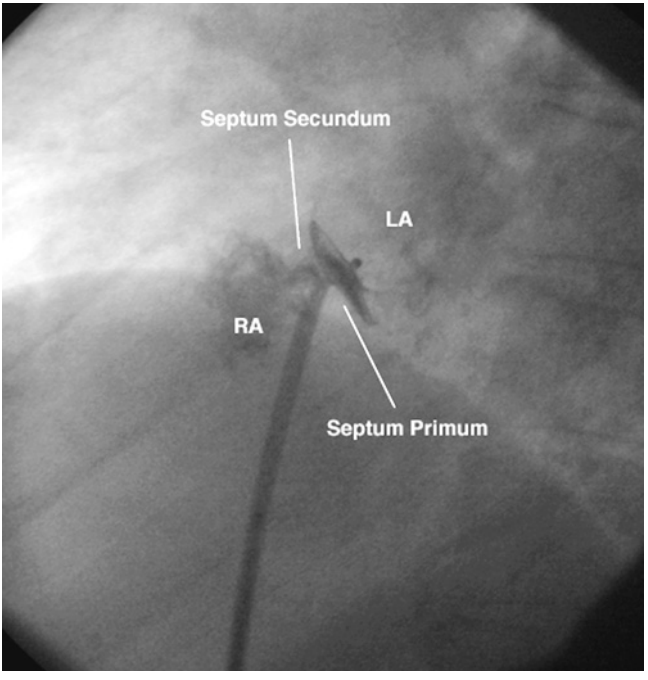

Figure 29-2 Lateral angiogram through long sheath in RA showing the LA side of an 18mm AGA PFO ocludder snug against the septum.

Consider the diagnosis of pulmonary aterio–venous malformations that are associated with paradoxical embolism and will have right to left contrast shunting on echo that can be mistaken for a PFO shunt. Perform selective right and left pulmonary artery angiography to make the diagnosis. If present these can be treated with coil embolization.

TECHNIQUE Choosing Device Size: In general, the smallest device which effectively covers the defect should be used to minimize foreign body mass and optimize closure rates. Most PFOs are 4–6 mm in diameter and stretch minimally in the left to right direction. Some operators use balloon stretch diameter to assist with device size choice. We have not found this helpful unless the anatomy is poorly defined on angiography or an ASD is suspected. For the Amplatzer device either the 18 mm or 25 mm devices suffice for most defects. If the right atrial side of the defect is quite large or a large atrial septal aneurysm is present then the larger 35 mm device can be used assuming the total atrial size is adequate. For the STARFlex device the 23 or 28 mm devices are adequate for most defects with the 33 mm device chosen for exceptionally large defects or those with large atrial septal aneurysms.

TECHNIQUE Sheath Placement: The sheaths required for device closure are large but easily pass through the foramen ovale over a guide wire positioned in a left pulmonary vein, preferably the left upper. A multipurpose or directional end hole catheter such as a JR4 can be used to direct a stiff 0.035" wire with a soft tip (Rosen or Amplatz) through the PFO and into the left upper pulmonary vein (LUPV). The sheath and dilator are then advanced into the vein, wire and dilator removed and sheath cleared. It is imperative that these sheaths are cleared carefully because air embolism is directly into the systemic circulation and is by far the most common and serious side effect associated with this procedure.

TECHNICAL TIPS

****Backbleed and Flush the Sheath in Order to Avoid Air Embolism:** Flush the sheath continuously when advancing into the LA and during removal of the dilator and wire. Refrain from negative suction on these large sheaths. Allow passive bleed back and keep the end of the sheath significantly below the level of the patient's heart to facilitate bleed back. Be aware of the patients breathing and be sure to time clearance of the sheath with exhalation to minimize the risk of air embolism. Give supplemental nasal cannula O_2 during sheath and device placement to minimize effects if air embolism occurs.

TECHNIQUE Device Positioning: The device is soaked in heparinized saline and inspected for defects. It is compressed into the loader with constant flushing to remove any residual air and loaded into the sheath. Connect a large syringe with contrast to the side arm of the delivery sheath for hand angiography during device positioning. Withdraw the sheath tip to the middle of the left atrium and advance the distal patch of the device out of the sheath until it expands completely. Withdraw the sheath and device together until the device is in firm contact with the septum. You will feel the beat of the heart on the end of the sheath and can confirm position with your RA angiographic and balloon sizing roadmaps. Perform a hand angiogram through the delivery sheath. This should outline the RA side of the septum and confirm the distal patch position on the left atrial side snug against the septum (Figure 29-2). If the angiogram shows marked filling of the left atrium the device and sheath need to be pulled more tightly against the septum. Once appropriate LA patch position is confirmed the device is held firmly in place and the sheath withdrawn over the device uncovering the right atrial patch. Once the right atrial patch is completely open move the sheath and delivery cable to a neutral position and repeat a hand angiogram through the sheath to confirm optimal position. A small residual leak through the center or edge of the device is not atypical with this injection due to distortion of the device from the plane of the septum while connected to the delivery cable. If in appropriate position the device is released and the delivery cable removed.

TECHNICAL TIPS

****Device Positioning Across a Thick Septum Primum and a Stiff Tunnel Defect:** Occasionally the septum primum is extremely thick and creates a rigid tunnel that cannot be displaced by exerting pull on the left atrial patch. This can be recognized prior to device release by an inability to position the center point of the device on the right atrial side of the septum because the entire device is held up in the left atrial side of the stiff tunnel. After release the device will not lie flat to the septum but the inferior portion of the left atrial patch and the superior portion of the right atrial patch will protrude from the septum due to malposition (Figure 29-3). This can be avoided by performing a transeptal puncture in the thick septum primum, just below the foramenal opening. TEE or intra-cardiac echocardiography (ICE) guidance is needed to assist with optimal puncture site location. The long sheath is passed through the transeptal puncture site and the device positioned in the transeptal defect resulting in coverage of

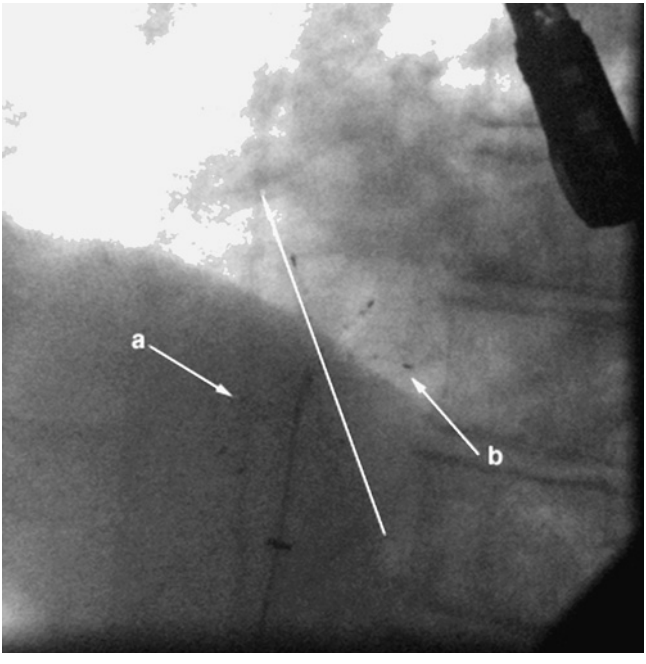

Figure 29-3 Lateral RA angiogram of malposition of a Starflex device in a PFO. Line denotes plane of the septum. Note that the superior right atrial arm (arrow a) and inferior left atrial arm (arrow b) are away from the septum indicating poor position due to a rigid septum primum maintaining the tunnel shape to the PFO.

the foramen without crossing it. This allows for excellent closure while avoiding device distortion due to the rigid foramenal tunnel.

TECHNIQUE Post Placement Assessment: Repeat pressure and saturation measurements in the RA should be performed to assure hemodynamic stability post device. An angiogram at the SVC-RA junction consisting of 24 cc of contrast injected at 24 cc/sec should be performed to confirm device position and evaluate for residual right to left shunting. The cameras are positioned to evaluate the device in the AP plane (usually 15 degrees RAO and 10 degrees caudal) and on profile in the lateral plane (75 degrees LAO and 5 degrees caudal). If echocardiographic assessment is used then a saline contrast echo should be performed to evaluate right to left shunting.

Indications: Potential indications for PFO device closure include any patient who has had or has substantial risk for a cryptogenic stroke in the setting of a PFO. Absolute indications for PFO device closure remain controversial since there is limited controlled data comparing different treatment strategies and evaluating long term follow up. However several clinical situations clearly warrant device closure including: patients with active venous thrombus in the setting of a cryptogenic stroke, patients with recurrent cryptogenic stroke while on anticoagulation, patients with recurrent cryptogenic stroke and contraindications to anticoagulation, and scuba divers who have had significant decompression sickness but insist on continuing to dive. Based on current data PFO device closure is a reasonable therapeutic alternative for patients with an initial cryptogenic stroke and no additional risk factors.

Contraindications: There are no absolute contraindications for device PFO closure except for patients with active thrombus in the LA and those with a known allergy to the device implant materials, particularly the nickel in Nitinol, an extremely rare condition. Patients who are hyper-coagulable, particularly those with disorders that predispose to arterial clots, should be considered very carefully as the post placement risk of clot formation during the endocardialization process may be significantly increased. However, those patients who are predisposed to venous clots may be the very patients who benefit the most in the long term, albeit with a potentially increased thrombus risk during the first 6 months after implant. Patients who require anticoagulation long term for other issues may get limited benefit from device closure.

ATRIAL SEPTAL DEFECT CLOSURE

Secundum ASDs are one of the more common congenital heart defects making up 6–10% of all congenital anomalies, occurring in 1/1500 live births [18]. Anatomically secundum ASDs are due to absence, perforation or deficiency of the septum primum. This defect

typically occurs sporadically but has been linked to genetic abnormalities such as Holt Oram syndrome and mutations on chromosome 5p.

Device closure of an ASD was first performed in 1974 by King and Mills [19,20] using a 24 gauge surgically placed femoral sheath and a double-sided disc device. Technology and technique have been modified and refined over the years, however, the procedure remains conceptually identical. A collapsible double-sided disc device with a metal frame and fabric patches is positioned antegrade through a long femoral sheath across the secundum ASD. Upon extrusion from the sheath the device expands creating a patch on both sides of the septum clamping the surrounding ASD tissue rim. The endocardium grows in to cover the device and create a permanent seal. Because of the need for surrounding rim tissue device closure is limited to secundum type defects, not applicable to either primum (no inferior posterior rim) or venosus (no superior rim) ASDs. With recent technological advances device closure has rapidly become the treatment of choice for secundum ASDs.

Concurrent controlled trials comparing surgical closure with device closure has shown efficacy rates of over 96% with significantly fewer complications and shorter hospital stay [21]. Most patients can be discharged on the day of the procedure with return to full activity within 48 to 72 hours, significantly reducing costs and medical resources [22]. Early complications have been minor occurring in $<9\%$ of patients consisting primarily of transient arrhythmias, vascular injury or asymptomatic device embolization. Serious complications have been quite rare but include thrombus formation on the device, heart block requiring pacing, and cardiac perforation [23].

There are currently four devices used recently for ASD closure including the Amplatzer septal occludder, CardioSEAL STARflex, Helix, and the Button device (Table 29.1). By far the most commonly used device, and the one capable of closing the largest ASD's, is the Amplatzer septal occluder. Unlike the others this device has a central stenting mechanism that expands to the edges of the defect filling it with frame and patch material improving stability and complete closure rates in large ASD's. It is available in sizes up to 4 cm capable of closing a 3.8 cm defect. The combined global experience of these devices for ASD closure is well over 50,000 patients with extremely high success and low complication rates.

Pre-procedure Evaluation/Management: A complete echocardiogram, omniplane transesophageal if the patient is an older adolescent or adult, is optimal to define the atrial septal anatomy prior to the procedure. Secundum ASDs are rarely found so attention to defect dimensions in multiple planes is essential for a complete anatomical understanding. Documentation of an adequate atrial septal rim circumferentially (>3 mm, especially at the posterior inferior inlet portion), and evaluation for additional defects, tissue strands or septal aneurysms with perforations is essential (Figure 29-4).

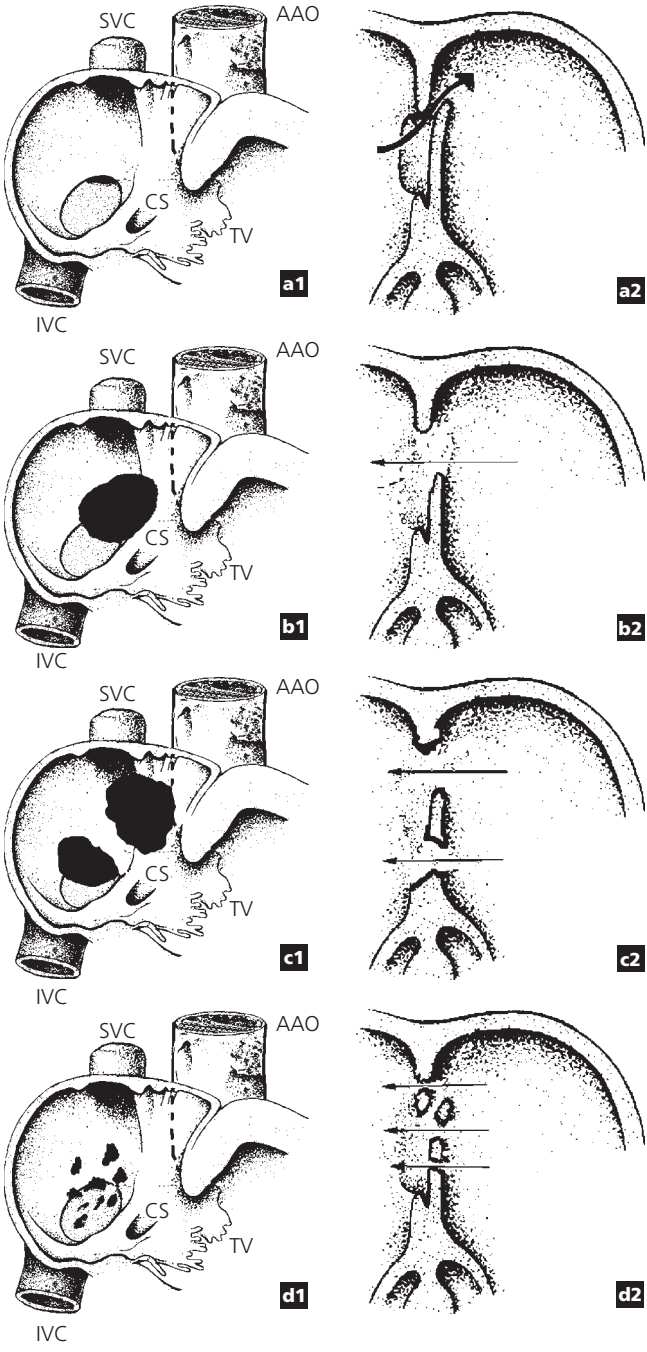

Figure 29-4 En faus and lateral view schematic drawing of the atrial septum: (a) patent foramen ovale, (b) isolated secundum ASD, (c) multiple defects, (d) fenestrated defects. Ao, ascending aorta, CS, coronary sinus, IVC, inferior vena cava, SVC, superior vena cava.

