Practical Manual of Interventional Cardiology

Annapoorna Kini
Samin K. Sharma
Editors

Second Edition

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Basics of Radiation Safety

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Gurpreet S. Johal, Reza Masoomi, and Joseph Sweeny

Introduction

Cardiac catheterization laboratories use X-ray radiation to perform various cardiac procedures. The X-ray beam is emitted from the tube below the catheterization table. X-rays are composed of photons of different energies, and those which have abundant energy to disrupt the cellular structure or genetic makeup of an individual cell are characterized as ionizing radiation. A biomolecule can be altered by reacting with radiation-generated free radicals or by being directly ionized by radiation [1].

More than 95% to 99% of the X-ray energy that enters the patient is either absorbed or scattered within the patient [1]. The remaining 1% to 5% of the incident X-ray penetrates the subject, reaching the image detector to form the image or scattered to nearby medical personnel [1]. The risk of radiation injury is of particular concern for physicians who perform invasive cardiovascular procedures, as they are the most highly exposed healthcare workers [1].

Often, focus on procedure complexity dominates the importance of personal and patient radiation protection. More complex procedures such as percutaneous interventions of chronic total occlusions and structural heart diseases are being performed, leading to longer procedure times and radiation exposure [2]. Fortunately, technological advances have improved image quality while utilizing lower doses of X-ray radiation [2]. Still, it is imperative to emphasize personal and preventative strategies to minimize radiation exposure, as the dangers of repeated radiation exposure are serious, but not obvious.

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Biological Effects

The biological effects of radiation are classified as either tissue reactions (formerly called deterministic effects) or stochastic effects (also known as probabilistic effects) (Table 1.1) [3]. Tissue reactions occur if the radiation-induced injury to a cell exceeds its ability to repair itself and maintain function [1]. Tissue reactions are dose-dependent and become macroscopically apparent once a threshold radiation dose is exceeded [1]. Subsequent larger doses of ionizing radiation lead to more extensive injury in a dose-related manner, as more cells are affected [1]. Subthreshold doses may also cause cellular injury and death but the injury is not evident because an insignificant number of cells are involved [1].

Skin injury is the most common tissue reaction seen with X-ray fluoroscopy exposure. Skin injuries typically occur at the site of the X-ray beam entrance (usually the patient's back) and take on the rectangular shape of the X-ray beam [1]. These injuries vary in severity from erythema to desquamation, ulceration, and necrosis [1]. Other tissue reactions include cataract formation, bone necrosis, fetal organogenesis, and cardiac defects including damage to the myocardium, cardiac valves, and coronary arteries [1]. Tissue reactions usually occur days to months following radiation exposure, as it takes time for molecular damage to evolve and cause sufficient cellular dysfunction that later manifests at the macroscopic level [1, 3].

Stochastic effects result from radiation-induced damage of a cell's genetic material, deoxyribonucleic acid (DNA), with the cell surviving the intrinsic repair process [3]. Unlike tissue reactions, stochastic effects are not known to have a dose threshold, and the severity of an injury is not dose-related [1]. Rather, the probability of a stochastic event is thought to increase approximately linearly [1]. These relationships are the foundation for the "linear-no threshold" theory and the basis for ALARA "As Low As Reasonably Achievable" principle. Theoretically, a single X-ray photon ionizing a strategic atom within a portion of DNA that encodes a critical gene could develop into a concerning malignancy, and this is the reason why radiation exposure should always be minimized [1]. Stochastic effects typically

Table 1.1 Biological effects of ionizing radiation

	Tissue reactions "Deterministic"	Chance damage "Stochastic"
Dose level	Medium to high	Low
Latency period	Short (days or weeks)	Long (years)
Threshold dose	Yes (the level at which effects begin to appear is known)	Probably no threshold exists, but some uncertainty. The chance of developing a disease is directly proportional to the amount of radiation exposure.
Biological mechanism	Predominantly cell death	Cell damage
Sample clinical effects	Skin injury, cataract, hair loss, sterility	Cancer, hereditary disorders, inherited defect in offspring

Adapted from Picano et al. [3]

occur after a period of latency of several years, and the likelihood an effect will occur is further influenced by several factors including a person's age, gender, genetics, and many environmental factors including background radiation [1, 3]. For every 100 patients exposed to 100 mSv of radiation, one is at risk of developing a solid malignancy or hematological malignancy [4].

Regulatory Recommendations

The Society for Cardiac Angiography and Intervention outlines the basic principles for radiation safety and protection in the cardiac catheterization laboratory.

- Radiation-induced biological effects are the result of random statistical probability for low radiation doses. The probability of these effects is directly proportional to the radiation dose received.
- Since radiation-induced biological effects are random, and no threshold dose exists for these effects, even a small dose could potentially induce biological effects; therefore, no level of radiation exposure can be considered completely safe.
- Radiation exposure is cumulative and there is no washout phenomenon as with other toxin exposures.
- Each person involved in the cardiac catheterization laboratory has accepted a certain degree of risk posed by radiation exposure.

The National Council on Radiation Protection (NCRP) and the International Commission on Radiological Protection (ICRP) are regulatory bodies from the United States and Europe. They recommend healthcare workers not receive occupational radiation exposure higher than the dose limits published, with standards set forth by the ICRP being more stringent [1]. There are two types of occupational dose limits in the NCRP and ICRP recommendations. The first provides occupational dose limits for specific organs or tissues, and is expressed as an equivalent dose for deterministic effects involving an organ or tissue [5]. The second establishes an acceptable risk level for cancer induction, and is expressed as an effective dose for stochastic effects throughout the body [5]. An effective dose is intended to be proportional to the risk of radiation-induced cancer [5]. In the United States, the Occupational Safety and Health Administration and state regulations provide specific requirements for personal dosimetry when using X-rays [5]. Current regulatory considerations for radiation dose limits are listed in Table 1.2.

Pregnancy does not preclude one from working in an area of occupational radiation exposure [5]. However, additional restrictions and safety measures should be taken to reduce occupational exposure of the patient and developing fetus than those detailed in Table 1.2 [5]. Once a worker has voluntarily declared her pregnancy, the ICRP recommends an additional dose to the conceptus does not exceed more than 1 mSv during the remainder of the pregnancy [5]. The NCRP in this situation recommends a 0.5 mSv equivalent dose monthly limit for the conceptus (excluding

Table 1.2 Current regulatory considerations for radiation dose limits

Tissue	Risk	Recommended maximum dose	
		NCRP	ICRP
Whole body	Stochastic	50 mSv/year	20 mSv/year, averaged over defined periods of 5 years, with no single year exceeding 50 mSv
Lens of the eye	Deterministic	150 mSv/year	20 mSv/year, averaged over defined periods of 5 years, with no single year exceeding 50 mSv
Extremities (hands and feet), skin or individual organ	Deterministic	500 mSv/year	500 mSv/year (for skin: averaged over 1 cm² of skin regardless of the area exposed)
Embryo-fetus	Deterministic	5 mSv/entire pregnancy, and not to exceed 0.5 mSv/month	1 mSv/remainder of the pregnancy once declared
General public	Stochastic	1 mSv/year	1 mSv/year

National Council on Radiation Protection and Measurements (NCRP)
International Commission on Radiological Protection (ICRP)
10 mSv = 1 rem
Adapted from Miller et al. [5]

medical and natural background radiation) [5]. Moreover, workers in the United States who do not wish to declare their pregnancy are not required to do so [5].

ALARA

ALARA, which stands for "As Low As Reasonably Achievable," is the fundamental radiation safety principle and unifying goal of radiation safety programs. All operators are to follow protocols to minimize the use of radiation while performing procedures and adhere to ALARA limits as detailed in Table 1.2. If the radiation exposure exceeds the limit, the radiation safety officer at a given facility is expected to review the individual case. The focus of the review is to determine the reason for high radiation exposure and improve safety procedures to minimize excessive exposure to radiation in the future. Three principles help in maintaining ALARA practice:

- Time: Reducing the duration of radiation exposure will reduce the dose.
- Distance: Radiation follows the inverse square law. Doubling the distance from the source will reduce radiation exposure by a factor of four.
- Shielding: Using absorbent material like lead for X-rays reduces exposure.

Radiation Units and Dose Monitoring

Patient exposure to radiation must be documented and is measured as Air Kerma (Kinetic Energy Released in MAtter) and dose area product (DAP) [6]. Air Kerma is the amount of kinetic energy delivered to air and is measured 15 cm towards the

Table 1.3 Description of radiation units for dose monitoring

	Description
Fluoroscopic time	Total time of fluoroscopy used during a procedure. This does not include the total time of cine imaging. As a result, this parameter underestimates the total radiation dose delivered to a patient. It is recorded in minutes (min)
Air Kerma (AK)	Refers to X-ray energy delivered to air at the interventional reference point. It is the amount of energy released by the interaction of the radiation with a unit mass of air. It is a measure of radiation exposure which correlates with the risk of deterministic effects. It is recorded in Grays (Gy)
Dose area product (DAP)/Air Kerma-area product	Product of total radiation dose and area of the X-ray field. It is a measure of radiation exposure which correlates with the risk of stochastic effects. It is recorded in $Gy.cm^2$

x-ray tube side of the isocenter, the point where the primary x-ray beam intersects with the rotational axis of the C-arm gantry [6]. Air Kerma has been associated with the deterministic effects of radiation. DAP, is also referred to as Air Kerma Area Product, is the product of Air Kerma and the x-ray radiation field area [6]. It is measured in Gray·cm2 and is thought to correlate with the stochastic effects of radiation [6]. Refer to Table 1.3 for the description of radiation units for dose monitoring.

Operator exposure is expressed as an equivalent dose for organ-specific exposure and an effective dose for whole-body exposure [6]. The effective dose represents the sum of equivalent doses from different tissues, adjusted to the radiation sensitivity of each tissue [6]. The effective dose can be estimated by multiplying the average equivalent dose in each exposed tissue by a tissue weighting factor and summing these values over the whole body.

Safety Components

During cardiac catheterization, patients could receive a radiation dose on average of 8–10 mSv and this increases significantly with more complex procedures. Protective equipment, if worn correctly, prevents absorption of nearly 95% of scatter radiation, and operators receive an average effective dose of 0.2 to >100 microsieverts (μSv) per procedure with a per-procedure average of 8 to 10 (μSv) [1, 4]. Therefore, an interventional cardiologist who performs around 500 procedures/year will receive, in addition to background exposure, a cumulative dose of nearly 10 mSv/year, and in most extreme situations, approximately 300 mSv over an active 30-year career [1]; a projected professional lifetime attributable excess cancer risk of 1 in 100 [3].