TECHNICAL TIPS

****Identification of Different Types of ASD:** Identification of all pulmonary veins, particularly the right upper, is essential due to the association of partial anomalous pulmonary venous return with sinus venosus ASD. Sinus venosus defects should not be closed by device as this will only complicate surgical repair of the anomalously draining vein.

Because of a small incidence of atrial arrhythmias after device placement, a baseline EKG should also be obtained. Begin daily aspirin 2 days before the procedure to prevent clot formation on the implant immediately after the procedure. If patients are on Coumadin before the procedure they should hold the dose two days before. The ACT is maintained at >250 seconds during the procedure with IV heparin. Local anesthesia and mild to moderate sedation are used to maintain patient comfort. A dose of antibiotics (Kefsol or Clindamycin) is given IV prior to device implantation for prophylaxis against procedural related sepsis/endocarditis.

TECHNIQUE Defining the Anatomy: An 8 or 10 French sheath is placed in the femoral vein and right heart catheterization is performed using a Berman balloon tipped or multipurpose catheter with measurement of pressures and saturations in the SVC, RA, RV and PAs to assess the degree of left to right intra-cardiac shunt, exclude pulmonary hypertension and additional pathology, especially anomalous pulmonary vein. An angiogram is then performed in the right upper pulmonary vein (RUPV) (this promotes contrast flow along the atrial septum to define the ASD optimally) with the AP camera angled 20 degrees RAO and 20 degrees cranial, and the lateral camera 70 degrees LAO and 10 degrees caudal. 24 cc of contrast is injected at a rate of 24 cc/sec. The lateral projection will profile the ASD nicely while the AP camera will define LA free wall landmarks so that both can be used as roadmaps for device delivery (Figure 29-5). Echocardiographic evaluation of the ASD is performed using either transesophageal or intracardiac echocardiography (ICE) and will be used for device placement guidance as well as post placement evaluation.

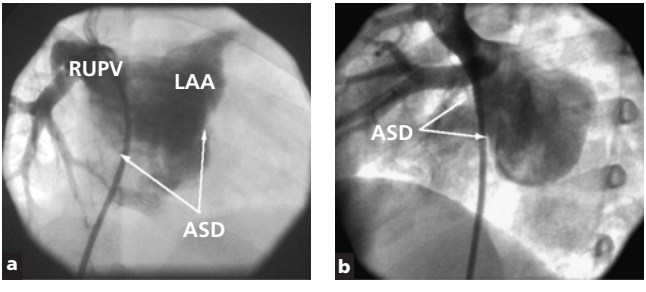

Figure 29-5 AP and lateral angiogram of a secundum ASD.

TECHNICAL TIPS

****Reshaping the Tip of the Catheter to Enter the ASD:** Cross the ASD with the Berman catheter by inserting the stiff end of a 0.035" straight wire shaped with a 45 degree angle at the distal 3 cm. This will give the end of the Berman catheter a "hockey stick" shape that can be easily directed slightly leftward and posterior to slip through the ASD. Clockwise rotation then turns the tip into the right upper pulmonary vein.

Balloon sizing of the defect is then performed. Exchange the Berman catheter for a directional, JR4 or Bentson tip to direct a 0.035" wire through the ASD into the left upper pulmonary vein. Position a compliant sizing balloon (both AGA and NMT make ASD sizing balloon up to 3.5 cm in diameter) with reference markers across the defect and inflate color Doppler flow signal is gone. Measure the occlusion diameter on AP and lateral angiogram as the echocardiographic measurement may not be as accurate or reliable.

****Detecting Additional Defects:** It is essential to evaluate the defect with echocardiography while the defect is occluded with the balloon. This allows careful assessment of the septum for additional defects and assures accurate occlusion diameter measurement by confirming absence of residual shunt with the balloon. We prefer ICE assessment due to improved patient comfort, reduced need for deep sedation and reduced need for echo personnel support. In general ICE has been equivalent to TEE for assessing the atrial septum in experienced hands.

TECHNIQUE Choosing Device Size: In general, the smallest device that effectively covers the defect should be used to minimize foreign body mass, and interference with intra-cardiac structures such as AV valves or pulmonary vein/SVC inflow. Total septal length should be measured both angiographically and by echo to determine the largest device that can safely fit in the patient's atrium. Specific sizing depends on the type of device being used. In general, for the CardioSEAL STARFlex, Button, and Helix devices the size should be chosen roughly twice the occlusion diameter of the defect. The Amplatzer device, which is sized by the central stent diameter, should be 2–4 mm larger than the occlusion diameter.

TECHNICAL TIPS

****Selecting the Size of the Device:** The smaller the defect occlusion diameter the less you need to oversize devices, especially the Amplatzer device. Also the more effective are the softer STARflex and Helix devices. For defects <16 mm we often use devices equal to the stretch diameter, for defects 17 to 32 mm we use the stretch diameter $+2$ mm and for very large defects >32 mm we will oversize by 4 mm. If there is limited rim, particularly in the inferior portion, posterior portion or the anterior superior portion (aortic region on echo short axis) of the defect we will use the Amplatzer device and oversize

by 3 or 4 mm from the occlusion diameter. For the other devices the same concept holds, if there is limited rim in a region, choose a device closer to 2.5 × stretch diameter if total atrial chamber size will allow.

TECHNIQUE Sheath Placement: The sheaths required for device closure range in size from 6 to 12 French depending on device type and size. Because air embolus remains one of the major concerns and causes of significant complications, proper flushing of the sheath is imperative. Use of a curved tip sheath that can be manipulated directly from the RA to the LA without the use of a guide wire is preferred. The long sheath is placed in the RA over a wire, the wire and dilator removed and the sheath cleared of air and flushed. The sheath is then manipulated across the defect into the LA for placement of the device. For small defects the tip of the sheath can be positioned in the center of the LA, for large defects the tip should be positioned in the mouth of the right or left upper pulmonary vein.

TECHNICAL TIPS

****Sheath Placement in Fenestrated ASDs:** Sheath placement should be modified for fenestrated or multiple defect closure. In these cases proper placement of the sheath across the exact defect of interest is crucial to the success of the procedure. To assure the sheath crosses the same defect that was balloon sized, a long 0.035" guidewire should be left across the defect of interest in the left upper pulmonary vein and the long sheath exchanged over the wire for the balloon sizing catheter. Flush the sheath continuously when advancing into the LA and during removal of the dilator and wire. Refrain from negative suction on these large sheaths. Allow passive bleed back and keep the end of the sheath significantly below the level of the patient's heart to facilitate bleed back. Be aware of the patients breathing and be sure to time clearance of the sheath with exhalation to minimize the risk of air embolism. Give supplemental nasal cannula O_2 during sheath and device placement to minimize effects if air embolism occurs.

TECHNIQUE Device Positioning: The device is soaked in heparinized saline and inspected for defects. It is compressed into the loader with constant flushing to remove any residual air and loaded into the sheath. The tip of the sheath is positioned in the mid LA and the distal disc of the device opened. Use angiographic and echocardiographic landmarks to assure the device is not opened in a pulmonary vein or pressed against the LA roof.

TECHNICAL TIPS

****How to Position the Sheath Perpendicular to the Atrial Septum:** Often the angle of the sheath with the atrial septum is quite acute making the approach of the device to the septum difficult often resulting in device edge prolapse particularly in the anterior superior

region (especially if limited aortic knob rim) or superior SVC region (especially if a superiorly located defect). To improve the angle and bring the device more perpendicular to the atrial septum, rotate the sheath clockwise to drive the tip of the sheath posterior and superior. The sheath can be shaped with a posterior superior curve to improve device alignment (Cook Inc. currently has a commercial sheath available with this bend on the tip called a Lock-Hausdorf sheath). Another trick to align the device with the septum in this situation is to cut the tip of the sheath creating an oval beveled hole. The device then exits the sheath at an angle but care must be taken to advance the modified sheath in the heart with the dilator until in position for device delivery [24].

The LA side of the device is brought back toward the atrial septum but not snug as with PFO closure because this will promote device prolapse into the RA. For the CardioSEAL STARFlex type device the centerpin of the device should be kept slightly into the LA side of the septum for RA disc delivery. Both the Amplatzer and Helix devices can be kept centered on the atrial septum during RA disc delivery.

****Final Check of Device Position:** The larger the defect the further into the LA the device center should be kept during RA disc delivery to prevent LA disc prolapse into the RA.

After RA disc delivery but before device release compete echocardiographic assessment of the device and the relationship to surrounding structures must be completed. Evaluation for new onset TR or MR, residual left to right ASD flow, and obstruction of SVC or right upper pulmonary vein flow all must be carefully assessed. For the STARFlex device all frame arms must be identified on the appropriate side of the septum. For the Amplatzer device atrial septum must be identified between the two discs circumferentially. Pulling and pushing slightly on the delivery cable to separate the two discs will facilitate this process and confirms device stability. If in appropriate position the device is released and the delivery cable removed.

TECHNIQUE Post Placement Assessment: Repeat pressure and saturation measurements throughout the right heart should be performed to assure hemodynamic stability post device and assess residual shunt. An angiogram in the main pulmonary artery (MPA) or right pulmonary artery (RPA) consisting of 24 cc of contrast injected at 24 cc/sec should be performed to confirm device position and evaluate for residual left to right shunt. The cameras can be positioned to evaluate the device on faux in the AP plane (usually 15 degrees RAO and 10 degrees caudal) and on profile in the lateral plane (75 degrees LAO and 5 degrees cranial). Echocardiographic assessment should be repeated following device release to assess final device position and residual shunt.

Indications: Indications for ASD device closure include any size secundum ASD with evidence on echocardiogram of right ventricular volume overload. Patients with ASD and symptoms of exercise intolerance or history of cryptogenic stroke should also be closed. There

is mounting evidence that closure of small ASDs, even in the elderly, improves maximal oxygen consumption [25]. ASDs can and have been closed by device in small children including infants, however, the optimal timing for elective closure appears to be between 2 and 4 years of age.

Contraindications: There are no absolute contraindications for device ASD closure except for patients with active thrombus in the LA and those with a known allergy to the device implant materials, particularly the nickel in Nitinol, an extremely rare condition. Patients who are hypercoagulable, particularly those with disorders that predispose to arterial clots, should be considered very carefully as the post placement risk of clot formation during the endocardialization process may be significantly increased. Patients with existing IVC filters can be closed by advancing the sheath through the filter, or approaching the ASD from the internal jugular or hepatic veins. Patients with significant left ventricular dysfunction also must be monitored closely after the procedure due to possible acute LA hypertension resulting in pulmonary edema. Diuretics immediately post closure may be very helpful in this subgroup of patients. Patients with pulmonary hypertension must be considered carefully but may benefit as long as there is a baseline left to right shunt [26].

PATENT DUCTUS ARTERIOSUS

PDA is the persistence of a normal fetal connection between the proximal descending aorta and proximal left pulmonary artery which allows the right ventricle to bypass the lungs and pump deoxygenated blood via the descending aorta to the placenta for oxygenation. Normal ductal closure occurs within the first 12 hours after birth by contraction and cellular migration of the medial smooth muscle in the wall of the ductus resulting in protrusion of the thickened intima into the lumen causing functional closure. Final closure and creation of the ligamentum arteriosum is completed by 3 weeks of age with permanent sealing of the duct by in folding of the endothelium, disruption of the internal elastic lamina, and hemorrhage and necrosis in the subintimal region leading to replacement of muscle fibers with fibrosis. This process of closure is incomplete in 1/2000 live births and accounts for up to 10% of all congenital heart disease [27].

PDA closure was one of the first congenital heart lesions treated by interventional techniques, first reported by Dr Porstmann in 1968 [28,29]. There have been substantial refinements in devices and techniques over the last 35 years but for the last 15 years interventional catheter treatment has been the preferred therapy in many large centers worldwide. It is a particularly attractive technique in adults in whom surgical ligation and division can be problematic due to calcified ductal tissue and increased surgical risks. The technique is simple consisting of placement of a device or vascular occlusion coil in the PDA either antegrade from the femoral vein or retrograde from the femoral artery. Once implanted the device physically occludes ductal

flow and over the first 6 to 8 weeks after implant endothelial over growth covers the device or coil from both the pulmonary artery and aorta sealing the PDA permanently closed.

Several different closure devices are currently used due to the significant variability of ductal anatomy. The most common anatomical shape is conical with a large aortic ampulla that narrows at the pulmonary artery end, however, other distinct anatomical forms exist including "tubular" without a narrowing at the pulmonary artery end, "complex" with narrowing at both the aortic and pulmonary end, and a short "window" which is an anatomy commonly found in adults [30]. Different closure tools and techniques may be needed to effectively address these less common PDA anatomical subtypes, however, this section will focus on the two most common closure techniques for the conical shaped ductus. The most commonly used technique for closure of PDAs less than 3 mm is retrograde placement of embolization coils. For larger ducts antegrade placement of an Amplatzer duct occlude device is the preferred method. These two techniques are described below.

Transcatheter ductal closure procedural success has been extremely high with rates of complete closure >96% [31–36]. The procedure takes approximately 2 hours with discharge within 6 hours. Full activity may resume within 48 hours of the procedure. No anticoagulation or antiplatelet therapy is recommended post coil closure procedure although most centers recommend daily aspirin for 4 to 6 months after Amplatzer duct occluder or device closure. Procedural complications are uncommon, occurring in less than 5% [31,34]. Hemolysis causing anemia may occur if a residual shunt is present after closure with either coils or device and requires repeat catheterization with placement of additional embolization coils. The major complication associated with coil closure of the PDA is coil embolization to the lungs, however this is a technical issue that occurs at or immediately after implant whose incidence significantly decreases with operator experience. It is related to either under-sizing of the coil or malposition upon placement. In all but a very few patients the coils can be snared from their embolized position in the pulmonary artery and removed from the body without sequelae. Device embolization, thrombus, and ductal aneurysm have been reported in <1%.

Pre-procedure Evaluation/Management: A complete physical examination and surface echocardiogram is necessary prior to catheterization to make the diagnosis. Large PDAs will have a continuous murmur at the left infra-clavicular region, prominent pulses and a widened pulse pressure. Small PDAs may only have a systolic ejection murmur with normal pulses and pulse pressure. Echo will show an abnormal systolic left to right color flow jet into the MPA or proximal left pulmonic directed inferiorly and anteriorly. A complete blood count (CBC) and type and screen is obtained for the procedure. The activated clotting time (ACT) is maintained at >250 seconds during the procedure with IV heparin. Local anesthesia and mild to moderate IV sedation are used to maintain patient comfort. A dose of antibiotics

(Kefsol or Clindamycin) is given IV prior to device implantation to prophylax against procedural related sepsis/endocarditis.

TECHNICAL TIPS

****Misleading Systolic Flow Mimicking PDA:** Be wary of a color flow jet seen on echo directed posteriorly from the anterior wall of the MPA associated with a systolic or continuous murmur. This most often represents a small coronary to pulmonary artery fistula but can easily be mistaken for a PDA.

TECHNIQUE Defining the Anatomy: The procedure should be adjusted based on the size of the PDA and technique used for closure. Small PDAs can be addressed solely through a 5 or 6 French femoral artery sheath with only retrograde catheterization. Larger PDAs require both femoral and venous access. The anatomy of the PDA is evaluated with a proximal descending thoracic aortic angiogram in straight lateral plane using a pigtail catheter to inject 35 cc of contrast at 35 cc/sec (Figure 29-6). In a biplane lab the AP camera should be set at 30 degrees RAO and 15 degrees caudal, which will nicely separate the PDA from the descending aorta (DAO) showing the distal transverse arch. For small PDAs the pigtail catheter is then exchanged for a 5 or 6 French directional catheter, either Bentson or JR4 shape, with a 0.038" lumen. The catheter is advanced to the proximal descending thoracic aorta and directed anteriorly and leftward. Often the catheter itself can be advanced across the PDA into the MPA, particularly if the catheter tip has been shaped by hand with an exaggerated anterior curve. If the catheter itself will not advance through the PDA then the soft end of a straight 0.035" wire can be advanced into the MPA and the catheter advanced over the wire. Pressure and saturation

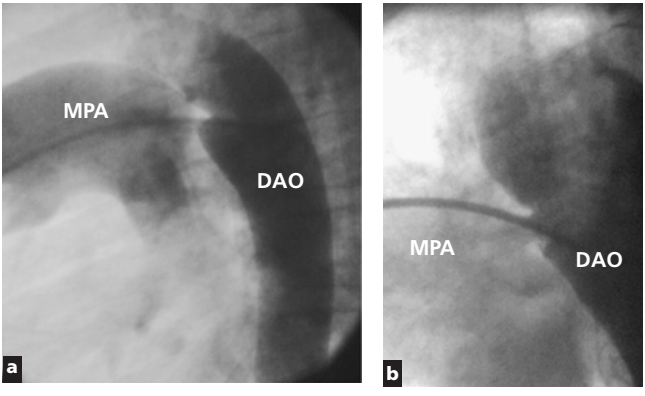

Figure 29-6 Lateral DAO angiogram showing a (a) typical conical shaped PDA and (b) a window type PDA.

measurements should be obtained in the MPA and DAO to confirm catheter location and document left to right ductal shunting.

TECHNICAL TIPS

****Locating the Point of Minimal Diameter of the PDA:** If the point of minimal diameter of the PDA is not well defined angiographically, ii can be located by correlating catheter tip position relationship to bony and tracheal air column landmarks during pressure pullback from MPA to DAO through PDA. The point of acute pressure change from low MPA pressure to systemic DAO pressure will correspond to the minimal PDA diameter. This typically occurs at or just anterior to the anterior edge of the tracheal air column on straight lateral projection.

For larger PDAs a 7 French balloon wedge or multipurpose catheter can be manipulated through the right heart to the branch pulmonary arteries with measurement of pressures and saturations to determine the degree of ductal shunting. The catheter can be manipulated antegrade through the PDA by advancing with clockwise rotation in the distal MPA. If this does not track easily a floppy directional wire such as a 0.035″ Terumo can be advanced across the PDA and the catheter advanced over the wire.

****Crossing the PDA from the Aorta:** If you are having difficulty crossing the PDA from the MPA, cross retrograde from the DAO with a directional catheter. Place a 10 mm snare through the retrograde catheter that is now in the MPA and snare the soft end of a 0.035″ straight wire protruding from the antegrade catheter in the MPA. The retrograde catheter can then be used to pull the antegrade catheter across the PDA for proper positioning.

TECHNIQUE Choosing Device Size: For small PDAs less than 3 mm in diameter Gianturco embolization coils can be used for closure. They are available in a variety of wire diameters (0.018″, 0.025″, 0.035″, 0.038″ or 0.052″), loop diameters (3 through 15 mm) and total wire lengths (3 to 15 cm). For the most part 0.038″ wire diameter coils are used although 0.052″ coils can be used for larger PDAs and 0.035″ for very small ducts. Initial coil size is chosen based on minimal PDA diameter with the loop diameter $\geqslant$2 times the minimal PDA diameter. Coil length should allow for at least four loops of coil (one loop on PA side of PDA and the remainder in the aortic ampulla) so length $\geqslant 4 \times \pi \times$ loop diameter. For example, a 2.5 mm minimum diameter PDA can be closed with a 0.038″, 7 cm long, 5 mm loop diameter coil which will provide a total of 4.4 loops.

For ducts 3 mm or greater, the Amplatzer duct occlude device can be used. This Nitinol wire mesh self-expanding device has a wider aortic flange measuring 2 mm larger than the central ductal plug that ranges in length from 5 to 8 mm (Figure 29-7). Central ductal plug diameters range from 4 mm to 14 mm. The diameter of the ductal portion of the device should be 2 mm larger than the minimal diameter of

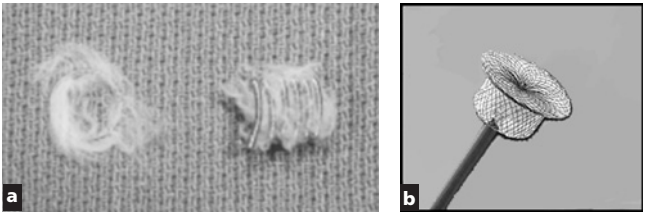

Figure 29-7 (a) Front and side view of .038″ × 7 cm × 5 mm Gianturco coil. (b) Side view of Aplatzer 8–10 mm duct occluder device.

the PDA so this device can close duct up to 14 or 15 mm in diameter. For example, a 5.7 mm minimal diameter ductus can be closed with a 10–8 mm diameter, 8 cm long Amplatzer ductal occluder.

TECHNICAL TIPS

****Use Fewer Coils in PDA Closure:** You can reduce the ratio of minimal PDA diameter to coil loop diameter to 1.7 if you use the thicker stiffer 0.052″ wire diameter embolization coils. In fact larger ducts, up to 7 mm in diameter can be effectively closed with these 0.052″ coils, particularly if simultaneous deployment of two 0.052″ coils is performed antegrade through a long 7 French sheath.

TECHNIQUE Sheath Placement: For retrograde coil closure of the PDA, a short 5 or 6 French sheath in the femoral artery is all that is needed. For antegrade Amplatzer duct occlude PDA closure an appropriate 6 or 7 French long sheath with a curved tip (180 degree transeptal shape) placed across the PDA into the DAO is needed. Once an end hole catheter has been advanced antegrade across the PDA advance it to the proximal abdominal aorta and place a 0.035″ J-tipped exchange wire through the catheter. Remove both the catheter and short sheath and advance a long sheath from the femoral vein over the wire through the right heart into the DAO.

TECHNICAL TIPS

****Inserting the Sheath into the RVOT:** To ease passage of the sheath through the right ventricular out-flow tract (RVOT) and minimize ectopy, rotate the sheath clockwise as it moves into the RVOT to avoid getting caught on the moderator band. If difficult, passage of the sheath can be facilitated by either using a stiffer wire (such as an 0.038″ or Amplatzer super stiff) or by snaring the tip of the wire in the DAO with the retrograde directional catheter and a 10 mm Nitinol snare.

TECHNIQUE Device Positioning: For coil closure of the PDA retrograde, the tip of the directional Bentson or JR4 with a 0.038″ lumen is positioned across the PDA in the main PA. Lateral floursocopy is used to guide the procedure with a roadmap image from the lateral

angiogram available to define ductal anatomy. A straight 0.035″ wire is used to load the embolization coil into the catheter and advance or "push" the coil to the tip. One loop of coil is extruded from the tip of the catheter by advancing the 0.035″ pushing wire and the entire catheter/coil/pushing wire is then brought back slowly together to position this extruded loop of coil against the PA end of the PDA. As the extruded end of the coil makes contact it will change shape by either rotating or opening slightly. The pushing wire is now held in position and the catheter is retracted over the pushing wire. This uncovers the proximal end of the coil in the aortic ampulla while maintaining the distal loop of coil on the PA side of the ductus. The catheter is brought back completely uncovering the proximal end of the coil that will then spring from the tip of the catheter and coil up in the aortic ductal ampulla. Controlled release coils are available allowing the pushing wire to be advanced once a secondary loop starts to form in descending aorta for a more controlled release of the proximal end of the coil near the aortic ampulla.

TECHNICAL TIPS

****Avoiding First Coil Embolism While Deploying the Second Coil:** Watch the PA loop of coil carefully while delivering the proximal portion of the coil. If additional coil loop is advancing forward into the PA as you deliver the proximal portion of the coil, then the catheter and pushing wire must be pulled back more aggressively to avoid embolization of the entire coil into the PA. If the PA loop of coil is getting smaller and pulling into the aorta during delivery, then the pushing wire must be held more stable or advanced to keep the distal loop in the PA and prevent embolization of the coil into the DAO.

Approximately 10 to 15 minutes after coil placement an angiogram should be performed by hand through the directional catheter, positioned at the tip at the inferior margin of the aortic ductal ampulla, pointing anterior and leftward. If a significant residual leak exists through the initial coil then additional coils should be placed. A significant leak is contrast passing through the coil as a jet or contrast filling into the MPA 5 mm or more past the PA end of the existing coil. The second coil should be 2 mm smaller in loop coil diameter size and can have a length providing three to four loops. To cross the PDA with an existing coil in position the directional catheter is positioned at the inferior edge of the aortic ductal ampulla pointing toward the PA. The soft end of a 0.035″ straight wire is advanced gently through the existing coil into the PA. This may take several attempts with slight angulation of the directional catheter on each attempt to find the residual defect.

****Avoiding Entangling the Already Deployed Coil in the Directional Wire:** Be careful to use a non-steerable wire when you cross the initial coil. Directional wires with floppy ends that can be easily rotated can inadvertently cause the tip to spin in the existing coil. This will wrap fibers of the implanted coil around the directional wire entangling the two and cause the implanted coil to dislodge.

Once the straight wire is through into the PA, advance the directional catheter over the wire. Delivery of the second smaller coil is performed similar to delivery of the first coil. Occasionally a third coil may be necessary for complete closure.

For the Amplatzer PDA duct occlusion device, the long sheath should be positioned antegrade in the mid thoracic DAO and kept there until the device is advanced to the tip of the sheath. This prevents the sheath from inadvertently being withdrawn through the duct into the MPA as the device advances. The entire system is then brought back until the tip of the sheath is just off the posterior wall of the DAO at the level of the ductal ampulla. The device is held in position and the sheath retracted to open the distal flange of the device only. The entire system is withdrawn together and the aortic flange is pulled firmly against the aortic ampulla. A pigtail catheter is positioned from the femoral artery in the thoracic DAO for a lateral angiogram to confirm appropriate position of the aortic end of the device. Once position is confirmed the device cable is held in position and the sheath is retracted opening the ductal plug within the PDA.

****Checking the Position of Device Prior to Deployment:** A hand angiogram through the delivery sheath can be performed to assess the PA side of the device. If the PA end protrudes >3 mm, or there is evidence of left pulmonary artery (LPA) obstruction, the device should be recaptured and repositioned.

A repeat angiogram is performed in the DAO to confirm appropriate device position and the cable is then unscrewed for device release keeping slight tension on the cable to maintain position.

****Closing the PDA with Occluder:** A window-shaped ductus may be more effectively closed with an Amplatzer septal occluder or CardioSEAL STARFlex device. The technique is similar to that described above for the Amplatzer PDA duct occluder device.

TECHNIQUE Post Placement Assessment: Repeat hemodynamic measurements are then performed with particular attention to pressure measurements in the LPA, MPA, transverse arch and DAO to assure no obstruction to the proximal LPA or DAO has occurred. A final angiogram through the pigtail catheter in the proximal thoracic DAO is performed in the lateral projection (35 cc at 35 cc/sec) to assess final positioning and closure. Some leak through the Amplatzer duct occluder device is expected as fibrin deposition on the fabric for complete closure occurs over hours (Figure 29-8).