If an operator receives a high occupational radiation dose, an investigation must be performed. This occurs if the total effective dose an operator receives is >0.5 mSv/month, lens equivalent dose >5 mSv/month, or extremity equivalent dose >15 mSv/month [6]. The first step in the investigation of a high (or unusually low) value is to confirm the validity of the dosimeter reading. If dosimeter reading is considered to be accurate, the next step in the investigative process is to have a trained physicist

or experienced colleague observe the operators' work habits with close monitoring of equipment settings, operator positioning relative to the radiation source, and use of personal protective equipment [6].

Radiation Monitoring

- Personal dosimetry monitors are the gold standard for radiation surveillance and are required to be used by all health care providers who are employed in areas where radiation is being utilized [6].
- There are two options for measuring effective dose equivalent for workers who use lead aprons.
  - The first option is to have one badge on the thyroid collar outside the apron and the other badge under the apron at the waist level [1, 6].
  - The second option is to use only one badge outside the lead apron on the thyroid collar [1, 6].
- Eye dose can be measured with a dedicated lens eye dosimeter, which gives the most accurate measurement when placed on the side of the head closer to the eye [6].

Shielding

- Lead garments to protect the gonads and approximately 80% of the bone marrow.
  - 0.5 mm lead apron stops approximately 95% of the scatter radiation [7].
- Separate thyroid collars, especially for the young and in those whose radiation dose exceeds 4 mSv/month [7].
- 0.25 mm lead eyeglasses for eye protection (radiation can cause posterior subcapsular cataracts).
- Use of below the table-mounted shields.
- Transparent ceiling-mounted shields.
- Disposable radiation-absorbing sterile drapes.
- Proper maintenance and periodic inspection (at least once a year) of lead aprons.

Procedural Issues

- Precautions to minimize exposure to patient and operator.
- Utilize radiation only when imaging is necessary to support clinical care.
- Minimize the use of cine angiography.
- Minimize the use of steep angles of the X-ray beam. Left anterior oblique cranial angulation has the highest degree of scatter radiation exposure to the operator.
- Minimize the use of magnification modes.
- Minimize the frame rate of fluoroscopy and cine.

- Keep the image intensifier close to the patient.
- Utilize collimation to the fullest extent possible.
- Monitor radiation dose in real-time.

Precautions to Minimize Operator Exposure

- Use and maintain appropriate protective garments.
- Maximize distance of the operator from the X-ray source and patient.
- Keep above and below table shields in the proper position at all times.
- Keep all body parts out of the field of view.

Precautions to Minimize Patient Exposure

- Keep table height as high as comfortable for the operator.
- The height of the table can significantly affect the scatter radiation. The patient should be placed away from the radiation source (ideally 70 cm away) and close to the image intensifier as much as possible [6].
- Vary the imaging beam angle to minimize exposure to any one-skin area.
- Minimizing steep LAO and anteroposterior cranial angles.
- Fluoroscopy dose is more sensitive to angulation changes compared with acquisition dose likely due to higher tissue-based attenuation of the lower-dose fluoroscopy X-ray beam in angulated projections [6].
- Keep the patient's extremities out of the beam.

Impact on Patient Care

Inclusion of radiation dose on cardiac catheterization reports is mandatory and it helps identify which patients need appropriate follow-up for possible radiation-related tissue injury postoperatively. Follow-up should be based on the radiation dose level as detailed below [7]:

- AK >5 Gy:
  - Patient education regarding potential skin changes like redness and report if seen.
  - The patient to be contacted within 30 days post-procedure.
- AK >10 Gy:
  - Qualified physicist to perform detailed analysis and calculate peak skin dose.
  - Office visit in 2–4 weeks with skin examination.
- Peak Skin Dose (PSD) >15 Gy:
  - Contact hospital risk management.
  - Notification to regulatory agencies.

Accordingly to the Society of Interventional Radiology, documentation of overexposure is also advised if DAP radiation exceeds 500 Gy.cm2, or fluoroscopy time is >60 minutes [6]. If a radiation-related tissue injury develops during the follow-up period, the patient should be referred to an experienced provider dealing with such complications. In most circumstances, a skin biopsy should not be performed.

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Vascular Access

2

Gurpreet S. Johal and Nitin Barman

Introduction

Over the past decade, the use of a transradial approach (TRA) to obtain arterial access for cardiac catheterization procedures has gained popularity and surpassed the use of a femoral artery approach (FA) [1, 2]. The brachial artery, axillary artery, ulnar artery, and femoral artery cut down for access are rarely used.

Femoral Access

Obtaining femoral artery access is crucial for procedural and clinical success, and remains one of the key technical challenges for an interventional cardiologist. Access site complications are an important cause of cardiac catheterization morbidity and mortality.

- A "high" sheath insertion above the inguinal ligament increases the risk of retroperitoneal bleeding.
- A "low" sheath insertion into the profunda femoris or the superficial femoral artery (SFA) may result in an arteriovenous fistula, pseudoaneurysm, or limb ischemia.

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Contraindications for Femoral Artery Access

- Recommend not to perform if INR ≥2.0.
- If a patient is taking warfarin (INR ≥2.0) and a percutaneous procedure needs to be performed urgently or emergently via a FA, 1–2 units of fresh frozen plasma should be given to correct the coagulopathy. Patients who are considered high-risk for thromboembolic events should be bridged with unfractionated heparin or low molecular weight heparin.
- Avoid in patients taking a factor Xa inhibitor (rivaroxaban, apixaban) or a direct thrombin inhibitor (dabigatran), unless these medications have been held for >24–48 hr [3].
- Avoid in patients with morbid obesity, severe peripheral vascular disease, or aortic dissection.

Sheath Selection

- A 5 French (Fr) sheath will suffice for most diagnostic cardiac procedures.
  - If the pretest probability of disease is low, consider using a 4 Fr sheath.
  - Common femoral artery (CFA) diameter in women and diabetics tends to be smaller; consider using a 4 Fr sheath [4].
- The sheath can be upsized as needed for interventions (Table 2.1).

Table 2.1 Femoral sheath size by the procedure

Procedure		Sheath size
Diagnostic cardiac catheterization		5 Fr
PCI— most PCIs, two-stent strategy for bifurcation lesions [DK Crush technique, Culotte technique], bailout stent technique [TAP technique, Reverse Crush (internal) technique], orbital atherectomy or rotational atherectomy burr <2 mm		6 Fr
PCI with a planned two-stent strategy for bifurcation lesions [Mini Crush technique, modified T technique, SKS technique, V technique] or rotational atherectomy burr of 2 mm		7 Fr
Rotational atherectomy burr of 2.15 mm or 2.25 mm		8 Fr
Balloon aortic valvuloplasty	Tyshak 16–20 mm balloon	8 Fr
	Vida 16–20 mm balloon	8 Fr
	Z-MED 20 mm balloon	10 Fr
	True 20 mm balloon	12 Fr
Impella	2.5	13 Fr
	CP	14 Fr
Transcatheter aortic valve replacement	SAPIEN 3 (23 mm, 26 mm) SAPIEN 3 (29 mm) Evolute Pro Plus (23 mm, 26 mm, 29 mm) Evolute Pro Plus (34 mm)	14 Fr 16 Fr 18 Fr 22 Fr

Pearls

- In obese patients, stretch the skin tightly over the femoral head by moving the pannus out of the way and taping it across the body.
- Use long sheaths (25 cm or 45 cm) in patients with tortuous iliac arteries or when the CFA is located deep below the subcutaneous tissue.

Needle Used

- High-risk patients may require the use of a micropuncture needle for more controlled access into the CFA, so a vascular closure device may be employed to close the access site after the procedure. These patients include:
  - Extremely obese patients with deep vasculature.
  - Patients who are anticoagulated or have a coagulopathy.
  - Patients with known or suspected peripheral arterial disease (arterial access should be obtained in a relatively non-diseased segment of the vessel).

Ideal Access Location

- The segment of the common femoral artery below the inguinal ligament (1–2 cm below the line traced from the anterior superior iliac spine to the pubic tubercle) is the ideal location for arterial access.
- This correlates roughly to an area that is at the mid-third of the femoral head, which is usually above the femoral bifurcation and below the lowest point of the course of the inferior epigastric artery (Fig. 2.1).

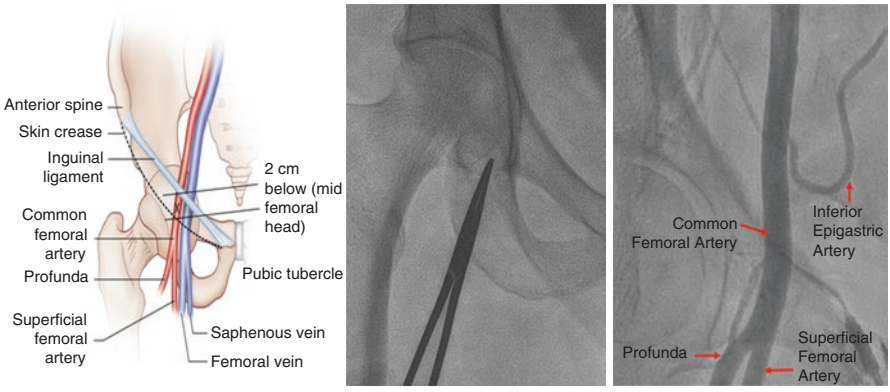

Fig. 2.1 Location and anatomy for femoral access

- The ultrasound (US) guided approach is safe and effective. US use to cannulate the CFA reduces the number of attempts, the time required to achieve successful access, and the rate of vascular complications [5].
- Femoral artery bifurcation is below the inferior border of the femoral head in 80% of cases, and below the inguinal ligament and middle of the femoral head in all patients [1].
- The femoral artery lies on the medial third of the femoral head in 92% of patients, and is completely medial to the femoral head in 8% of patients [6–8].

Pearls
- The inguinal crease is not a reliable landmark; however, we find it safer to puncture below it [9].

Common Steps for Micropuncture and Regular Access Needle

- Fluoro the femoral head with an overlying hemostat to mark the inferior border of the femoral head and palpate the point of maximal pulsation at this level.
- Give lidocaine 1–2% subcutaneously. Create a subcutaneous wheal at the entry site with 5 cc of lidocaine and then gradually deliver an additional 10–15 cc of local anesthetic to the deeper subcutaneous tissue, covering the anticipated needle path from the skin to the arterial wall.
- The needle entry point at the skin level should be at the lower border of the femoral head. Aim for an area between the inferior border of the femoral head to the mid femoral head (Fig. 2.1).
  - Monitor the patient for any vagal reaction, and the ECG for bradycardia.
- In rare cases, a small skin nick followed by the opening of the subcutaneous space gently with blunt forceps is required.
  - This provides a pathway for blood to ooze out of the skin in case of bleeding and allows for early identification of complications.
  - This step should be considered when there is difficulty inserting a sheath or when Perclose™ technique of closure is planned, especially for the closure of large sheaths.
- Enter the anterior wall of the femoral artery by advancing either the 18-gauge needle or the 21-gauge micropuncture needle at a 45° angle until there is backflow of arterial blood at the needle hub (Fig. 2.2. Step Ia).
  - Advancing the needle at a more vertical angle can result in kinking of the sheath and a more horizontal angle can result in a high stick.