Indications: PDA closure is indicated in all patients with LA or left ventricular (LV) enlargement due to left to right shunting or pulmonary artery pressure elevation. Small PDAs that do not result in hemodynamic effects are still at risk for the development of endocarditis. Controlled trails comparing antibiotic prophylaxis with device closure for the prevention of endocarditis have not and will not be performed due to the limited number of patients and low incidence of endocarditis. There have been no late

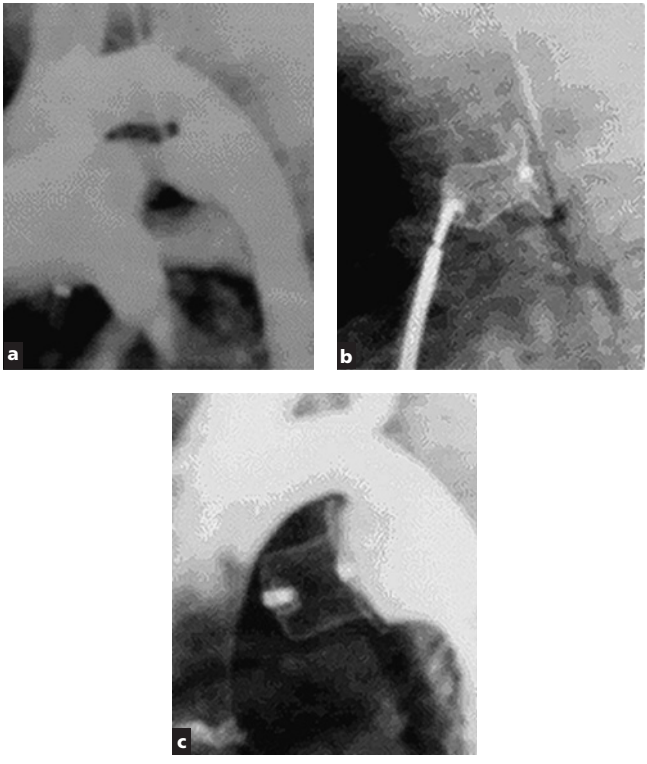

Figure 29-8 (a,b,c) Lateral angiograms of PDA before, during and after Amplatzer duct occlude device closure.

reports of endocarditis following interventional closure of the ductus although procedural infections have occurred rarely. Current clinical recommendations are for device or coil closure in small hemodynamically insignificant PDAs if they are audible on physical examination.

Contraindications: Patients with systemic pulmonary hypertension and right to left ductal shunting should not have their PDA closed. If pulmonary hypertension is noted during catheterization then an accurate assessment of the degree of hypertension and the reactivity of the pulmonary bed must be made during temporary occlusion of the ductus. A second venous sheath should be placed so that simultaneous PA pressure measurement and pulmonary vascular resistance calculations can be made while balloon occlusion of the PDA is performed. If there is baseline left to right shunt and a decrease in PA pressures with balloon occlusion then ductal closure is indicated.

COARCTATION OF THE AORTA

Coarctation is most often a discrete narrowing of the proximal descending thoracic aorta just distal to the origin of the left subclavian artery at the site of the ductus ligamentum. It makes up 7% of all patients with congenital heart disease and results in upper extremity hypertension, left ventricular hypertrophy and eventually ventricular failure if left untreated. It should be considered during the initial evaluation of systemic hypertension and can easily be diagnosed on physical examination by decreased femoral pulses with a delay compared to radial pulses and blood pressure differential between the arms and leg. Often a 2/6 systolic ejection murmur can be heard at the left upper sternal border and over the left back. The narrowing is due to thick intimal and medial ridges that protrude posteriorly and laterally into the aortic lumen [37]. Intimal proliferation and elastic lamina disruption occur distal to the ridges due to the high velocity jet impact on the distal aortic wall. Cystic medial necrosis with disarray and loss of medial elastic tissue occurs commonly in the adjacent aorta and may extend to the ascending aorta as well. It is this abnormality that may lead to late aneurysm formation. The body's compensatory response to coarctation is the development of vessels that bypass the obstruction, collateral vessels from the inominate, carotid, and subclavian arteries that connect to the thoracic aorta below the level of the coarctation, often connecting through the internal mammary and intercostal arteries. Enlargement of the intercostals arteries due to this collateral flow is the mechanism for rib notching seen on chest x-ray and subscapular pulsation on physical examination in adult patients with severe native coarctation.

Balloon dilation for treatment of coarctation was first performed in the early 1980's in children with good success in both native and post operative coarctation [38]. Its efficacy in adults was found similar to that in children [39–41], however, there remained a small but significant failure rate with residual gradient greater than 20 mmHg in approximately 15% of patients treated. Stent implantation for repair of coarctation was performed sporadically in the early 1990's in children being first reported in adults in 1995 with very promising results [42]. Since that time stent repair has become the treatment of choice for coarctation in many centers due to the improved success rate and low restensosis rate, although controlled trials are not available [43]. Procedural success has been reported in $>$95% of patients with residual obstruction of less than 20 mmHg. Recurrent stenosis has been extremely rare occurring in less than 5%, primarily younger patients and generally mild. Complications have been reported in up to 20% and include aneurysm, perforation, stroke and death [43–47]. In addition, femoral artery complications including arterial venous fistula and pseudo-aneurysm have been reported associated with the larger arterial sheaths required for the procedure.

The equipment available for angioplasty and stent repair of coarctation has improved significantly over the last 20 years. Balloons that

are specifically designed for large sent implantation and stents that have adequate radial strength at sizes appropriate for an adult thoracic aorta are only recently available. Currently there are three large stents designs, two stainless steel and one of platinum, that can reach diameters of 18 to 25 mm with adequate coverage and radial strength appropriate for treatment of coarctation.

Pre-procedure Evaluation/Management: A complete physical examination including upper and lower extremity blood pressure measurement is essential. Echocardiography may be helpful in confirming the diagnosis if the physical examination in unclear, however, echo often poorly defines the anatomical detail of the obstruction and frequently overestimates the degree of obstruction. Anatomical definition of the coarctation prior to catheterization is critical to determine the best approach for treatment. Patients with hypoplastic transverse arch or a "kinked" high third arch may respond poorly to stent repair and may best be treated surgically. MRI with magnetic resonance angiography is currently the best technique for defining the arch anatomy and can give functional data including estimation of degree of obstruction based on blood velocity at the site and percent of collateral flow, an excellent indication of the physiologic significance of the forestation [48]. In addition, the MRI gives accurate anatomical detail of the size, location, and length of coarctation so that appropriate equipment including dilation balloon and stent sizes planned in advance. A CBC and type and cross is obtained for the procedure. Blood is kept available in the cath lab during balloon dilation and stent implantation. The ACT is maintained at $>$250 seconds during the procedure with IV heparin. Local anesthesia and mild to moderate IV sedation are used to maintain patient comfort. Additional IV narcotic, Fentanyl, is given immediately prior to balloon dilation or stent implantation as aortic stretch causes a moderate amount of pain acutely. A dose of antibiotics (Kefsol or Clindamycin) is given IV prior to device implantation to prophylax against procedural related sepsis/endocarditis. Patients who are taking anti-hypertension medications continue those the morning of the procedure. Short acting IV beta-blocker is given immediately after balloon dilation or stent implantation if significant acute hypertension develops.

CAVEAT

A note of caution, this procedure can have relatively high rates of significant complications that can be reduced by careful patient selection and operator experience, however, in hospital cardiothoracic surgical availability to address emergencies is mandatory.

TECHNIQUE Defining the Anatomy: Right and left heart catheterization is performed in routine fashion. Because of the large sheath size required in the artery for this procedure suture closure of the femoral artery is recommended so be sure the sheath insertion site is appropriately superior. Cardiac index should be measured either with saturation or thermodilution techniques. This is essential both prior to and after balloon dilation or stenting to properly interpret the degree of stenosis measured across the coarctation. Pressure pullback across the area of coarctation is recorded.

TECHNICAL TIPS

****Measuring the Gradient Across the Coarctation:** Remember the pressure gradient across the coarctation depends primarily on the cross sectional area of the lesion, its length, and the amount of flow crossing the lesion. Severe coarctations may have very little pressure gradient from ascending aorta (AAO) to DAO if there are substantial collateral vessels limiting the flow through the lesion. Despite collateral vessels this obstruction remains a significant increased work load for the LV and stimulus for upper body hypertension.

An angiogram is performed in the distal transverse arch using a marker pigtail catheter to allow for accurate measurements. The AP camera should be angled 15 degrees LAO and 10 degrees caudal combined with a straight lateral projection with an injection of 35 cc at 35 cc/sec. Careful measurements are then made of the distal transverse arch diameter, coarctation diamter, coarctation length, distal normal vessel diameter, distance from the left subclavian artery origin to the coarctation, and diameter of the left subclavian artery (Figure 29-9).

TECHNIQUE Choosing Balloon and Stent Size: Balloon diameter should never exceed the smallest diameter of normal aorta surrounding the coarctation that the balloon may contact. In other words,

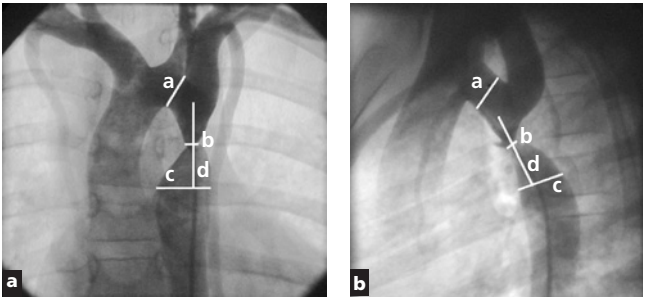

Figure 29-9 (a) AP angiogram of native coarctation with key measurements, **a** transverse arch diameter, **b** coarctation diameter, **c** DAO diameter, **d** coarctation length. (b) Lateral angiogram showing similar measurements.

the goal is to enlarge the coarctation to the size of the smallest contiguous normal aorta, not to stretch it larger than the normal diameter. Preferably the balloon should be at least 2.5 times larger than the coarctation diameter but not more than 3.5 times larger. Remember that if the wire and balloon tip are in the innominate or left subclavian artery for stabilization then the balloon diameter must not exceed the normal vessels diameter of the proximal innominate or subclavian. These guidelines will minimize the risk of aneurysm or rupture.

TECHNICAL TIPS

****Sequential Dilation of the Coarctation:** If the coarctation is severe with a diameter $<1/4$ of the normal aortic diameter (for a normal sized adult with a 20 mm distal transverse arch that would be a coarctation diameter of 5 mm or less), then complete repair should be performed in two or three stages at 3-month intervals to allow adequate healing of the aorta between procedures. The first procedure should be balloon dilation with stent implant and enlargement to 2.5 times the coarctation diameter (in the example given a stent would be implanted and dilated to 12 mm). Three months later the patient should have dilation of the implanted stent to the size of the surrounding normal aorta (in the example the stent would then be dilated to 20 mm).

Care must be taken to choose a balloon that is long enough to remain stable in the lesion but not extend around the arch or substantially into the head and neck vessels. Generally a 3 or 4 cm balloon length is optimal. The balloon should be of scratch resistant material, preferably designed for use with stents. Remember that the stent will need to be mounted during the procedure so care must be taken not to damage the balloon during the mounting process. Some operators have advocated the use of a double balloon delivery catheter. This has an inner balloon half the diameter of the final outer balloon. The concept is the inner balloon allows a more uniform enlargement of the stent with minimal stent tip flaring and the ability to adjust stent position prior to final implant with the larger balloon in a more controlled manner. We have not found the balloon in a balloon technique to offer any significant advantage over careful delivery with a single lumen balloon, however it adds some complexity to coordinate inflation of both balloons sequentially.

The stent used should be able to reach a diameter appropriate for the normal aorta and the patients size, which for most adults will range between 18 to 22 mm. The stent length should be kept as short as possible maintaining adequate length after foreshortening with dilation to completely cover the length of the lesion. Unnecessary stent length may be a disadvantage due to increased length of noncompliant aorta after implant that may influence blood pressure, particularly in response to exercise. Although a theoretical risk, animal models have not shown this to be a practical concern [49].

****Advantage of MRI Compatible Stents:** Although not readily available at present, platinum or Nitinol stents may be preferred over stainless steel stents due to their MRI compatibility allowing follow up MRI assessment of these patients coarctation site that is not possible after stainless steel stent placement.

TECHNIQUE Sheath and Wire Placement: Wire position is important to optimize balloon and stent positioning as well as minimize risk of complications. A relatively stiff exchange length wire should be used, we prefer and Amplatz wire with a short soft tip. The first choice for wire position is the left subclavian artery if there is adequate distance (1.5 cm) from its origin to the site of coarctation. This position is easy to obtain and allows for a straight balloon/stent course while minimizing wire/sheath/balloon exposure to the carotid arteries, and thereby minimizing the risk of a neurologic complication. If the distance between the site of coarctation and the origin of the left subclavian is too short or the diameter of the proximal left subclavian is too small to accept the tip of the dilating/implanting balloon, then the wire should be looped in the AAO or LV apex.

TECHNICAL TIPS

****Optimal Wire Position:** If the wire is positioned in the ascending aorta care must be taken to prevent the inadvertent cannulation of the coronaries, or prolapse through the aortic valve, resulting in significant ectopy. If wire placement in the AO or LV is necessary, choose the shortest balloon possible to minimize the straightening of the arotic arch that will occur during dilation or stent implantation.

The sheath should be straight and long enough to reach the coarctation from the femoral artery. For stent implantation increase the French size of the sheath one or two above that recommended for the balloon alone (This will generally be 10 to 12 French sheath size). To minimize the risk of a neurologic complication we prefer to keep the sheath at or below the area of coarctation, particularly if the wire is positioned in the right inominate or left ventricle. The sheath is continuously flushed with heparinized saline to minimize risk of clot formation.

TECHNIQUE Balloon and Stent Positioning: The stent is flared open on the table using an appropriate dilator to allow it to easily slip onto the delivery balloon (which is under negative pressure) without contacting the balloon material. The stent is hand crimped onto the balloon and the negative balloon pressure released. The long sheath is positioned in the abdominal aorta and the stent balloon combination advanced to the tip of the sheath, allowing only the balloon tip to protrude. The sheath and balloon/stent system is advanced across the lesion and the sheath pulled back just below the coarctation. A hand angiogram through the sheath is then performed in the lateral projection to define the coarctation, and origin of the subclavian artery relative to the position of the stent. The stent should be

centered on the coarctation with care taken so the proximal edge of the stent is distal to the origin of the subclavian artery.

TECHNICAL TIPS

****No Problem with Subclavian Jailing:** The subclavian artery can be crossed and jailed if absolutely necessary to effectively stent the coarctation. Because the subclavian originates at approximately 90 degrees to the aortic arch and the interspaces of these large stents are quite sizable, no obstruction to flow will occur. There have been no recent reports of either stenosis or distal thrombus following subclavian "jailing". However, daily aspirin is recommended for at least 12 months after implant if the subclavian is "jailed".

The sheath is retracted over the balloon catheter just to the proximal edge of the balloon. This way the sheath can help maintain balloon position during inflation to help prevent distal movement due to the force of the ejecting blood. This fact should be considered when positioning the balloon and stent prior to delivery by having the stent centered just proximal to the center of the coarctation. Inflation of the stent should initially proceed slowly until both ends of the stent are partially flared. The balloons position can still be adjusted at this point if necessary. Full inflation is then performed taking care not to exceed the burst pressure of the balloon.

****Post-Dilation with High Pressure Balloon:** It is much better to post dilate a stent with a residual waist by placing a high pressure balloon after initial implant, than to attempt resolution of a residual waist by excessive pressure with the initial implanting balloon. Removal of a ruptured balloon from a freshly implanted stent can be problematic and the effectiveness of a post implant high pressure dilation is usually significantly greater than the initial implant dilation.

TECHNIQUE Post Placement Assessment: Following balloon dilation or stent implantation repeat hemodynamic assessment should be performed including measurement of cardiac index by saturation or thermodilution techniques. Pressure pullback across the dilated and or stented coarctation should be performed with a "Y" adapter over a wire to maintain distal wire position and not disturb the site.

TECHNICAL TIPS

****Accurate Pressure Gradient Measurement:** To get an accurate pressure measurement and optimal picture the wire used with the pigtail catheter should to be downsized to a 0.025" Rosen or Amplatz wire once the pigtail catheter has been advanced well passed the site of coarctation.

The pigtail catheter should then be advanced just proximal to the coarctation for an angiogram. Cameras should be kept with the AP angled 15 degrees LAO and 10 degrees caudal and a straight lateral projection with an injection of 25 cc at 25 cc/sec (rate and volume

need to be reduced from initial picture to safely use the power injector with a wire and the "Y" adaptor). Additional views with different camera angles may be necessary if an aneurysm or extravasation of contrast is suspected on the initial post implant angiogram.

Indications: Any coarctation with a gradient of >10 mmHg and significant upper body hypertension or left ventricular hypertrophy without additional cause should be treated. For mild coarctation it is imperative to use stent implantation to assure complete resolution of the mild obstruction. Mild coarctations with <20 mmHg gradient without hypertension or LV hypertrophy should be considered for stent repair if collaterals are present or the patient has an abnormal blood pressure response to exercise. Patients with coarctation gradients of >20 mmHg at rest should be repaired.

Contraindications: Patients with coarctation gradients <20 mmHg with no evidence of collateral flow, hypertension, LV hypertrophy or abnormal blood pressure response to exercise do not need treatment. Patients with significant hypoplasia and obstruction of the transverse aortic arch in the area of the origin of the carotids should be excluded. Stent repair with jailing of the carotids may be appropriate in the rare patient at extremely high surgical risk, however for the majority of patients with this lesion surgical repair should be performed. Any patient with an existing aneurysm should also be cautiously considered. The use of covered thoracic stents may have a role in this setting although there is limited data at present.

PULMONARY VALVE STENOSIS

Pulmonary valve stenosis was first described by John Baptist Morgagni in 1761 [50] and although initially thought to be rare makes up approximately 8 to 10% of all congenital heart disease. The pathology of congenital pulmonary valve stenosis is variable. In the most common form the valve is dome shaped with two to four raphes present but no separation into valve leaflets [51]. Occasionally the valve may be diffusely thick with commissural fussion of one, two or all three leaflets. In up to 15% of cases the valve is trileaflet but with dysplastic diffusely thickened cusps of myxomatous tissue without commissural fusion. This pathology is associated with valve annulus hypoplasia and commonly seen in patients with Noonans syndrome [52]. Valve stenosis leads to secondary changes in the right ventricle consisting of right ventricular hypertrophy, and if severe, tricuspid regurgitation and right heart failure. The MPA becomes dilated due to the jet from the stenotic valve and on rare occasion this post stenotic dilatation can extend into the proximal LPA. Patients are usually asymptomatic until the stenosis is severe with presenting symptoms most typically exercise intolerance. The diagnosis is made on physical examination with an audible systolic ejection click and murmur heard loudest over the left upper sternal border. Confirmation and determination of severity is

made by echocardiogram by estimating the gradient across the valve, the RV systolic pressure and the RV wall thickness.

Balloon dilation of valvar pulmonary stenosis was first described by Kan *et al.* in 1982, the first congenital lesion to be treated with balloon dilation [53]. The technique has remained unchanged since that time although the balloon technology has progressed substantially to allow for faster, more effective and lower risk procedures. Efficacy of balloon valvuloplasty in children and adults with congenital pulmonary valve stenosis is excellent with procedural success in more than 95% and a residual gradient of <35 mmHg in over 75% of patients [54–56]. Complications occur in $<5\%$ and primarily relate to local femoral vein injury or transient ventricular ectopy although valve annulus rupture and death have been reported in children. Recurrence of stenosis with need for repeat intervention is necessary in $<15\%$ of older patients with typical valvar stenosis. Mild insufficiency is commonly seen in up to 65% of patients after dilation but moderate or severe insufficiency is rare, seen in less than 7% [55].

Pre-procedure Evaluation/Management: A complete physical examination is important to rule out associated RV outflow obstruction. Surface echocardiogram should be complete to evaluate the pulmonary valve annulus, degree of stenosis, significance of RV hypertrophy by measuring wall thickness, RV pressure measurement and any evidence of additional RVOT obstruction including infundibular, supravalvar or branch stenosis. A 12 lead EKG is important at baseline to assess the degree of RV hypertrophy and monitor progress following balloon dilation. A CBC and type and screen is obtained for the procedure. The ACT is maintained at >250 seconds during the procedure with IV heparin. Local anesthesia and mild to moderate IV sedation are used to maintain patient comfort. Additional IV narcotic, fentanyl, is given immediately prior to balloon dilation as pulmonary artery stretch causes a moderate amount of pain acutely.

TECHNIQUE Defining the Anatomy: Right heart catheterization is performed in the usual fashion using a 7 French balloon tip wedge catheter through a femoral vein. Measurement of cardiac index using either saturation data or thermodilution technique is important to assess the significance of the valvar obstruction. A pressure pullback measurement should be obtained from MPA to RV to determine the systolic transvalvar gradient. An apex or Rosen 0.035″ exchange wire is then positioned in the RV apex and the wedge catheter exchanged for a marker pigtail catheter for angiography of 35 cc at 35 cc/sec. The AP camera should be set with 15 degrees of cranial angulation and the lateral plane in a straight lateral projection. Measurement of the pulmonary valve annulus can then be made in both planes (Figure 29-10) to guide balloon size determination.

TECHNIQUE Choosing Balloon Size: Balloon dilation can either be performed with a single or double balloon technique. There is no evidence that either technique offers a significant advantage regarding

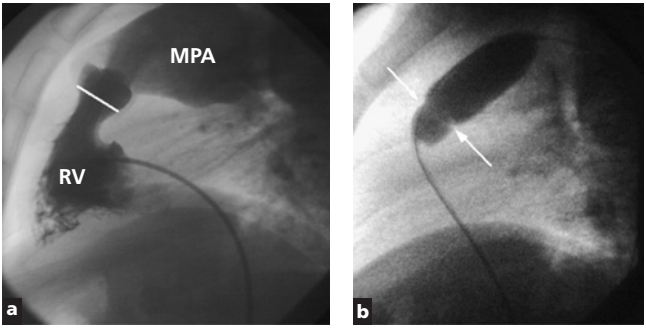

Figure 29-10 AP and Lat angiogram of valvar PS with measurements of valve annulus, Lateral angiogram of balloon inflated with waist present (arrows). Notice this balloon is poorly positioned, the balloon should be centered on the valve for optimal dilation.

success, development of insufficiency or recurrence of stenosis. A single balloon is technically less complicated but can be problematic for large pulmonary valve annuli. In experimental animal studies it has been shown that dilation with a balloon >150% the diameter of a normal pulmonary valve annulus can result in rupture [57]. In addition, there is reasonable clinical evidence in children and young adults with congenital valvar pulmonary stenosis that significant improvement in stenosis relief is achieved with balloon diameters greater than 100% of the annulus diameter [58]. Based on these observations the target balloon diameter should be 120–140% of the pulmonary valve annulus diameter. If a double balloon technique is used then two similarly sized balloons are used if possible to facilitate positioning [59]. The total circumference of the two balloons should equal 120–140% of the circumference of the valve annulus. The formula for calculating the double balloon diameter is:

$$\text{Double balloon diameter} = 1.2\,\pi\,(\text{PV annulus diameter})/(2 + \pi)$$

For example, a 22 mm pulmonary valve could either be dilated with a single 26 mm diameter balloon or two 16 mm diameter balloons. The balloon should be long enough to allow stable position across the RV outflow tract but not so long it protrudes into either the tricuspid valve proximally or a distal branch pulmonary artery. In animal experiment the majority damage due to balloon inflation occurs in the right ventricular outflow tract due to the proximal end of the balloon straightening anteriorly. Generally, balloons 4 cm long are adequate although occasionally a 6 cm balloon is required to maintain position across the valve.

TECHNIQUE Sheath and Wire Placement: A long sheath is not required for pulmonary balloon valvuloplasty. An exchange length relatively stiff 0.035″ wire such as a Rosen or Amplatz is positioned in the distal lower pulmonary artery using a 7 wedge catheter to obtain

initial position. Either the left or right PA can be used although the left provides a straighter coarse in most patients.

TECHNICAL TIPS

****How to Track the Balloon Across the Valve:** Take care to get very stable distal wire position in one of the lower segmental pulmonary arteries. This will facilitate tracking the balloon catheter across the valve and improve stability during inflation. If the balloon wedge catheter does not pass easily to a lower lobe vessel, use a directional wire to advance it into a desired distal branch. The tip of the wedge catheter can then be held in place in the distal vessel by gently inflating the balloon while advancing the exchange wire out the tip.

TECHNIQUE Balloon Position and Dilation: A roadmap from the lateral angiogram should be referenced to give landmarks for the pulmonary valve annulus position. The balloon is centered on the valve annulus and inflated to 6 atm. Balloon movement during inflation is common so the catheter must be maintained in position and the inflation recorded so that it may be reviewed. If the initial inflation did not result in complete resolution of the balloon waist centered on the valve then the balloon should be repositioned and the inflation repeated.

Post-dilation Assessment: Following dilation the balloon catheter can be exchanged for the wedge catheter and right heart pullback hemodynamic pressure measurements repeated including cardiac index measurement. The pigtail catheter is then placed in the RV for repeat RV angiogram in the AP and lateral projections to assess for dynamic RV outflow tract stenosis, aneurysm formation, or new onset tricuspid regurgitation. An increase in dynamic RV outflow tract obstruction after relief of significant valvar pulmonary stenosis is common and often causes significant residual gradient which can be confirmed on careful pressure pullback tracing and on the lateral angiogram. This residual dynamic obstruction will resolve gradually with regression of the RV hypertrophy following valve obstruction relief.

Indications: All patients with valvar pulmonary stenosis gradients >40 mmHg, or milder gradients with evidence of right ventricular hypertrophy, should have treatment with balloon dilation. There is no evidence that patients with mild pulmonary valve stenosis without evidence of right ventricular hypertrophy will benefit from balloon dilation. Dysplastic valve morphology and annular hypoplasia are independent predictors of poor response to balloon dilation, however age and associated anomalies such as Noonan's syndrome are not [55].

Contraindications: Patients with supravalvar narrowing of the main pulmonary artery can often appear on echo to have valvar pulmonary stenosis. This diagnosis can be difficult to differentiate angiographically due to the close proximity of the MPA narrowing to the pulmonary valve and a common association of thickened dysplastic

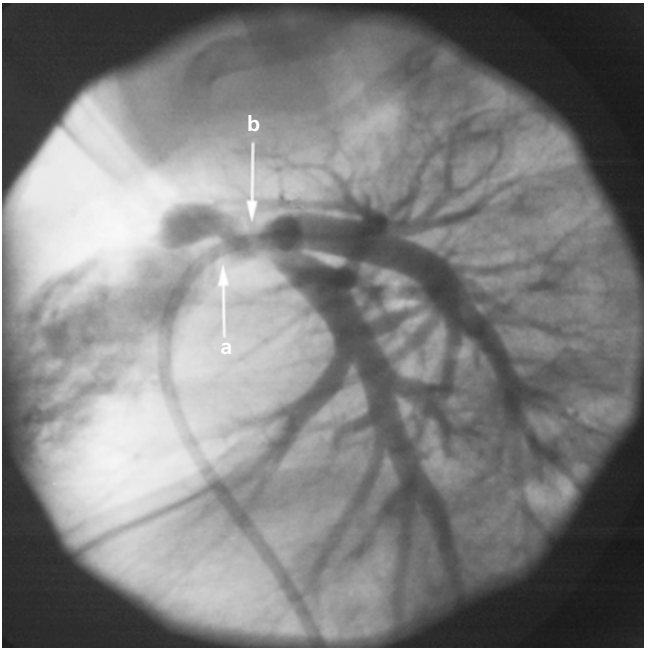

Figure 29-11 Lateral angiogram of supravalvar PS, Note the proximity of the tips of the valve leaflets (a) to the MPA narrowing (b).

valve leaflets (Figure 29-11). These lesions do not respond to balloon dilation and should be referred for surgical repair. Similarly dynamic subvalvar pulmonary stenois or double chmber RV do not respond to balloon dilation and are currently best addressed surgically.