– The backflow of blood when an 18-gauge needle is used should be brisk and pulsatile.
– The flow through a micropuncture needle is six times less compared with blood flow through an 18-gauge needle. The backflow may not appear pulsatile, but should be steady.

18-gauge Needle Steps

- The 0.035″ guidewire is threaded through the needle. There should be no resistance as the guidewire is advanced. If resistance is encountered, fluoroscopy should be used to ensure guidewire advancement is correct (Fig. 2.2. Step II a, b, and c).

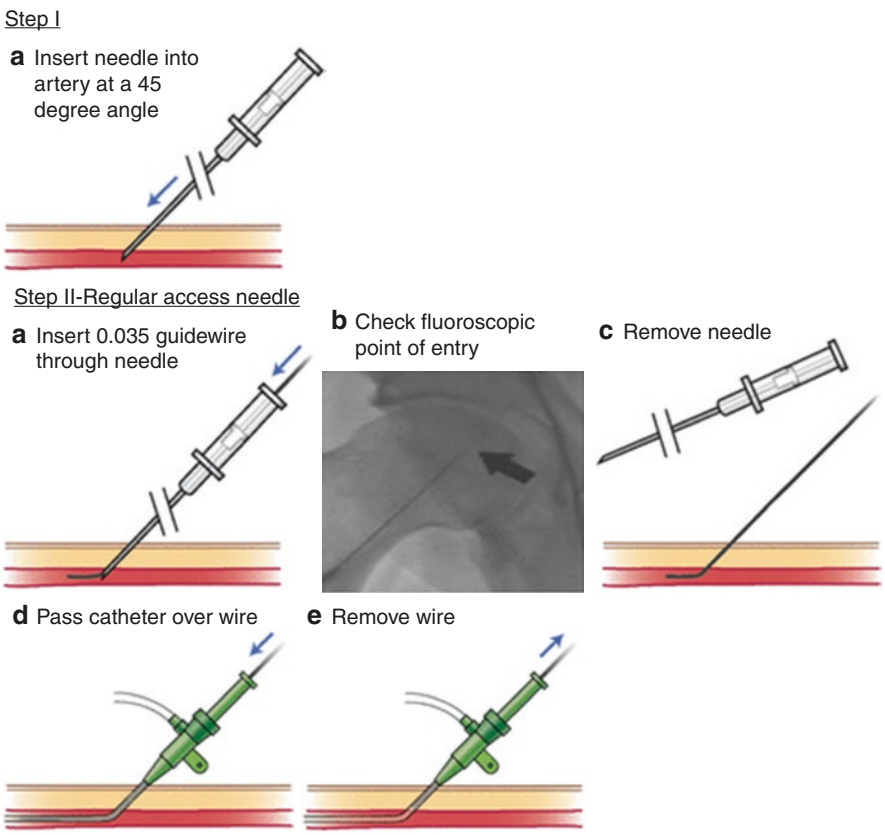

Fig. 2.2 Steps of femoral access

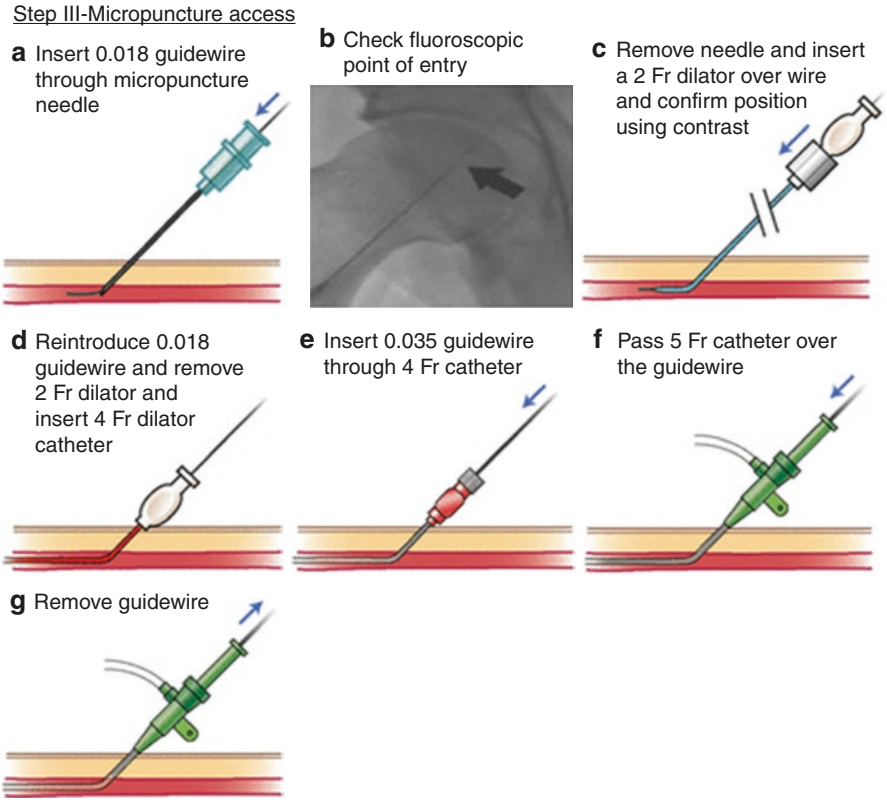

Fig. 2.2 (continued)

- Next, the needle is removed, the sheath/dilator system is advanced over the guidewire until the unit is well within the lumen of the vessel, and the guidewire and dilator are removed, leaving the sheath within the artery. (Fig. 2.2. Step II d and e).
- Once arterial access is obtained, a femoral arteriogram should be obtained by injecting dye through the sheath.

Micropuncture Steps

- Access the artery as described above using the needle from the micropuncture access kit.
- Once arterial access is obtained, the accompanying 0.018″ guidewire is advanced through the needle (Fig. 2.2. Step III a). Because the wire is straight (not J-tipped),

it is essential to check by fluoroscopy that the wire tip is in the iliac artery and has not advanced into a small branch (Fig. 2.2. Step III b).

- Remove the needle, thread the 2 Fr dilator accompanying the 4 Fr sheath/dilator system over the guidewire into the artery (Fig. 2.2. Step III c), and remove the guidewire. Alternatively, the 4 Fr micropuncture sheath/dilator system can be inserted directly over the guidewire. The 2 Fr dilator and guidewire is removed, leaving the 4 Fr micropuncture sheath within the artery.
- Perform an arteriogram with a 3–5 cc injection of contrast through the 2 Fr dilator to confirm the site of entry. If the arterial puncture site is not optimal, remove the catheter, hold pressure for 3–5 min to achieve hemostasis, and re-access the artery with the micropuncture needle. If the 4 Fr microcatheter was used to inject contrast and access is acceptable, insert the 0.035" guidewire and follow the steps as detailed below.
- Reinsert the 0.018" guidewire, remove the 2 Fr dilator and re-assemble the 4 Fr micropuncture sheath/dilator system, advance the system over the guidewire until the unit is well within the lumen of the vessel, remove the guidewire and dilator, and leave the sheath within the artery (Fig. 2.2. Step III d).
- Exchange the 0.018″ wire with a 0.035″ guidewire, remove the 4 Fr sheath, advance the 5 Fr or 6 Fr sheath/dilator system over the 0.035" guidewire, remove the guidewire and dilator, and leave the sheath within the artery (Fig. 2.2. Step III e, f, and g).

Angiographic Views

- The common femoral artery bifurcation is usually best visualized in the ipsilateral view 30°.
- In some cases, a contralateral view with a slight caudal projection may allow better visualization of the bifurcation.
- The angiogram should be evaluated carefully to see the level of puncture and the presence of arterial dissection or extravasation of dye due to peri-sheath leak, perforation, or back wall puncture.

Complications

- Clinical features, prevention, and treatment of various femoral artery complications are listed in Table 2.2 [2].

Table 2.2 Complications of femoral access: clinical features, prevention, and treatment

Type	Clinical features	Prevention	Treatment
Hematoma	Incidence: 5–23% Pain, swelling, or hardened area under the skin at the site If severe can cause tachycardia, hypotension, and fall in Hct Most resolve in a few weeks	Adequate compression after sheath removal Avoid arterial puncture below the femoral bifurcation	Manual pressure Mark the area to monitor change in size Obtain a vascular ultrasound to evaluate for pseudoaneurysm Other steps same as for retroperitoneal hematoma
Pseudoaneurysm	Incidence: 0.5–9% Swelling at the insertion site Large, painful pulsatile mass Bruit and/or thrill in the groin area A pseudoaneurysm can rupture or cause nerve compression resulting in limb weakness Diagnosed by ultrasound	Adequate compression after sheath removal and good hemostasis Avoid arterial puncture below the femoral bifurcation	Bed rest Small: Monitor as they spontaneously resolve after cessation of anticoagulation Large: manual or ultrasound-guided compression/thrombin injection or surgery
Arteriovenous fistula	Incidence: 0.2–2.1% Bruit ± thrill at the access site Extremity swelling, tenderness Diagnosed by ultrasound	Adequate compression after sheath removal and good hemostasis Avoid arterial puncture below the femoral bifurcation or above the inguinal ligament	Some resolve spontaneously Ultrasound-guided compression Surgical repair
Infection	Incidence ~ 0.4% 80% are diabetics with a mortality rate of 6% 50% ~ mycotic aneurysm, 50% result in mycotic aneurysm with S. aureus in 75% of cases *S. aureus* in 75% of cases The incubation period is 1 week to 1 month. Have a high degree of suspicion for any pain, erythema, swelling, drainage from the puncture site, or systemic signs of infection up to 1-month post-procedure, especially in diabetics	*VCD/manual sheath removal protocol* Clean area with an antiseptic solution. Place sterile towels and change gloves. Pull out in one fluid movement and let it bleed back and hold pressure just above the puncture site. Do not rub into the wound Avoid VCD in patients with significant PVD, CFA <5 mm, >3 prior procedures at the same site, or if the puncture is below the femoral bifurcation. Give 1 g of IV Cefazolin or 1 g of IV vancomycin (PCN allergic patients) in DM or morbidly obese patients receiving VCD	Long-term IV antibiotics or antifungals Surgical debridement and removal

Retroperitoneal hematoma	Incidence: 0.15–0.4% Moderate to severe abdominal, back, or ipsilateral flank pain Groin/hip pain with radiation to back if close to the iliopsoas muscle especially during extension of the hip Hypotension and tachycardia. Ecchymosis and decrease in hematocrit are late signs Diagnosed by CT Potentially fatal if not recognized early	Avoid puncture above the inguinal ligament Use micropuncture access in obese patients or difficult access and confirm site before sheath insertion Adequate compression after sheath removal and good hemostasis	Do not delay treatment if suspicion is high IVF/bed rest Interrupt anticoagulants and antiplatelets with blood transfusion if required May need surgical evacuation/ percutaneous balloon tamponade
Arterial occlusion by thrombo-embolism (TE)	Incidence: <0.8% 5 Ps: pain, paralysis, pallor, paresthesias, pulselessness Doppler studies/angiogram	Anticoagulation Vasodilators Careful monitoring during sheath removal and injection	Small TE may undergo spontaneous lysis Larger TE may need surgical or percutaneous thromboembolectomy or thrombolytic agents
	Percutaneous thrombectomy:		
	Access from the contralateral side and give 5000 units of heparin if no Anticoagulated		
	Cross TE with a 0.014″ or 0.018″ wire		
	Thrombectomy device is then introduced over the wire to remove any thrombi ± PTA/stent		
Iliac dissection	Usually painless and retrograde		Bed rest with follow-up clinical exams and imaging if nonflow limiting If flow limiting, PTCA + stent is the treatment of choice
Femoral neuropathy	Incidence: ~0.2% Pain ± tingling at the access site Numbness ± weakness at access site or down the leg Decreased patellar tendon reflex	Avoid injection or insertion lateral to the arterial pulsation	Physical therapy Local anesthetic injections

Radial Access

Performing cardiac procedures via a TRA compared with a FA is associated with a reduction in bleeding events and vascular complications. This is largely driven by lower rates of minor bleeding [10]. A shift to a "radial-first" strategy in the United States has improved acute coronary syndrome related outcomes, quality of life metrics, and reduced healthcare costs [10]. The failure rate for cardiac procedures using a TRA is higher than the FA and ranges from 1–5% [11].

TRA Failure

- TRA failure is usually seen in patients who are:
  - Short.
  - Elderly.
  - Female.
  - Post-CABG.
- TRA failure is usually attributed to [11]:
  - The steeper learning curve for obtaining radial access.
  - The smaller caliber of the radial artery.
  - Anatomical variations in radial artery distribution.

Advantages and Limitations of TRA for Cardiac Procedures

- Advantages [11]:
  - Reduced duration of post-procedure bed rest and length of stay.
  - Lower incidence of access site complications (bleeding, pseudoaneurysm, and arteriovenous fistulas).
  - Lower in-hospital mortality.
  - Patient comfort.
  - Reduction in overall costs.
- Limitations [11]:
  - Inability to use larger sheaths (largest recommended is 6–7 Fr sheath).
  - Increased radiation exposure (exposure is greater with the right TRA compared with a left TRA; overall exposure decreases with experience).
  - Potential for radial spasm, radial artery occlusion, other vascular complications (refer to radial complication section below).
  - Potential need for crossover to a FA.

Vascular Anatomy of the Hand

- The ulnar artery and the radial artery provide a dual blood supply to the hand (Fig. 2.3).
- The superficial palmar arch lies below the palmar fascia. It is supplied predominantly by the ulnar artery and to a lesser degree by the superficial branch of the radial artery.
- The deep palmar arch lies beneath the flexor tendons and proximal to the superficial arch. It is supplied predominantly by the deep branch of the radial artery and to a lesser degree by the deep branch of the ulnar artery.
- Patients may have variations in anatomy limiting or precluding dual blood supply to the hand. If this is the case, a TRA should be avoided.
- Allen's test or Barbeau's test can help evaluate the presence of adequate blood supply to the hand before performing a procedure via the TRA.

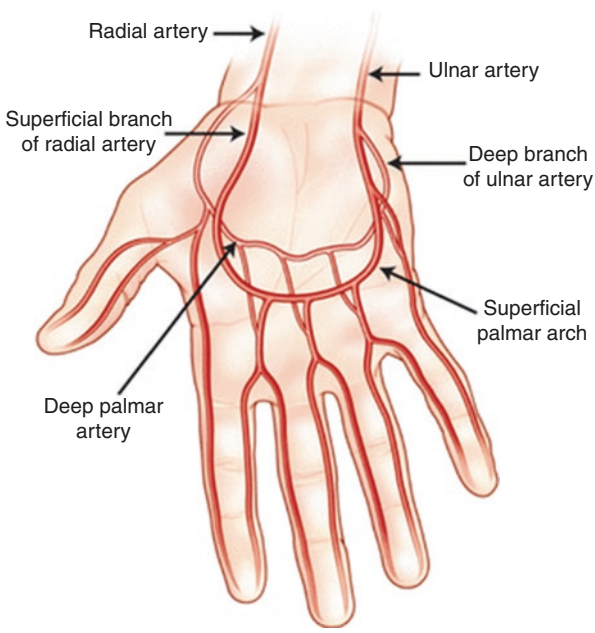

Fig. 2.3 Arterial supply of the hand

Patient Screening and Selection

- TRA is preferred in patients who are at high risk for femoral vascular access complications. These patients include:
  - Morbid obesity (>125 kg).
  - Severe lower extremity peripheral vascular disease.
  - Abdominal aortic aneurysm with thrombus.
  - Anticoagulated patients.
  - Patients who cannot lie flat.
  - Patients with bleeding diathesis.

Pearl

- Left TRA is preferred in patients of short stature, older age, with a high probability of tortuosity in the right subclavian artery, and those requiring LIMA angiography.

Contraindications for Radial Access

- Non-palpable radial artery pulse.
- INR > 2.5 for elective procedures.
- Patients with existent AV fistula for dialysis or those at risk for starting dialysis [10].
- Severe vaso-occlusive disease (i.e. Raynaud disease, Takayasu arteritis, thromboangiitis obliterans) [10].
- Documented small radial artery size or known complex radial/brachiocephalic anatomy [10].

Preprocedural Testing

- Adequate collateral circulation to the hand can be assessed by performing Allen's test or the Barbeau's test (more objective) [2].
  - Neither test has been shown to predict clinically significant periprocedural complications and performing these tests is not mandatory.
- The most reliable method to assess collateral circulation is by using Doppler ultrasonography [2].
  - Allows for evaluation of the blood flow in the arteries and collaterals.
  - Allows for a better understanding of vascular anatomy.

Allen's Test

- The patient is asked to make a fist.
- The operator simultaneously compresses the radial and ulnar arteries, occluding both arteries (Fig. 2.4).

- The patient opens and closes their hand five times. The final opened palm should appear blanched.
- Pressure on the ulnar artery is released while maintaining occlusive pressure on the radial artery. The hand is observed for color changes.
  - Return of the hand color to pink within 8–10 s is a "positive" Allen's test and suggests that the ulnar blood supply in that hand will be sufficient if the radial artery is occluded (Fig. 2.5).
  - If the release of the ulnar artery occlusive pressure does not result in a return of pink hand color within 8–10 s then it is a 'negative' Allen's test. This suggests that the ulnar blood supply to the hand will be insufficient if the radial artery is occluded. TRA on that hand should be avoided, and alternative access should be obtained.

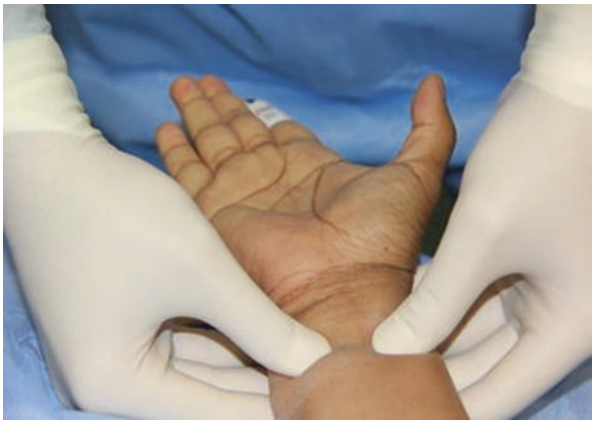

Fig. 2.4 Allen's test, compress the radial and ulnar arteries simultaneously

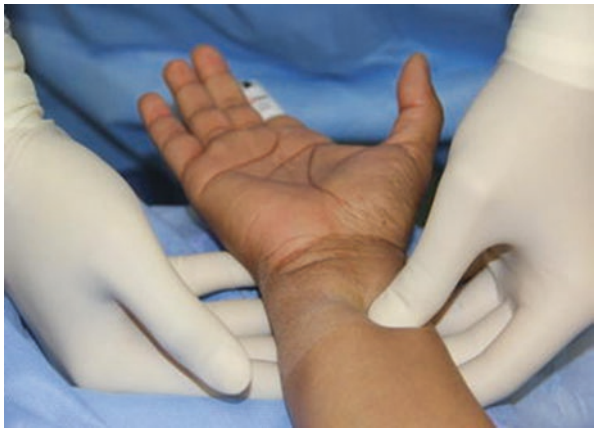

Fig. 2.5 Release the ulnar artery

Barbeau's Test (Allen's Oximetry Test)

- Attach a pulse oximeter to the access site hand and observe the pulse waveform on plethysmography.
- Apply occlusive pressure to both the radial and ulnar arteries simultaneously so the waveform on plethysmography is absent.
- Release pressure from the ulnar artery while maintaining occlusive pressure on the radial artery, monitor the pulse waveform on plethysmography, and compare findings with baseline pulse waveform on plethysmography (Fig. 2.6).
- Changes of the tracing are classified by the Barbeau classification into four categories (Fig. 2.7).