PULMONARY ARTERY STENOSIS

Branch pulmonary artery stenosis is a rare congenital lesion in isolation but is often associated with complex congenital heart lesions following surgical repair, especially tetralogy of Fallot. Other associated lesions include truncus arteriosus or pulmonary atresia with ventricular septal defect after RV to PA conduit placement, transposition of the great arteries following arterial switch repair, and pulmonary artery sling after re-implantation. Branch pulmonary artery stenosis decreases perfusion to the affected lung and if severe, causes hypertension in the non-affected lung and right ventricle. Distal pulmonary artery stenosis promotes pulmonary insufficiency that compounds the decrease in cardiac output and increased workload on the right ventricle seen in these patients. Patients are often asymptomatic but may present with exercise intolerance.

Balloon dilation of branch pulmonary artery stenosis was initially described in 1983 by Lock *et al.* [60]. However only 50% of lesions responded primarily with a significant re-stenosis rate [61]. With the application of large peripheral stents to pulmonary artery branch stenosis in the early 1990's [62], stent placement has rapidly become the treatment of choice in adults because of improved success and low restenosis rates. Shaffer *et al.* reported results in over 130 children and adults with post operative branch pulmonary artery stenosis showing that in over 65% stent implantation increased lesion diameter by over 100% with a median gradient reduction from 46 to 10 mmHg, and RV to systemic pressure ratio reduction from 60 to 40% [63]. Long term results have been excellent with restenosis rates <5% [64]. Complications are rare occurring in less than 4% of cases overall, and include hemoptysis, aneurysm, perforation, refractory ventilation perfusion mismatch, and death [63]. Technical issues such as device malposition or embolization have been reported in <2% and are quite rare with recent improvements in balloon and stent technology.

Pre-procedure Evaluation/Management: A complete physical examination is important to rule out associated RV outflow obstruction. Surface echocardiogram should be completed to evaluate the significance of RV hypertrophy by measuring wall thickness, RV pressure measurement estimate and any evidence of additional RVOT obstruction including infundibular, supravalvar or valvar stenosis. A 12 lead EKG is important at baseline to assess the degree of RV hypertrophy and monitor progress following stent repair. A nuclear pulmonary perfusion scan is critical to determine the functional significance of branch pulmonary artery stenosis and should be performed prior to catheterization to provide context for pressure measurement interpretation during the catheterization. A CBC and type and cross are obtained for the procedure. The ACT is maintained at >250 seconds during the procedure with IV heparin. Local anesthesia and mild to moderate IV sedation are used to maintain patient comfort. A dose of antibiotics (Kefsol or Clindamycin) is given IV prior to stent implant to prophylax against procedural related sepsis/endocarditis. Additional IV narcotic, fentanyl, is given immediately prior to balloon dilation with stent implantation as pulmonary artery stretch causes a moderate amount of pain acutely.

TECHNIQUE Defining the Anatomy: Right heart catheterization is performed in usual fashion using a wedge end hole or multipurpose catheter, including measurement of cardiac index and pulmonary flow if residual left to right shunts are present. Both branch pulmonary arteries should be entered for pressure measurements including the distal lower lobe segments. A stiff 0.035" exchange wire should be positioned in the distal pulmonary artery and the wedge catheter replaced with a marker pigtail catheter for a main pulmonary artery angiogram (30 cc of contrast at 35 cc/second). The AP camera should be angled approximately 30 degrees RAO with the lateral camera orthogonal at 60 degrees LAO and 10 degrees caudal. This will

profile the right pulmonary artery well in the AP projection and the left pulmonary artery in the lateral projection. Measure the affected artery including lesion diameter, vessel diameter proximal and distal to the lesion, and lower lobe segment origin diameter from the angiogram.

TECHNIQUE Choosing Balloon and Stent Size: The goal of stent repair is to enlarge the stenosis to equal the diameter of the surrounding normal pulmonary vessel using the shortest stent possible that will completely cover the lesion. Distension of the lesion greater than the diameter of the surrounding normal vessel is not helpful. Intimal hyperplasia will result in the over distended stent reducing intraluminal diameter to that of the surrounding vessel or even smaller. In addition, vessel over-distention may cause pain up to several weeks after implant. Excessively long stents are problematic due to difficulties advancing them through the tortuous RVOT during implantation, and protrusion after implant into a segmental branch or main pulmonary artery. There are currently three stent types available and effective for treatment of branch pulmonary artery stenosis. The balloon used for stent delivery should be of scratch resistant material and equal to or just slightly longer than the length of the stent.

TECHNIQUE Sheath and Wire Placement: An appropriate sized long sheath, two French sizes above that recommended for the balloon alone, should be positioned in the right atrium over a stiff 0.035" exchange wire that has been positioned in the lower lobe pulmonary artery of the affected branch.

TECHNICAL TIPS

****Stabilizing Wire Position in the Pulmonary Artery Branch:** Take care to get very stable distal wire position in one of a lower segmental pulmonary artery. This will facilitate tracking the stent through the tortuous RVOT into position. If a catheter does not pass easily to a lower lobe vessel for initial wire placement, use a directional wire to advance it into a desired distal branch for the stiff wire exchange

TECHNIQUE Stent Position and Implantation: Because of the tortuous nature of the RVOT and MPA it is best to track the stent together with the long sheath through the RVOT and across the lesion. If the sheath is placed across the lesion when initially inserted it will often kink upon removal of the dilator or when advancing the stent. To avoid this problem leave the long sheath in the RA and advance the balloon/stent combination to the tip of the sheath until the distal edge of the stent is just covered by the end of the sheath while the tip of the balloon catheter protrudes. The sheath together with the balloon/stent is advanced over the wire as a unit through the RVOT and across the lesion in the proximal branch pulmonary artery.

TECHNICAL TIPS

****Using a Stiffer Wire to Track a Stent:** If tracking the sheath stent combination remains difficult resulting in loss of wire position, exchange the 0.035″ stiff wire for a super-stiff 0.038″ wire. This should be shaped by hand with a gentle curve in the distal 15 cm to conform to the RVOT and branch PA to allow positioning without excessive distortion of the heart.

The stent is centered on the lesion and the sheath retracted proximal to the proximal tip of the balloon. A hand angiogram should be performed through the side arm of the long sheath to define the stents position in relation to the lesion, MPA, and upper lobe segmental artery (Figure 29-12). Once optimal position is confirmed the stent is delivered using a pressure inflation device to achieve delivery pressures of up to 16 atm, taking care not to rupture the balloon. Complete expansion of the stent with resolution of the waist is desired on the initial inflation.

TECHNIQUE Post Dilation Assessment: Following stent implantation it is critical to maintain distal wire position both to facilitate assessment and address any complications if they develop.

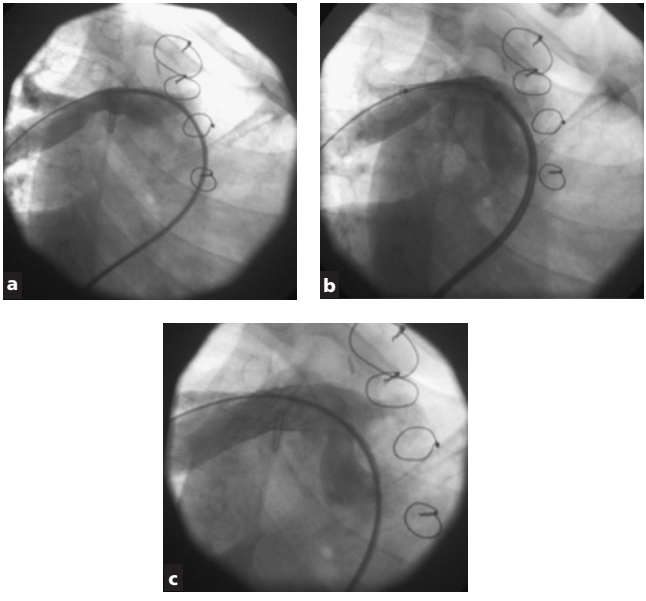

Figure 29-12 RAO angiogram showing proximal RPA stenosis in a patient following Tetrology of Fallot repair. (b) RAO angiogram through delivery sheath confirming stent positioning prior to implantation. (c) Resolution of stenosis immediately after stent implantation.

A catheter with both side and end holes such as a pigtail or Gensini catheter is advanced over the wire and positioned in the distal PA.

TECHNICAL TIPS

****Reshaping the Pigtail Catheter:** If a pigtail catheter is used cut the tip so only a 90 degree curve remains. This will facilitate catheter manipulation over the wire and through the implanted stent.

Using a "Y" connector measure a pressure pullback across the stent into the main pulmonary artery and measure saturation in the main pulmonary artery to calculate pulmonary flow. An angiogram is performed through the "Y" adaptor with the side holes of the catheter positioned in the lumen of the stent. Camera angles should be the same as the initial angiogram with 20 cc of contrast injected at 20 cc/second with a maximal injection pressure of 600 psi. If the stent is incompletely expanded then repeat dilation with a high pressure balloon should be performed. If there is residual stenosis beyond the edge of the stent then placement of an additional stent will be necessary.

Indications: The systolic gradient across branch pulmonary artery stenosis is, in isolation, a poor determinant for the need for treatment and can only be interpreted by also considering quantitative pulmonary flow data. Normal distribution of pulmonary flow is 55% to the right lung and 45% to the left lung. Patients with a reduction of $\geqslant$15% of flow or an absolute flow of $<$1 L/min/M^2 in the affected lung should be considered for stent repair. Patients with any degree of contralateral pulmonary artery hypertension, RV hypertension, or RV hypertrophy should be aggressively treated to prevent progression, as should patients with significant pulmonary insufficiency associated with the branch pulmonary artery stenosis.

Contraindications: Adult patients following repair of complex congenital heart disease such as tetralogy of Fallot or truncus arteriosus are complex, often with multiple anatomic, hemodynamic, and arrhythmia issues in addition to their branch pulmonary artery stenosis. It is critical these patients are evaluated completely and a comprehensive plan made coordinated by a cardiologist familiar with congenital heart disease, and involving an electrophysiologist, cardiothoracic surgeon and interventionalist. If surgical revision of the underlying repair is required, a surgical approach to the branch pulmonary artery steniosis may be preferable.

PULMONARY INSUFFICIENCY – PERCUTANEOUS VALVE IMPLANT

Pulmonary insufficiency is a common problem in adults after repair of congenital heart disease such as tetralogy of Fallot, pulmonary atresia and truncus arteriosus that requires either pulmonary trans-annular patch or RV to PA homograft placement. Although for years it was

thought that insufficiency had little effect on RV function, it has now been shown that significant damage occurs if left unattended that can be reversed by restoring valve competency [65]. Insufficiency leads to RV dilation causing decreased RV contractility. Perhaps more important is the cross talk with the LV through ventricular septal position change and compression causing decreased end diastolic volume and LV compliance. This results in a decrease in cardiac index as well as cardiac reserve [66]. Because surgical pulmonary valve placement is invasive, valves have limited longevity requiring repeat operation and poses risk for the RV long term, in 2006 Bonhoeffer [67] developed a percutaneous stent valve alternative. Preliminary clinical results are encouraging [68] although applicability is limited, but expanding, due to size constraints on commercially available stent valves.

Pre-procedure Evaluation/Management: A complete physical examination and surface echocardiogram is important to assess associated tricuspid valve competency, RV outflow obstruction, RV size and function, as well as the degree of RV hypertrophy by measuring wall thickness and RV pressure. A 12 lead EKG is important at baseline to assess the degree of RV hypertrophy and conduction delay, common in this post-op population. Cardiac MRI with MRA, CT angiogram, is crucial to quantify pulmonary regurgitant fraction, RV and LV size and function, distal pulmonary branch stenoses, and anatomy of the RVOT and MPA to determine suitability for percutaneous valve implant. Although not mandatory, cardiopulmonary exercise testing to determine baseline exercise capacity and arrhythmia risk is recommended. A CBC and type and cross are obtained for the procedure. The ACT is maintained at >250 seconds during the procedure with IV heparin. Either general anesthesia or local anesthesia with mild to moderate IV sedation can be used to maintain patient comfort. A dose of antibiotics (Kefsol or Clindamycin) is given IV prior to stent valve implant to prophylax against procedural related sepsis/endocarditis. Additional IV narcotic, fentanyl, is given immediately prior to stent implantation as RVOT–MPA stretch causes a moderate amount of pain acutely.

TECHNIQUE Defining the Anatomy: This procedure is currently limited to patients whose RV outflow tract and MPA is less than 22 mm in diameter, and if stenosis is present can be adequately enlarged with balloon dilation. Right heart catheterization is performed in the usual fashion using a wedge end hole or multipurpose catheter, including measurement of cardiac index. Both branch pulmonary arteries should be entered for pressure measurements to exclude significant branch stenosis. A stiff 0.035" exchange wire should be positioned in the distal pulmonary artery and the wedge catheter replaced with a marker pigtail catheter for a main pulmonary artery angiogram (30 cc of contrast at 35 cc per second). The AP camera should be angled approximately 15 degrees cranial with the lateral camera in straight lateral position, to profile the RVOT–MPA. Measure the RVOT, pulmonary valve annulus and MPA diameters as well as distance from the annulus to the branch pulmonary arteries.

TECHNIQUE Choosing Valve Stent Size: The goal of valve stent implantation is to relieve all existing stenosis and provide the largest competent pulmonary valve possible. Remember, however, that many of these patients have existing conduits or homografts within which the stent valve will be implanted. Knowledge of the size of the existing surgical conduit or homograft is critical and can be used as a target size for the valved stent. If stenosis is present test balloon dilation to assure expansion of the areas to acceptable diameter is mandatory and will determine size of stent valve implant. If no stenosis is present the valve diameter should be 1–2 mm larger than the existing annulus/conduit diameter. Current stent lengths are limited but complete coverage of any narrowing without extension into the branch pulmonary arteries is the goal. Currently the only commercially available valved stent designed for the pulmonary position is a jugular venous valve (Contegra, Medtronic, Minneapolis, MN) sewn into a platinum balloon expandable stent mounted on a dilation balloon preloaded into a 22 French delivery system (Melody Transcatheter Pulmonary Valve, Medtronic, Minneapolis, MN). Valve diameters range from 16 mm to 22 mm. There is ongoing development of new valved stent designs including self expanding stents [69,70] although none are commercially available at present. In addition several of the valved stents designed for use in the aortic position are being clinically evaluated for treatment of pulmonary insufficiency.

TECHNIQUE Sheath and Wire Placement: Because of the size and stiffness of the current delivery system it is mandatory to establish excellent wire position in a distal lower lobe pulmonary artery (right or left) sub-segment with a stiff wire. Use a balloon wedge or directional catheter with a high torque directional wire (Turumo or Wholley wires) to access the distal segment then exchange for a 0.035" superstiff Amplatz wire. The balloon tipped catheter is preferred to assure you cross through the center of the tricuspid valve and not through chordea since you will be passing a large delivery sheath that could do valve damage. The balloon also makes the wire exchange easy as the tip of the catheter can be fixed in place distally by gently inflating the balloon during the wire exchange.

TECHNICAL TIPS

****Crossing the RVOT:** If stenotic and tortuous, crossing the RVOT can be challenging. If approaching from the IVC loop the balloon wedge catheter along the right lateral wall of the RA crossing the tricuspid valve with an existing RA loop. This directs the tip towards a medially displaced RVOT making it easier to cross with the directional wire. If unable to complete this maneuver consider approaching from the SVC as it is an easier catheter course.

TECHNIQUE Stent Position and Implantation: Once the wire is in position the existing sheath is removed and the delivery

system inserted over the wire and advanced to the appropriate position. Confirmation of position with both echo and angiography is critical prior to inflation. Once the sheath is retracted off the stent an angiogram can be performed through the side arm or a second catheter can be positioned in the right ventricle. Inflation should be performed with a pressure gauge to minimize risk of balloon rupture but assure full and maximal expansion. Although post implant dilation is possible if needed it does pose additional trauma risk to the tissue valve.

TECHNIQUE Post-dilation Assessment: Following stent valve implantation it is critical to maintain distal wire position both to facilitate assessment and address any complications if they develop. A catheter with both side and end holes such as a pigtail or Gensini catheter is advanced over the wire and positioned in the main PA. Pullback right heart hemodynamic measurements and RV angiogram should be performed. Echocardiographic evaluation of the implanted valve gives a baseline noninvasive measurement of stenosis and insufficiency for follow up comparison.

Indications: Percutaneous pulmonary valve implant is indicated for isolated pulmonary insufficiency if the existing pulmonary annulus and MPA measure 16–22 mm in diameter and there is RV hypertension greater than or equal to 2/3 systemic, RV dilation or symptoms of exercise intolerance. Valve implant is appropriate in the setting of combined pulmonary insufficiency and stenosis if the area of stenosis can be dilated to $\geqslant$16 mm.

TECHNICAL TIPS

*** Be Careful of Compression on Proximal Organs:** Remember that the left main or proximal right coronary artery may be in close proximity to the existing pulmonary valve annulus. Particularly in patients whose homograft or conduit is compressed by the sternum and patients with transposed great vessels there is a potential risk of coronary compression when a stent is implanted in the RVOT–MPA region. Evaluation for proximity of coronaries on pre catheterization MRI or CT as well as selective coronary angiography is necessary in this situation. If there is a concern simultaneous selective coronary angiography during balloon dilation of the RVOT is required.

Contraindications: Because of the large size of the current delivery system vascular acess that will accept a 22 French introducer is mandatory. Patients who have RVOT, homograft, or MPA stenosis $<$16 mm diameter that is not dilatable should not have a valve implanted. Patients with a RVOT–MPA region with a minimum diameter $>$22 mm are currently not candidates although there is research into new devices or valves that would be appropriate for large outflow tracts. Certainly the procedure is contraindicated in any patient in whom coronary compression will result from RVOT stent implantation.

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Chapter 30
Delivery of Biologics for Angiogenesis and Myogenesis

Peter K. Law, Sze Piaw Chin, Huynh Duong Hung,
Thach N. Nguyen, Quan Zhou Feng

GENERAL OVERVIEW

The issue of how to optimize delivery of autologous skeletal myoblasts, bone marrow stem cells, endothelial progenitor cell, angiogenic proteins, or genes to the site of myocardial injury is an ongoing and active area of investigation. If these biologics are not able to reach these sites they will not be able to improve any of the multiple cardiac functions.

*Basic; **Advanced; ***Rare, exotic, or investigational.
$ <100.00 $US extra; **$$** >100.00 $US extra
⌛ <10 minutes extra; ⌛⌛ >10 minutes extra
♦ low risk of complications; ♦♦ high risk of complications

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Several modes of delivery have been investigated, including direct intramuscular injection into the myocardium, systemic intravenous administration, and transendocardial and trans-epicardial injection into the endocardium or epicardium, as well as intracoronary injection [1].

Direct intramuscular injection into the damaged myocardium has been used extensively. The main advantage of this means of delivery is the ability to deliver one or more biologics directly to the damaged area. It requires time-consuming surgical procedures but allow direct visualization of the epicardium [1].

Intravenous administration of cells is the easiest mode of delivery. However, the ability of the cells to home to the site of injury is still a topic of intense research [1].

The transendocardial and trans-epicardial modes of delivery of stem cells have been used in large animal experiments as well as in the clinical setting. The main advantage of these types of delivery is an even distribution of the cells. Currently, the clinical use of catheter-based transendocardial injection is limited to one injection system, using electromechanical mapping (EMM) to generate a three-dimensional left ventricle (LV) reconstruction before the injection [1].

A less invasive technique available for cell delivery is the intra-coronary approach. With this technique, cells are delivered to the heart via an over-the-wire balloon catheter. Still unclear with this and all other modes of delivery are the appropriate number of cells needed as well as the time frame of administration required to adequately deliver the most effective number of cells to the damaged myocardium [1].

All of these approaches have been associated with relatively low uptake in the target tissue and potential homing of transplanted cells into other organs. The EMM system offers the advantage of assessing the viability of target sites before each injection and ensuring intramural delivery [2].

ELECTROMAGNETIC-GUIDED MYOCARDIAL INJECTION

Equipment: The electromechanical mapping system uses (1) a location pad containing three coils generating ultra-low magnetic field energy, (2) a stationary reference catheter with a miniature magnetic field sensor located on the body surface, (3) a navigation sensor mapping catheter (7Fr) with a deflectable-tip and electrodes providing endocardial signals, and (4) a workstation for information processing and 3D LV reconstruction.

Electro-Mechanical Mapping: The mapping catheter was introduced retrograde across the aortic valve into the LV. The initial three points outlining the boundaries of the LV (apex, aortic outflow and mitral inflow) were acquired with fluoroscopic guidance. Subsequently, no fluoroscopy was needed to acquire additional sampled points within the LV chamber. The mapping process proceeded only when the catheter tip was stable on the endocardial surface as evidenced by local activation time, location, loop and cycle length stability parameters.

The system used a triangulation algorithm to reconstruct the LV anatomy, which was presented in real-time on a workstation [3]. The EMM was then used to target the specific treatment area by identifying viable myocardium (unipolar voltage $\geqslant$ 6.9 mV) within that region. Areas associated with decreased mechanical activity (local linear shortening $<$12%, indicating hibernating myocardium) were preferred [4].

The exact catheter-tip location, orientation and the injection sites were indicated in real-time on the LV map, and local electrical and location signals were traced to assure catheter stability and optimal endocardial contact. Subjects were monitored for systemic blood pressure and surface electrocardiograms throughout the study protocol and during the recovery period. Arrhythmia was evaluated from the LV mapping catheter data during mapping and by electrocardiographic recording after injection [3].

Technique of Myocardial Injection: After completion of LVEMM, the mapping catheter was replaced by the injection catheter (Biosense-Webster, Diamond Barr, CA), a modified 8Fr mapping catheter, the distal tip of which incorporates a 27-gauge needle, that can be advanced or retracted by 4–6 mm. The catheter was flushed with sterile saline for 30–45 minutes before injections, thus pre-filling the lumen before introduction of the catheter into the circulation. The injection catheter was then advanced retrograde via an 8Fr femoral arterial introducer sheath, across the aortic valve into the LV, and manipulated to acquire stable points within the target region that had been superimposed on the previously acquired 3D map [5]. Each injection site was carefully evaluated before the cells were injected. Before every injection of cells into the LV wall, the following criteria had to be met: (1) excellent loop stability ($<$4 mm); (2) underlying voltage $>$6.9 mV; and (3) presence of a premature ventricular contraction on extension of the needle into the myocardium [4].

Once a stable NOGA point was attained, the needle was advanced 4–6 mm into the myocardium. In each patient, injections were performed into foci of myocardial ischemia identified as areas of preserved unipolar voltage and abnormal wall motion via EMM (NOGA) mapping. After completion of each injection, the needle was retracted, the catheter manipulated to another endocardial site within the zone of ischemia, and a new syringe was used to perform an additional injection. After the final injection and before needle retraction, the lumen was again flushed with 0.1 mL of sterile saline [5].

TECHNICAL TIPS

***Optimal Myoblast Injection:** Endomyocardial Myoblast Injection (EMI) is an interventional procedure involves positioning an injection catheter obliquely against areas of myocardial infarction inside the left ventricle with guided endovascular navigation. An injection needle is deployed from the tip of the catheter, piercing through $<$5 mm of an infarcted zone at its inner border adjacent to the normal myocardium. Pure human myoblasts, manufactured according to current Good

Manufacturing Practice (cGMP), are slowly infused through the needle as it is being withdrawn from the myocardium. Twenty injections or less are made, delivering one billion (10^9) cells at a high concentration of 100×10^6 cells/mL into the infarcted zones. Each injection delivers 50×10^6 myoblasts in 0.5 mL.

****Injection Method Regulates Cell Distribution and Fusion:** Various injection methods aimed at even and wide distribution of donor myoblasts were tested and compared. The result indicates that delivery of myoblasts is best conducted by diagonal placement of needle into the host myocardium with slow infusion of the myoblasts as the needle is slowly withdrawn [6]. This method of myoblast injection yields homogenous and wide distribution of donor myoblasts with a high rate of cell fusion. Myoblasts injected perpendicular to myofiber orientation are partially distributed. Myoblasts injected parallel to the myofibers are poorly distributed.

Considering that a small volume of a concentrated cell suspension causes less muscle damage than a larger volume of a relatively less concentrated cell suspension, and considering that injection trauma of the healthy myocardium can cause arrhythmia, the method of myoblast delivery is essential to EMI success.

High concentration of myoblasts in the injectate expedites cell fusion because of the high cell confluence and low serum environment that are known to promote myoblast fusion. Thus, 100×10^6 cells/mL will be more effective than 50×10^6 cells/ml. Beyond this high concentration, the myoblasts tend to become too viscous for injection. They crump together and die.

The high concentration of cells also allows delivery in small volume per injection. The delivery of approximately 1.0 billion allogenic myoblasts into the infarcted myocardium of two ischemic patients using about 20 injections of 0.5 mL each was found to be safe and efficacious [7,8]. A common mistake is to make large number of injections with small volumes. The injection trauma, potentially on the viable myocardium as the needle traverses the myocardium, prompts arrhythmia and tachycardia.

Ideally, the myoblasts should be injected within the infarction bordering the viable myocardium [7,8], and not into the viable myocardium. In this way, rapid vascularization of the myotubes formed from myoblast fusion can occur. Needle puncture by itself is an act of transmyocardial revascularization, bring the developing myogenic cells in close proximity with the vasculature. The latter is essential for the maturation of myotubes into myofibers.

In summary, EMI is most effective by having more cells delivered at high concentration in low volumes, with minimal number of injections into the inner border of the infarcted area of the myocardium.

TECHNICAL TIPS

****Intramuscular and Not Coronary Injection:** EMI is intramuscular administration of cells. It deposits live myogenic cells into

the degenerative myocardium to compensate for the death of cardiomyocytes. Approximately 10^9 human myoblasts were injected through a needle timed to protrude into the myocardium for no more than 6 mm from the tip of the catheter. Twenty injections were made within 40 minutes at cell concentration of 100×10^6/mL. There was no significant change in heart rate, EKG and body temperature monitored throughout the injection period. Vital dye staining of the myoblasts before versus after the procedure showed no significant difference in cell viability. Cell passage through the injection catheter showed less than 5% of cell death [7,8]. Avoid injecting into the LV apex or mitral valve area [9]. This differs significantly from intracoronary or transcoronary infusion of cells. In the latter approach, cells were infused into the blood vessel supplying the most dyskinetic left ventricular area by means of a balloon catheter with a stop-flow technique [10].

Cell Tests Before Injection: The following form outlines the quality tests and acceptance criteria for quality tests performed on in-process product and final product of harvested myoblasts prior to autologous or allogenic EMI. It is the responsibility of the quality control (QC) manager to approve final product for batch release and final review of the Final QC Release Test Form. Physicians should not proceed with EMI unless the form is approved without failure in any one of the test categories.

Final product is the injectate. It is myoblasts plus the carrier solution. The carrier solution is formulated to enhance the survival and development of the injected myoblasts. Except for the sterility test which takes at least 14 days, all tests on the final product should be performed within one hour prior to cell implantation.

At present, there is no final product that the physician can aspirate out of a container and inject into a patient (see next Technical Tips). Myoblasts are highly metabolic cells that require constant nourishing in a rich medium such as a serum. The latter cannot be administered into a patient unless the serum is the patient's very own. Myoblast viability and potency are greatly hampered if the cells are transported in saline or serum-free media. Only 15% of myoblasts will survive a half-hour transit at room temperature. Thus, one must insist on passing the following test requirements on the final product before implantation. One cannot substitute these requirements with the specifications on the label of the shipment. The shipment may contain non-viable, impotent, and/or contaminated cells. The EMI interventional cardiologist assumes the responsibility of the cell manufacturer without quality assurance.