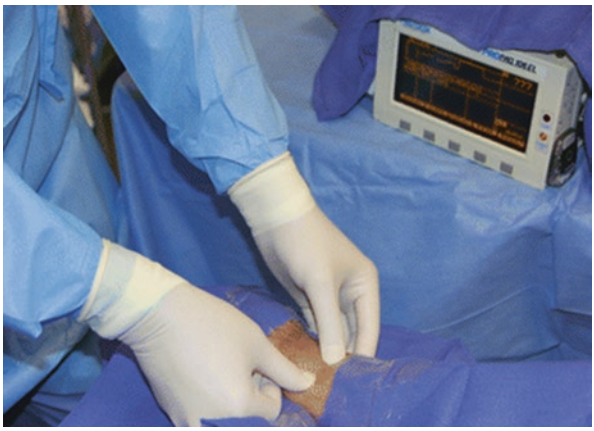

Fig. 2.6 Compress the radial artery and ulnar artery to occlude both vessels until the waveform on plethysmography is absent, release the ulnar artery, and observe the pulse waveform of the ulnar artery

Type	Precompression	Radial artery compression			
		Start		After 2 min	
			Oximetry		Oximetry
A			+		+
B			+		+
C			–		+
D			–		–

Fig. 2.7 Barbeau test

  - Type A: no change in pulse wave.
  - Type B: damped but distinct pulse waveform.
  - Type C: loss of phasic pulse waveform, followed by recovery in 2 min.
  - Type D: no recovery of pulse tracing within 2 min.

- Interpretation of the Barbeau's test findings.
  - Normal test:
    Return of a waveform (Type A waveform).
  - Abnormal test if any of the following:
    Oximetry readings are different after the release of ulnar artery occlusive pressure (Type B or C waveform).
    Continued absence of a waveform (Type D waveform) (Do not cannulate the radial artery if a type D waveform is present).

Prepping the Arm

- The arm is immobilized on the radial arm board with the palm facing upward and obliquely.
- The wrist is hyperextended with a wrist brace or towels. In this position, the radial artery is more superficial, making it easier to palpate (Fig. 2.8a).
- A pulse oximeter is placed on the index finger for continuous monitoring during the procedure.
- Sterilizing solution is applied to the area from the flexor crease to the mid-forearm. Also, prepare and sterilize the right and left groin because of the possibility of cross-over from the TRA, or the need for mechanical support.
- Drape the arm and hand so only the area from the styloid process of the radius to approximately 5 cm proximal is exposed.

Radial Artery Puncture and Sheath Insertion

- Pre-procedure planning is crucial for TRA success. It helps avoid multiple arterial punctures, reduces the risk of radial artery spasm, and vascular complications.
- Ultrasound can be used to visually identify radial artery location, depth, course, and patency.
- Palpate the radial artery, stabilize it with the tip of your finger, and apply local skin anesthesia with 0.5–1 ml 1% lidocaine.
- The radial artery puncture site should be 2 cm proximal to the styloid process (Fig. 2.8b).
- Use one of two techniques to gain successful radial artery access.
  - *Seldinger technique with back wall puncture (Double-wall 'Through-and-Through' approach).*

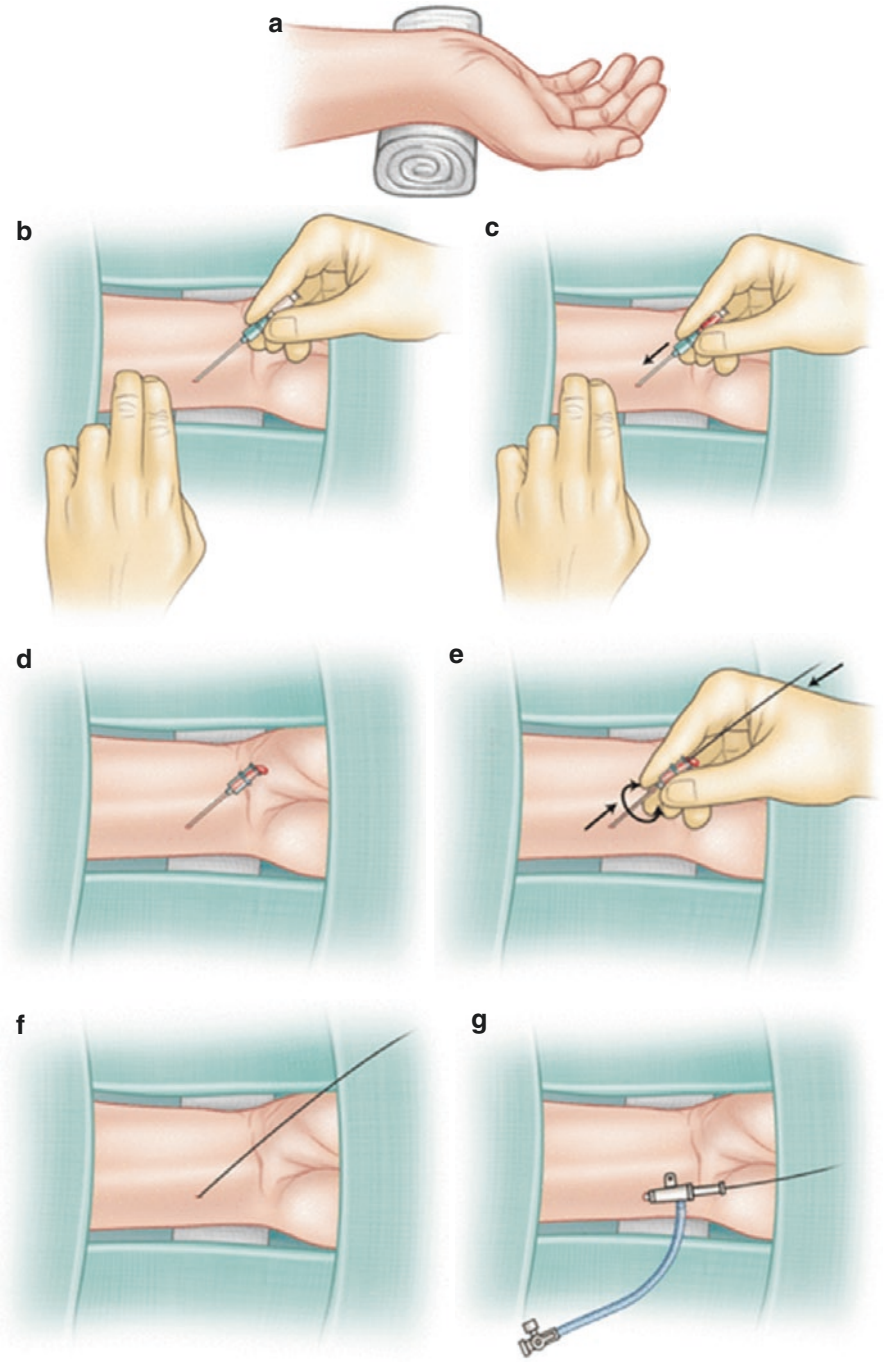

Fig. 2.8 Steps of radial access. (**a**) Hyper-extend the wrist. (**b**) Puncture 2 cm above the styloid process. (**c**) Pulsatile blood flow is seen. (**d**) Advance needle a few millimeters. (**e**) Thread the 0.018" guidewire through the needle. (**f**) Remove the needle. (**g**) Insert the Terumo sheath

When blood appears in the hub of the IV catheter (Fig. 2.8c), the needle and catheter system is advanced a few millimeters through the back wall of the artery to transfix the artery.
Subsequently, the needle is withdrawn leaving the catheter in place (Fig. 2.8d).
The catheter is gently drawn back until pulsatile backflow appears.
Once pulsatile blood flow is observed, advance a 0.018″ nitinol floppy guidewire (30–50 cm in length, with a floppy tip and more rigid shaft) through the cannula of the catheter (Fig. 2.8e).
Once the wire is passed to the desired level through the cannula, the catheter is removed leaving the wire in place (Fig. 2.8f).

- *Single-wall anterior puncture technique.*

  This approach uses a 21-gauge micropuncture needle that is 2–5 cm in length.
  You may or may not feel a tactile "pop" as the needle passes through the anterior wall of the radial artery.
  When the needle is in the lumen, you will have immediate brisk blood flow, which may not necessarily be pulsatile.
  Once you have steady blood flow insert a 0.018″ nitinol floppy guidewire, remove the needle, and leave the guidewire in place.

- Advance a 10 cm hydrophilic sheath over the 0.018″ nitinol floppy guidewire (Fig. 2.8g).
  - Sheath size is selected based on the minimum inner diameter needed to accommodate the equipment to be used for the procedure.
  - To reduce radial artery occlusion, try to maintain a radial artery inner diameter to sheath outer diameter ratio of <1.10.
  - For most patients, a 6 Fr sheath is generally safe to use and preferred (should intervention be required, the sheath does not necessarily need to be exchanged).
- Remove the guidewire and dilator, and leave the sheath within the artery.
- Next, a cocktail consisting of an antithrombotic agent (unfractionated heparin, enoxaparin, or bivalirudin), and one or more vasodilators (nitroglycerin, verapamil, and nicardipine) is prepared in a single 50 cc syringe and given through the sheath sidearm to prevent thrombosis and vasospasm.
  - When giving the cocktail aspirate 20–30 cc of blood to dilute the mixture before injection and deliver the cocktail slowly over 30–60 s, as this helps reduce patient discomfort.
- After delivery of the cocktail, flush the sheath with 10 cc of heparinized saline.
- Secure the sheath in place with a transparent adhesive dressing.

Antithrombotic Agents

- The recommended doses of antithrombotic agents are as follows [2]:

- Unfractionated heparin 50 IU/kg, up to 5000 IU total.
- Enoxaparin 60 mg.
- Bivalirudin 0.75 mg/kg bolus intravenously for diagnostic procedures, followed by infusion at 1.75 mg/kg/h if PCI is indicated.

Vasodilators Agents

- The recommended doses of vasodilator agents are as follows [1]:
  - Verapamil 2.5 mg; Verapamil is to be used cautiously (or avoided entirely) if the patient has severely reduced systolic left ventricular function or bradycardia.
  - Nitroglycerin 200μg; Nitroglycerin is to be avoided in hypotensive patients or those with severe aortic stenosis.

Pearls

- When using the Seldinger technique with back wall puncture, ascertaining pulsatile flow from the IV catheter is essential before proceeding. Pulsatile flow may not be seen when using a micropuncture needle.
- Quickly recognize problems by tactile feedback from a floppy tipped guidewire. If you feel resistance while advancing the 0.018″ nitinol floppy guidewire, stop, and perform the following steps to delineate the level and type of impedance encountered [12].
  - Fix the guidewire and remove the IV catheter.
  - Partially insert a 5 Fr or 6 Fr sheath dilator into the artery over the guidewire to a distance of 5–10 cm and remove the guidewire.
  - Perform angiography using 3–5 cc of contrast.

Radial Complications

Radial Artery Spasm

- Most common reason for TRA failure (~40% of cases) [13].
- Risk factors predisposing for spasm:
  - Younger age.
  - Women.
  - Lower body weight.
  - Small radial artery diameter.
  - Large sheath size.
  - Multiple access attempts.
  - Number of catheters used.
  - Procedure duration.

- Suspect spasm if the patient reports pain in the arm or if there is resistance to catheter advancement.
- Angiographically the vessel will appear narrowed with smooth arterial contours.
- Differential diagnosis must include inadvertent recurrent radial artery access, anomalous radial origin (high origin) and course, medial calcific sclerosis (Mönckeberg disease).
- Prevention and Treatment [12]:
  - Gaining radial access on the first attempt is imperative because the vessel is prone to spasm with repeated attempts.
  - Medications:
    Give additional sedation.
    Direct administration of vasodilatory/antispasmodic agents through the sidearm of the introducing sheath (nitroglycerin, verapamil, diltiazem, adenosine, nitroprusside, or nitric oxide).
  - Repeat angiography after several minutes to assess if the spasm has resolved

    If spasm resolves, use a 0.035″ wire to traverse the area of spasm. If the wire passes, then load and advance the catheter over the wire.
    If spasm does not resolve, consider using alternative access.

Radial Artery Occlusion

- Most common complication following TRA (typically subclinical).
- Occurs usually shortly after the procedure (~1–10% of cases) [2]:
- Concerning because [2]:
  - Increased risk of developing hand ischemia.
  - Unable to use a TRA for future procedures.
  - Radial artery is no longer a suitable conduit for:
    Coronary artery bypass grafting.
    Arteriovenous fistula formation for patients requiring dialysis access.
    Intra-arterial pressure monitoring.
- Risk factors predisposing for RAO [2]:
  - Older age.
  - Women.
  - Lower body weight.
  - Elevated creatinine.
  - Peripheral artery disease.
  - Diabetes.
  - Smoking.
- Procedural factors associated with RAO [2]:
  - Sheath size relative to the radial artery diameter.
    Larger sheaths increase the risk of vascular trauma and create a prothrombotic state.
    It is important to maintain a sheath-to-artery diameter ratio < 1.10.
  - Inadequate anticoagulation.

  - Inadequate patent hemostasis.
- Majority of patients are asymptomatic because of dual blood supply and extensive collateral circulation involving the hand.
- Suspect RAO if the radial pulse is absent.
  - However, the presence of a radial pulse does not exclude the diagnosis of RAO because there may be collateral blood supply to the area around the RAO.
- Patency is best assessed clinically using the reverse Barbeau's test or Doppler US [2].
  - Reverse Barbeau's test: pulse oximeter is placed on the thumb or 1st digit of the ipsilateral hand, and then the ulnar artery is occluded. If the plethysmography waveform is absent, this highly suggests RAO.
  - Doppler US: allows for direct imaging of the artery and assessment of blood flow within the vessel. If visible obstruction or absent blood flow, it is diagnostic of RAO.
- Prevention and treatment [2]:
  - Spontaneous recanalization occurs within 1–3 months in 50% of patients.
  - Smaller and appropriately sized sheaths should be used, and upgraded to a larger sheath when required.
  - Consider using sheath-less guide catheters. These catheters reduce the outer diameter of the vascular access system by 1–2 Fr compared with conventional sheaths and catheters.
  - Administer adequate anticoagulation as part of the initial cocktail.
  - Consider administration of unfractionated heparin 50 IU/kg, up to 5000 IU total at the end of the case [14].
  - Initial treatment is usually conservative:

    Begin with compression of the ipsilateral ulnar artery for an extended period (up to 60 min).

    If the above method fails, most cases of RAO can be managed with enoxaparin or fondaparinux for 4 weeks duration.

    If this fails, percutaneous recanalization can be considered.

    In rare cases, acute treatment and emergent vascular surgery consultation may be required (i.e. acute ischemia of the hand).

Access Site Hematoma

- Generally results from improper hemostatic device application or device failure.
- Usually, it is easily managed with compression of the radial artery by placing a vascular band in the correct position.
  - The radial artery should be compressed proximal and distal to the puncture site to control antegrade and retrograde flow.

Forearm Hematoma

- Occurs in <0.3% of cases [13].

- Early recognition of this complication is of critical importance because bleeding may already be significant before it even manifests with forearm swelling.
- Management and treatment [13]:
  - Crucial to rapidly assess and treat this complication.
  - If not controlled and managed appropriately, a trivial forearm hematoma can develop into a serious compartment syndrome.
  - Immediately palpate the forearm, compare findings of softness and size with the opposite arm.
  - Apply hemostatic compression along the length of the access artery to prevent further blood extravasation.

    Application of an Ace bandage to the forearm.

    Application of an Ace bandage with gauze balls placed along the course of the artery. Tightening the ace bandage over the gauze balls selectively compresses the artery.

    Use a sphygmomanometer cuff to compress the brachial artery.
    - Inflate the cuff to a pressure > 10–20 mmHg more than systolic blood pressure, and then intermittently deflate it every 2–3 min for 10–15 s. Repeat this cycle until adequate hemostasis is achieved.

    Sealing of the perforation with a long sheath (rarely necessary).
    - If a perforation occurs before angioplasty, one can continue to use a guiding catheter and complete the procedure, by which time the perforation usually seals off.
    - Sealing of the perforation with a covered stent (rarely necessary)
- If the patient develops pain, pallor, paresthesia, paralysis, or absent pulse suspect compartment syndrome.
  - Direct measurement of the compartment pressure is a useful confirmatory tool and helps guide treatment strategy (conservative vs. urgent surgical fasciotomy).

Pseudoaneurysm Formation

- Rarely occurs with TRA.
- Antecedent oral anticoagulation is the biggest risk factor.
- Usually managed with prolonged compression for 10–20 min.
- If pseudoaneurysm fails to occlude with compression, surgery may be required.
  - This can be performed using local anesthesia, and done as an outpatient.

Radial Artery Tortuosity or Loop

- Tortuosity occurs in <10% of cases [12].
- Loops occur in <1% of cases [12].
- Treatment:
  - Perform angiography to help define anatomy if there is difficulty advancing a 0.035″ guidewire.

Position the access sheath appropriately.
Perform arteriogram using 3–5 cc of contrast.

- Use an angle-tip hydrophilic-coated wire with catheter support, and advance it using fluoroscopy.
  Hydrophilic wires allow for smooth, rapid movement through a tortuous segment of a vessel. However, they are highly prone to navigate into small side branch vessels and should be advanced using fluoroscopy.
- If the above technique fails to navigate the loop, use a 0.014″ coronary wire.
  Occasionally, you might need to use two coronary wires to assist with the tracking of the catheter.
- If the above technique fails to navigate the loop, use a 2.0–2.5/12 mm balloon to perform balloon-assisted tracking of the catheter.
  Remember to position the balloon so half of its length protrudes outside the tip of the catheter and insufflate to low pressure (4 atm).
- If the catheter navigates the loop, exchange the wire to a stiff angle glidewire and reduce the loop with traction and counterclockwise rotation of the catheter and wire.

Pearl

- Patients should be well sedated before performing the procedure.
- If the catheter fails to advance despite the techniques described above, obtain alternative access.

Radial Artery Stenosis

- Very rare.
- If the stenosis is focal and equipment can easily traverse the lesion, then it is reasonable to continue with the procedure, otherwise, alternative access should be pursued [12].

Other Rare Complications

- List of other potential transradial access site complications [2, 13]:
  - Radial arteriovenous fistula formation.
  - Radial artery eversion during sheath removal.
  - Hand ischemia.
  - Compartment syndrome.
  - Radial artery avulsion due to intense spasm.
  - Sterile abscess formation at the radial artery access site.
  - Persistent post-procedural pain.
  - Upper extremity loss of strength.

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Coronary Anatomy and Angiography

3

Gurpreet S. Johal, Sunny Goel, and Annapoorna Kini

Introduction

The purpose of coronary angiography is to accurately define coronary anatomy, and obtain optimal angiographic images of the main epicardial coronary arteries and their branches. It is important to limit radiation exposure to the patient and medical personnel while trying to obtain the essential number of images to define the anatomy, and minimize contrast use. Information obtained from coronary angiography is significant because it assists in the diagnosis and therapeutic intervention of coronary artery disease. Inadequate knowledge of coronary anatomy and angiographic views can lead to serious complications, inappropriate interventions, and wasteful healthcare expenditures.

Coronary Anatomy and Segment Classification

Having a detailed understanding of coronary anatomy is fundamental to perform coronary angiography safely and efficiently.

- The right and left coronary arteries originate from their respective aortic sinuses, which are normally located in the upper third of the Sinus of Valsalva. The left coronary artery (LCA) orifice is superior and posterior relative to the right coronary artery (RCA) orifice [1].
- The LCA begins as the left main coronary artery (LMCA), and bifurcates into the left anterior descending artery (LAD) and the left circumflex artery (LCx) (85–90% of cases). Occasionally, the LCA trifurcates giving rise to an additional artery called the ramus intermedius (10–15% of cases) [1].

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- The LAD gives rise to septal perforators and diagonal branches. The LCx gives rise to obtuse marginal branches, atrial branches, and in a left dominant system the posterior descending artery (PDA) and posterolateral branches (PLB). The PDA also gives rise to septal perforators [1].
- The RCA begins and the first branch it gives rise to is the conus artery (50% cases it arises from a separate ostium). The RCA also gives rise to the sinoatrial node artery (40–50% of the cases it arises from the LCx), atrioventricular nodal artery, several smaller marginal branches, and a more prominent acute marginal branch. The RCA usually gives rise to the PLB and continues as the PDA at the crux of the heart [1].
- Left or right coronary dominance is based on the artery from which the PDA arises (Fig. 3.1). Dominance is usually right in 80% of individuals and left in 20% of individuals (left dominance is slightly more frequent in females). Codominance is no longer used [1].
- We use the sixteen-segment–based coronary segment classification system from the SYNTAX (Synergy between PCI with Taxus and Cardiac Surgery) score with slight modification to define the individual coronary segments (Table 3.1) [2].