Carrier Solution: Early publications often describe the use of saline as the carrier solution for EMI. Saline is not nutritional enough to sustain myoblast survival. An ideal carrier solution for the myoblasts into the patient is the patient's own serum isolated on the day of the EMI. Of the 12 mL of serum separated, 2 mL will be used for tests and cryopreservation for future identification. The remaining will be mixed with one billion pure myoblasts to produce the final product. EMI proceeds upon passing the cell test requirements.

FINAL QUALITY CONTROL RELEASE TEST FORM FOR AUTOGRAFT HEART CELL THERAPY CLINICAL TRIALS

Cell Bank Number: _____ **Patient Name/ID:**_____

I Muscle Tissue Dissociation				
Test	**Specification**	**Results**	**Pass/Fail**	**Date**
Mycoplasma – Direct Culture	DNA Fluorochrome Assay – Negative 28 days culture – Negative			
Sterility	Negative/14 days culture			
II Cell Expansion				
Sterility	Negative/14 days culture			
Endotoxin	$<$1.0 EU/ml			
Potency	Myotube formation within 10 days			
Sterility	Negative/4–7 days culture			
	Continue culture for 14 days			
Identity	$>$90.0% myoblast cells			
III Final Product				
Cell Count	= 0.5 billion cells			
Viability	= 80.0% viable			
Gram Stain	Negative/No gram (+) or (−) bacteria			
Endotoxin	$<$1.0 EU/mL			
Mycoplasma – ELISA	Negative			
Sterility	Negative/14 days culture			

Final Reviewed By/Date:_____

Figure 30-1 Test Requirements Specifications for In-Process Product and Final Product for Heart Cell Therapy Clinical Trials.

In rare situations when it is not possible to obtain enough serum from the patient, 10% injectable human albumin in saline is added to form the final product for EMI. This is also a method for thinning the injectates that are too viscous for injection.

DELIVERY OF BONE MARROW MONONUCLEAR CELLS

The patients were taken to the cardiac catheterization laboratory ≈1 hour before the anticipated arrival of the bone marrow cells (BMC) from the laboratory. Left heart catheterization with biplane LV angiography was performed. Subsequently, EMM of the LV was performed. The general region for treatment was selected by matching the area identified as ischemic by previous SPECT perfusion imaging [4].

TECHNICAL TIPS

****Cell Size:** Many studies underscore the cautious approach necessary to the use of intracoronary stem-cell infusion in humans. Safety assessments in humans must account for the small diameters of the bone marrow and progenitor cells used in clinical studies (10–12 μm) compared with the mesenchymal stromal cells or umbilical cord blood–derived somatic stem cells (20 μm) that caused microinfarctions in animals. Also, freshly aspirated cells are smaller than processed cells. In addition, parameters such as cell dose and concentration, compound viscosity, and rate of coronary infusion may have important impacts on the safety of the treated myocardium, as may the status of the recipient tissue, because ischemic myocardium may differ in microresponses and/or macroresponses from infarcted myocardium. Thus, clinical research needs to be carefully planned with selection of the appropriate delivery method as well as cell population and dosing parameters that may carry the best risk-to-benefit profile [4].

MYOBLAST IMPLANTATION DURING CABG SURGERY

Three to four weeks after muscle biopsy all patients underwent conventional aortocoronary bypass surgery. During cardiopulmonary bypass surgery, after all graft sutures had been finalized immediately before removing extracorporeal circulation and while the heart was initiating spontaneous heartbeat, the myoblasts were injected subepicardially by multiple injections with an angled needle (Steriseal Ophthalmic cannula 23G, Maersk Medical Ltd. Redditch, B98 9NL GB) that allows tangential injection of cells under the epicardium. Myoblasts were implanted in those segments previously identified by echocardiography as akinetic or dyskinetic in and around the infarct. Areas receiving cells were identified before surgery by echocardiogram and these same areas were analyzed during follow-up to determine changes in regional contractility. Before proceeding to myoblast implantation, a sample of the harvested myoblasts was used for

microbiology cultures, including gram staining to determine culture contamination [11].

INTRACORONARY INJECTION

In human studies, the combined experience from more than 100 patients with recent myocardial infarction suggested that transplantation of bone-marrow-derived adult progenitor cells by intracoronary infusion was both feasible and safe. There was no excessive myocardial damage (e.g. repeated elevation of troponin) or adverse systemic inflammatory response related to the injection (e.g. elevation of C-reactive protein), and there were no deaths, malignant arrhythmias, or arrhythmias induced by electrophysiological studies during the post-discharge follow-up (range 3 to 12 months). However, in a pilot study of six patients with ischemic cardiomyopathy who received intracoronary infusion of mononuclear autologous bone marrow cells, one patient acquired hypotension with troponin elevation, probably because of microembolization of the cellular compound [12].

COMPLICATIONS

Intramyocardial delivery of therapeutic substances can be achieved by either direct injection after open-chest thoracotomy (transepicardial) or catheter-based techniques (transendocardial) using electromechanical mapping or fluoroscope guidance. However, in some of the injection sites, the endocardial injury seemed to be exaggerated. These injuries could be classified into three types: (1) type A injury included the desired formation of endomyocardial slots combined with microscopic tracks of hemorrhagic infiltration surrounded by the injected compound; (2) type B injury induced the formation of endocardial ecchymosis caused by an exaggerated or forceful injection; (3) type C was a transmyocardial injury with possible deterioration to myocardial perforation and pericardial tamponade, also related to an excessive injected volume. Data derived from animal and human studies indicated no arrhythmia, infection, myocardial inflammation, increased fibrosis, or perforation caused by catheter-based techniques. In a report by Dohmann *et al.* on the post mortem findings on a single patient after transendocardial injection of bone-marrow-derived mononuclear cells, there was no abnormal or disorganized tissue growth, no abnormal vascular growth, and no enhanced inflammatory reaction in the heart [12].

By contrast, Kastrup *et al.* [13] reported direct injury from intramyocardial injection of vascular endothelial growth factor (VEGF) in five patients (6.25%) with severe stable angina who participated in the Euroinject One study. Complications included pericardial tamponade, high-degree atrioventricular block, ST-segment elevation, myocardial infarction, embolic events, and sepsis, all of which were procedure related and independent of the injected compound. These findings emphasize the importance of technical considerations, especially in infrequently used procedures, and they may be minimized by careful patient selection and preprocedural assessment by different imaging

modalities. Detailed patient notification of the potential procedure-related risks is mandatory [12].

Regardless of the delivery method, the local effects of the transplanted cells must be thoroughly considered. These include exaggerated inflammation or aberrant tissue formation that might impair myocardial function. Yoon *et al.* [14] showed that direct transplantation of unselected bone marrow cells into the acutely infarcted myocardium may induce significant myocardial calcification in both infarcted and normal myocardial regions. Similarly, transplanted undifferentiated mesenchymal stem cells have been shown to develop into fibroblastic scar tissue [15]. Li *et al.* [16] did not find calcification after injection of bone marrow cells, although there were fibrotic changes within the myocardium that could have been cell- or injection-related [12].

NEW TECHNIQUES

Guidance of Targeted Injections into Border and Core of Scarred Myocardium: The purpose of the study is to use (a) dysprosium-based contrast agent (sprodiamide) to confirm the site of myocardial injection and (b) T1-enhancing magnetic resonance (MR) contrast media to mark the myocardial target and T2*-enhancing contrast media to demonstrate injection sites in the margins or core of infarction on delayed contrast-enhanced images. A phantom and six pigs subjected to chronic infarction (8 weeks) underwent MR-guided experiments. At inversion-recovery gradient-echo imaging, gadoterate meglumine (0.1 mmol/kg) was intravenously administered to delineate scar tissue. A catheter fitted with multiple receiver coils was used to visualize catheter navigation and injection sites. A steady-state free precession (balanced fast field-echo) sequence was used for MR fluoroscopy. A high-resolution multiphase balanced gradient-echo cine MR sequence was used after intramyocardial deposition of sprodiamide. The results showed that in the phantom and in vivo, the actively guided catheter produced a high signal intensity at the terminal portion of the shaft and tip. Scarred myocardium was recognized as a bright region on gadoterate melamine-enhanced images. Intramyocardial injection of sprodiamide caused local and persistent signal intensity loss, and the extent was volume dependent on balanced fast field-echo and T2-weighted turbo spin-echo images. So sprodiamide allows visualization of injection sites within enhanced infarction. A catheter with integrated receiver coils aided in effective catheter guidance and precise intramyocardial injection [17].

Trans (coronary) Venous Myocardial Access Procedure: The swine were sedated, ventilated, and monitored (cardiac rhythm, oxygenation, and blood pressure), and then prepped and draped in standard surgical fashion. Six Fr arterial (Cordis, Miami, Florida) and 14Fr venous (Transvascular, Menlo Park, California) femoral sheaths were placed percutaneously. The left main coronary artery was selectively

engaged with a 6Fr Hockeystick diagnostic catheter (Medtronic) and angiography performed with emphasis on venous follow-through phases to determine coronary venous anatomy, anomalies, and coronary sinus location.

The coronary sinus (CS) was accessed by placing a 7Fr Porcine 3 catheter into the right ventricle, withdrawing with clockwise torque across the tricuspid valve. Using this technique, the catheter tends to fall into, or near, the CS. An exchange length, 0.035-inch hydrophilic angled wire (Terumo) with J-tip was advanced into the CS, through the great cardiac vein (GCV), and into the anterior interventricular coronary vein (AIV). The diagnostic catheter was withdrawn, with the wire in place, and a 14Fr CS guide (Transvascular) and introducer were placed with conventional over-the-wire technique. After removal of introducer, a subselective catheter (Transvascular) was then placed over the wire, through the CS guide, and into the AIV. The hydrophilic wire was then exchanged for a 0.014-inch HiTorque floppy (Guidant, Temecula, California) wire. The TransAccess catheter is a 6Fr, monorail, composite catheter system combining a phased-array IVUS (compatible with JOMED IVUS, JOMED, NV) and a pre-shaped, sheathed, extendable 24-gauge nitinol needle. This TransAccess catheter was advanced over the 0.014-inch wire and into the AIV in preparation for myocardial access [18].

Intravascular orientation was performed using the corresponding artery, pericardium, and ventricular chamber as landmarks with IVUS imaging. After confirmation of position within the coronary vein and with respect to surrounding structures, the nitinol needle was extended into the myocardium. A 27-gauge microinfusion (IntraLume) catheter was advanced through the needle and into the myocardial tissue. Because the myocardial tissue is a potential space, and without room for prolapse, all of the force for the otherwise floppy IntraLume catheter is forward, essentially allowing this catheter tip to become a drill capable of tunneling through remote myocardium in plane with the needle puncture [18].

Magnetic Navigation in Cell Delivery: Magnetic navigation is the use of adjustable magnetic fields to precisely direct wires and equipment for clinical applications. Procedures are based on the production of a three-dimensional reconstruction of the vessel lumen from standard angiographic images. Knowledge of the positions of the table and image intensifier during angiography allows calculation of the vessel coordinates in real space within the patient's chest. The applied magnetic field can be changed at any time to redirect the wire tip in order to improve navigation through complex and tortuous anatomy [19].

However, delivery of stem cells to the correct areas has remained inexact with relatively crude methods of identifying where the injections are to be placed on delivery and where they have been placed on restudy. The identification of infarcted areas on MSCT[34] may add a further tool that can be integrated into the magnetic navigation system (MNS) for such target localization. The MNS gives highly precise

three-dimensional control and, with the possible development of a magnetically enabled injection catheter, the current MSCT integration may help localization of the infarct area to give a more tailored delivery. The combination of such localization techniques with the mapping abilities of systems like the recently developed NOGA® XP Cardiac Navigation System (Biosense Webster Inc./Biologics Delivery System Group, Diamond Bar, CA) may give complementary information and result in more exact delivery [19].

Automatic Injection Catheter: The rapid development and the great demand for EMI will soon necessitate the development and production of the automatic injection catheters capable of delivering automatically larger amounts of myoblasts via locational precision targeting, optionally controlled injection depth and timing to the infarcted endocardium. The automatic injection catheter will have an electrode-feedback system that detects the electrical silence or diminished electrical activity of the scarred tissue and automatically and rapidly injects large quantities of myoblasts into the target in real time. The catheter detects the infarcted site at virtually the same time as it implants the cells, thus overcoming the severe problem of the heart moving during the procedure. The latter motion often dislodges the needle and expels the myoblasts while the interventional cardiologist is determining whether the needle is inside the endocardium. The automatic injection catheter tip has a needle that is triggered to protrude by a defined distance that is long enough to safely inject cells into the infarcted endocardium but not long enough to puncture the epicardium. The system minimizes operator error by virtue of an electronic-mechanical coupled system that electronically detects one or more spots within the infarcted endocardium and automatically injects a controlled amount of myogenic cells into and/or around that same site, all without human intervention [20].

An important feature of this catheter is that the injection be carried out rapidly while the heart is beating. During anesthesia, the heart may contract approximately once per second. The catheter solves the timing problem by detecting contact with a weak or degenerative spot and then quickly injecting the cells automatically. Preferably this time is less than one second and most preferably less than 0.5 seconds.

The catheter: (1) accesses the interior of the heart via a percutaneous and endovascular route without necessitating opening of the chest cavity or an open heart surgery; (2) electrically detects even minute degenerative areas of the endocardium; (3) inserts a needle of 3–5 mm long, 27–22 gauge, diagonally into the endocardium, at and/or around the infarcted myocardium; (4) injecting 30–50 million myoblasts; (5) through an electrically coupled feedback circuit and mechanical system; and (6) within less than one second [20].

The automation promises to increase injection precision and reduces cell administration time. It can easily be incorporated into the Bioheart's MyoCath™ [21] and /or the Cordis' Mozart [22] cell injection catheters with enhancement.

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