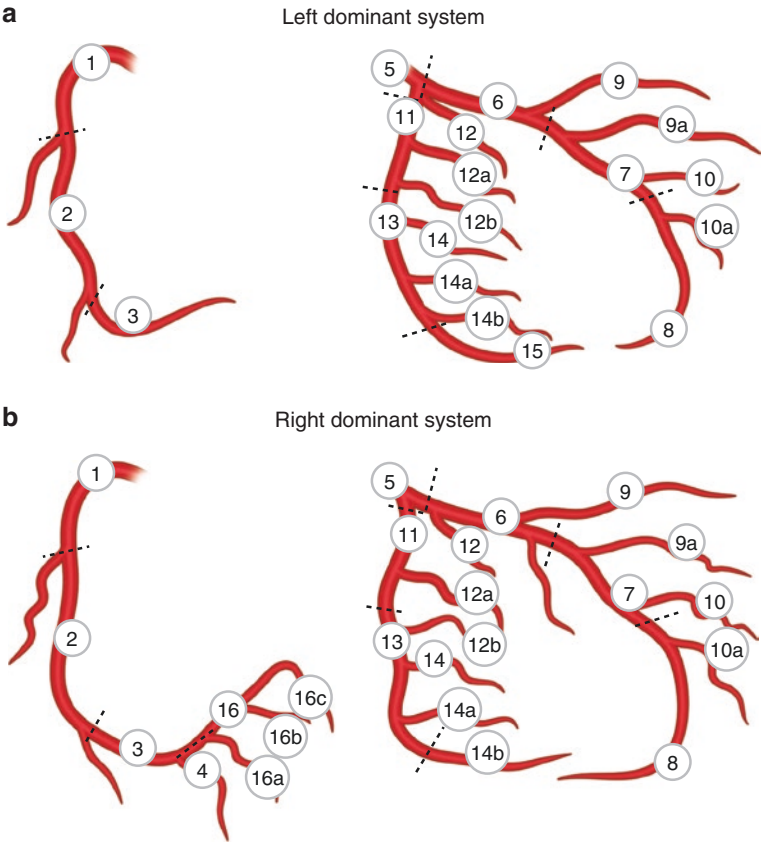


Fig. 3.1 (**a**) Left dominant system segment anatomy. (**b**) Right dominant system segment anatomy. (Adapted from Sianos et al. [2])

Table 3.1 Definition of the coronary tree segments

Segment	Name	Segment definition
1	RCA proximal	From ostium to and including the origin of the first RV branch.
2	RCA mid	RCA immediately distal to the origin of the first RV branch to the acute margin of the heart.
3	RCA distal	From the acute margin of the heart to the origin of the posterior descending artery.
4	Right posterior descending	Originating from the distal coronary artery distal to the crux and running in the posterior interventricular groove.
16	Atrioventrcular Continuation from RCA	Originating from the distal coronary artery distal to the crux and running in the atrioventricular groove.
16a	Posterolateral from RCA	First posterolateral branch from segment 16.
16b	Posterolateral from RCA	Second posterolateral branch from segment 16.
16c	Posterolateral from RCA	Third posterolateral branch from segment 16.
5	Left main	From the ostium of the LCA through bifurcation into left anterior descending and left circumflex branches.
6	LAD proximal	Proximal to and including the first major septal branch.
7	LAD mid	LAD immediately distal to the origin of the first septal branch and extending to the point where LAD forms an angle (RAO view). If this angle is not identifiable, this segment ends at one-half the distance from the first septal to the apex of the heart, usually after two diagonal branches have originated.
8	LAD distal	Terminal portion of LAD, beginning at the end of the mid segment and extending to or beyond the apex.
9	First diagonal	The first diagonal originating from segment 6 or 7.
9a	First diagonal a	Additional first diagonal originating from segment 6 or 7, before segment 8.
10	Second diagonal	Second diagonal originating from segment 8 or the transition between segments 7 and 8.
10a	Second diagonal a	Additional second diagonal originating from segment 8.
11	Proximal circumflex	Main stem of circumflex from its origin of left main to and including the origin of the first obtuse marginal branch.
12	Ramus intermedius	Branch from trifurcating left main other than proximal LAD or LCX. Belongs to the circumflex territory.
12a	Obtuse marginal a	First side branch of circumflex running in general to the area of the obtuse margin of the heart (down and out in RAO view)
12b	Obtuse marginal b	Second additional branch of circumflex running in the same direction as 12.
13	Distal circumflex	The stem of the circumflex distal to the origin of the most distal obtuse marginal branch and running along the posterior left atrioventricular grooves. Caliber may be small or artery absent.
14	Left posterolateral	Running to the posterolateral surface of the left ventricle (horizontal and down in RAO view). May be absent or a division of obtuse marginal branch.
14a	Left posterolateral a	Distal from 14 and running in the same direction.
14b	Left posterolateral b	Distal from 14, and 14a running in the same direction.
15	Left posterior descending	The most distal part of the dominant left circumflex when present. Gives origin to septal branches. When this artery is present, segment 4 is usually absent

Adapted from Sianos et al. [2]

Imaging Basics

When performing coronary angiography, it is necessary to obtain multiple views in different orthogonal planes to clearly define all vessel segments [3].

Image Orientation

- The orientation of the angiographic image is defined by the position of the imaging detector relative to the patient (not the X-ray tube). It is described using two angles from the center of the patient (Fig. 3.2) [4].
  - The first angle refers to 'rotation'. It describes the position of the image intensifier along the transverse (axial) plane of the patient. It is referred to as degrees right anterior oblique (RAO) (to the patient's right) or left anterior oblique (LAO) (to the patient's left).
  - The second angle refers to 'angulation'. It describes the position of the image intensifier along the sagittal plane of the patient. It is referred to as degrees cranial (towards the head of the patient) or caudal (towards the feet of the patient).
- All views are reported by convention with left or right rotation first, followed by the cranial or caudal angulation [3].

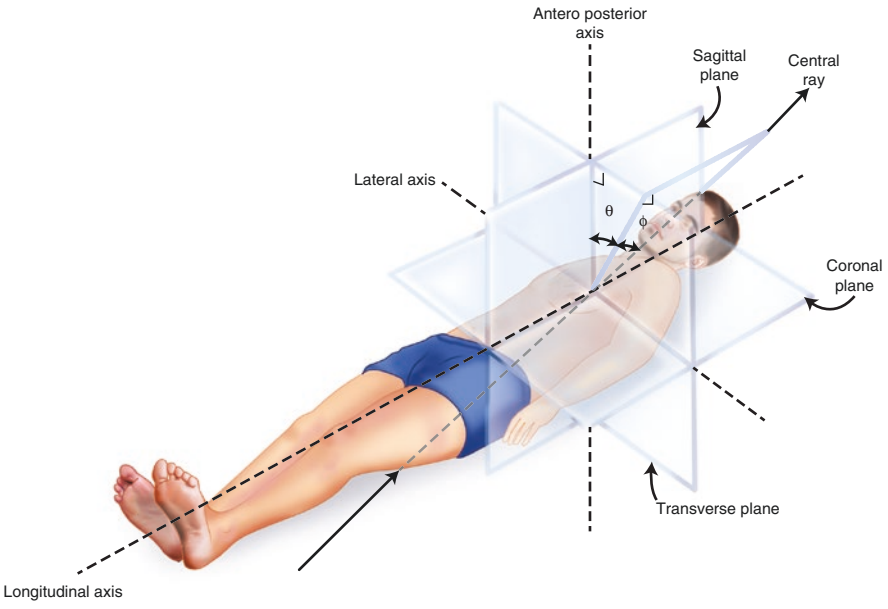


Fig. 3.2 Nomenclature of axes and planes relative to the patient. (Adapted from Aldridge et al. [4])

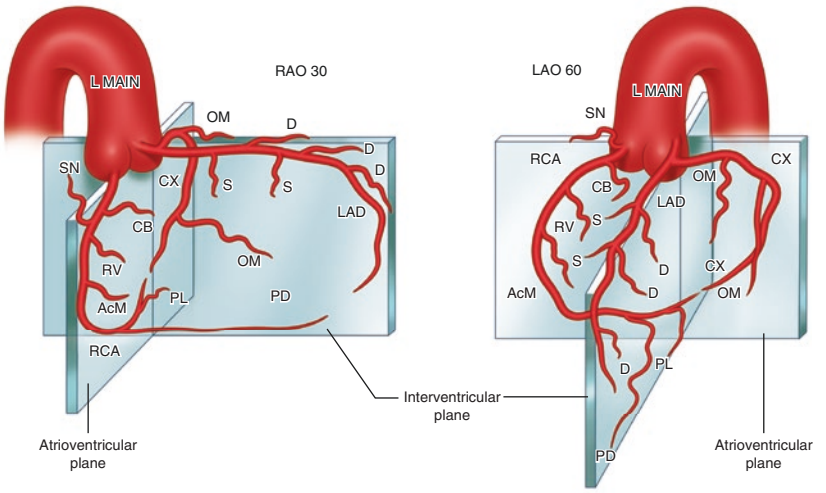


Fig. 3.3 Representation of coronary anatomy in relation to the interventricular and atrioventricular valve planes as seen in two views: right anterior oblique (RAO) and left anterior oblique (LAO). Coronary branches are as follows: AcM, acute marginal; CB, conus branch; CX, circumflex; D, diagonal; L Main, left main; LAD, left anterior descending; OM, obtuse marginal; PD, posterior descending; PL, posterolateral left ventricular; RCA, right coronary; RV, right ventricular; S, septal; SN, sinus node. (Adapted from *Grossman's cardiac catheterization, angiography, and intervention*. 6th edition [3])

- When the image intensifier is vertically above the patent without any rotation or angulation it is referred to as antero-posterior (AP).
- When the image intensifier is angled at 90° and parallel to the floor it is referred to as either the left or the right lateral projection, depending on the position of the image intensifier relative to the patient.

Image Projections

- All major coronary arteries lie in one of two planes, the interventricular septum or the AV groove (Fig. 3.3). The recommended image projections are intended to display the coronary artery and anatomy along these planes [3].

Our Protocol for Coronary Angiography

In most cases, for a right dominant system, the LCA can be adequately visualized using three views *(LAO Caudal, RAO Caudal, and RAO Cranial)*, and the RCA in two views *(LAO and LAO Cranial)*.

For a left dominant system, the LCA can be adequately visualized using four views *(LAO Caudal, RAO Caudal, RAO Cranial, and LAO Cranial)*, and the RCA in one view *(LAO)*.

Further specifics regarding the usual degree of angulation are detailed below. The degrees of rotation and angulation are general guidelines, and an operator must always take into account patient-specific details such as body habitus, patient rotation, coronary anatomy, and heart orientation.

Left Coronary System

- The following views help optimize specific segments of the LCA. However, no single view is exclusive and the vessel should be seen in its entirety. The following degrees of rotation and angulation are suggestions and need to be optimized for each patient on a case-by-case basis.
  - *LAO Caudal [40°, 40°]:*

    LM
    Proximal LAD.
    Proximal LCx and OM's
  - *LAO Cranial [30°, 30°]:*

    Mid and distal LAD.
    Separates the septal from the diagonal branches.
    Distal LCx and LPDA in a left dominant system.
  - *AP Caudal [0°, 30°]:*

    Distal LM bifurcation (or short LM).
    Proximal LAD.
    Proximal to mid-LCx and OM's.
  - *AP Cranial [0°, 30°]:*

    Proximal and mid LAD.
  - *RAO Caudal [25°, 25°]:*

    LM bifurcation.
    Proximal LAD.
    Proximal to mid-LCx.
  - *RAO Cranial [30°, 40°]:*

    Mid and distal LAD.
    Distal LCx, LPL branches, and LPDA (if a left dominant system).

Right Coronary System

- The following views help optimize specific segments of the RCA. However, no single view is exclusive and the vessel should be seen in its entirety. The following degrees of rotation and angulation are suggestions and need to be optimized for each patient on a case-by-case basis.
  - *LAO [30°, 0°]:*

    Ostial and proximal RCA.
  - *AP Cranial [0°, 30°]:*

    Distal RCA bifurcation, RPDA and RPL branches.
  - *RAO [30°, 0°].*

    Mid-RCA and mid RPDA.

Defining Specific Lesions

- We use the following views to better visualize specific lesions:
- Native Vessel
  - *LM*:

    Ostial: RAO cranial, AP cranial, LAO cranial.
    Body: RAO caudal, AP caudal, LAO caudal.
  - *LAD*:

    Ostial/proximal: LAO caudal, LAO cranial.
    Mid/distal: AP cranial, RAO cranial.
  - *Diagonals*:

    Origin: LAO cranial, LAO caudal.
    Mid/distal: AP cranial, RAO cranial.
  - *Septals*:

    RAO cranial, AP cranial.
  - *Ramus*:

    Ostial/proximal: spider, AP caudal.
    Distal: AP caudal.
  - *Circumflex*:

    Ostial/proximal: spider, AP caudal.
    Mid/distal: AP cranial, RAO cranial.

- *OMs*:

  Ostial/proximal: RAO caudal.
  Mid/distal: AP cranial, RAO cranial.
  LPDA: LAO cranial, AP cranial.

- *RCA:*

  Ostial/proximal: LAO caudal.
  Mid: RAO 30°.
  Distal: AP cranial/RAO cranial/LAO cranial.

- *RPDA/RPL Branches:*

  AP cranial/RAO cranial.
  Early bifurcating RPDA: RAO 30°.

- Grafts
  - *LIMA:*

    Ostium/body: AP straight [0°, 0°].
    Anastomosis to LAD: RAO 30°, or AP cranial.
    LAD after anastomosis: AP cranial.

  - *RIMA:*

    Ostium/body: AP straight [0°, 0°].
    Anastomosis and native vessel after anastomosis: depends on vessel bypassed (see above).

  - *SVG:*

    Ostium/body: LAO 30°, or RAO 30°.
    Anastomosis to RCA/PDA/PL: LAO cranial, RAO, AP cranial.
    Anastomosis to LCX/OM/Ramus: RAO caudal, LAO caudal.
    Anastomosis to LAD/Diagonal: AP cranial, RAO cranial, LAO cranial, AP, lateral (the lateral view is especially useful to visualize anastomosis to LAD).
    Anastomosis and native vessel after anastomosis: depends on the vessel bypassed (see above).

- Collaterals
  - *Left to right:*

    LAO cranial.
  - *Right to left*:

    RAO straight.

Left Ventricle Angiography

Angiography of the left ventricle (LV) helps with the evaluation of LV function, segmental wall motion, mitral regurgitation, ventricular septal defects, hypertrophic cardiomyopathy, and intraventricular thrombus.

Setup

- It is best performed using a pigtail catheter with the catheter placed in the mid LV cavity, and not entangled with the mitral valvular apparatus.
- Power injector setup:
  - Extreme care must be taken to assure no air is within the system (prevent air embolism).
  - Synchronize the timing of the injection with the R wave (prevent R on T phenomenon).
  - Power injector settings: 40 cc of contrast, 15 cc/s flow, 650 PSI, 0.2 s rise.

    If the LV cavity is large or high cardiac output, then you can increase the volume and rate.
    If the LV cavity is small or there is LV outflow obstruction/aortic stenosis, then you should decrease the volume and rate.
- When injecting, the catheter should be held close to the sheath to avoid migration, ectopy, or catheter blow-back into the aorta.
- Panning of the table may be required (usually pushing the table away from the operator—standing on the patient's right) to visualize regurgitation into the left atrium (particularly if the atrium is dilated).
- In patients with markedly elevated LVEDP (>20 mmHg), consider administrating intracavitary nitroglycerin (200 mcg) to reduce filling pressures, and repeat LV angiography (if there are no contraindications and clinically appropriate).

LV Imaging

- *RAO 30°:*
  - Anterobasal, anterolateral, apical, inferior, and posterobasal wall segments (Fig. 3.4).
  - Mitral regurgitation.
- *LAO 45–60°:*
  - Posterolateral, lateral, and septal wall segments (Fig. 3.4).
  - Ventricular septal defect.

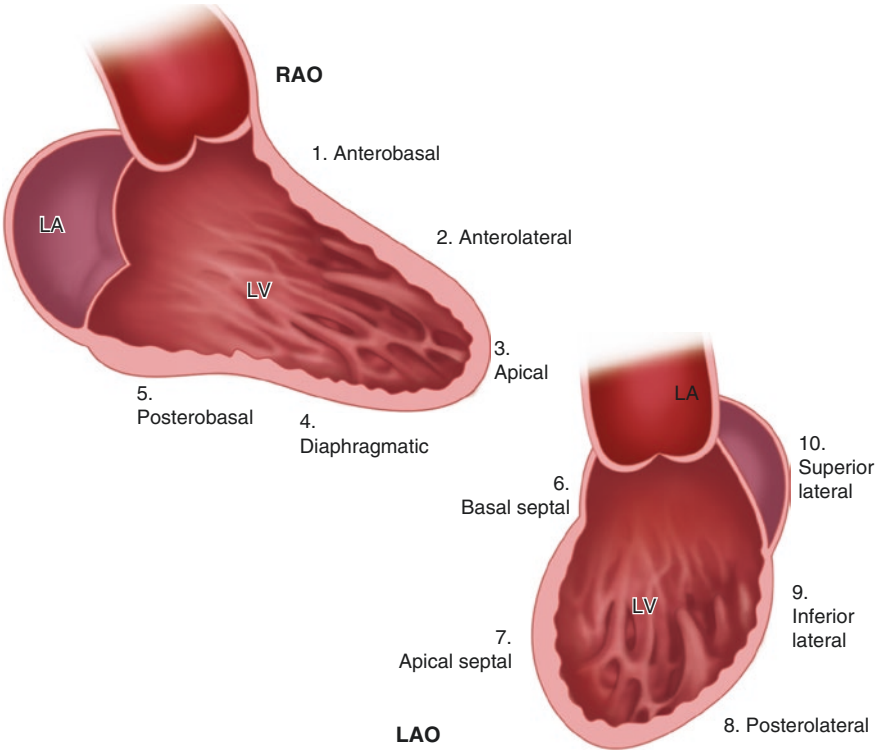


Fig. 3.4 Diagrammatic representation of RAO and LAO views of the LV obtained during contrast angiography showing division of the LV wall into 10 numbered segments. LA indicates left atrium; LAO = left anterior oblique; RAO = right anterior oblique. (Adapted from Hendel et al. [5])

Quantification of Aortic and Mitral Regurgitation (Table 3.2)

Table 3.2 Angiographic grades of aortic and mitral regurgitation

Grade	Aortic regurgitation	Mitral regurgitation
1+	Contrast refluxes from the aortic root into the left ventricle but clears on each beat, and does not outline the left ventricle	Contrast refluxes into the left atrium but clears on each beat, and does not outline the left atrium
2+	Contrast refluxes into the left ventricle with a gradually increasing density of contrast in the left ventricle that never equals contrast intensity in the aortic root, and outlines the left ventricle	Left atrial contrast density gradually increases but never equals left ventricle density, and outlines the left atrium
3+	Contrast refluxes into the left ventricle with a gradually increasing density; the left ventricle and aortic root density are equal after several beats	The density of contrast in the atrium and ventricle equalizes after several beats
4+	Contrast fills the left ventricle resulting in an equivalent radiographic density in the left ventricle and aortic root on the first beat, and persists for at least 3 beats	The left atrium becomes as dense as the left ventricle on the first beat, and contrast is seen refluxing into the pulmonary veins

Adapted from Apostolakis et al. [6]

Angiography Interpretation

A systematic interpretation of a coronary angiogram involves careful lesion quantification in at least 2–3 orthogonal views of the left coronary system, and 1–2 orthogonal views (depending on dominance) of the right coronary system.

In addition, to assessing coronary artery lesion stenosis, other characteristics of the vessel and lesion must be addressed (Table 3.3) [7].

Table 3.3 Angiographic description and parameters for lesion quantification

Coronary vessel size	**Diameter of vessel**
Small	< 2.0 mm
Moderate	2.0–3.0 mm
Large	> 3.0 mm
Eccentricity of lesion	**Lesions location description**
Concentric	Circumferential
Eccentric Type 1 Type 2	Lesion is located to one side of a vessel Smooth and broad neck Irregular surface and/or narrow neck
Complex	With multiple irregularities
Ostial lesion types	**Vessels involved (originate within 3 mm of an originating vessel)**
Aorto-ostial	LM, RCA, SVGs, free LIMA, free RIMA (also considered pedicle LIMA and RIMA even though they arise from subclavian arteries)
Non-aorto-ostial	LAD, LCx, RI
Branch ostial	Septals, Diagonals, OMs, PDA, AV continuation, RPLs
Calcification severity	**Calcification severity description**
None	No radiopacity
Mild	Faint radiopacities noted during cardiac cycles
Moderate	Dense radiopacities noted during the cardiac cycle before contrast injection
Severe	Dense radiopacities noted on both sides of the arterial wall "tram-track" without cardiac motion before contrast injection
Stenosis types	**Description of types of stenosis/length of stenosis**
Focal (discrete)	<10 mm
Tubular	10–20 mm
Diffuse (long)	>20 mm
Tandem	Two lesions close to one another with a normal segment in between but require two separate stents to cover
Sequential	Two lesions close to one another with a normal segment in between but can be covered by a single stent
Myocardial blush grade	**Myocardial blush grade description**
0	No or minimal blush
1	Stain present, blush persists on next injection
2	Dye strongly persistent at end of washout; gone by next injection
3	Normal ground glass appearance of blush; dye mildly persistent at end of washout

(continued)

Table 3.3 (continued)

TIMI grade	**TIMI grade description**
0	No anterograde flow beyond the point of occlusion (no flow).
1	Contrast passes the point of obstruction but hangs up and fails to opacify the entire distal coronary bed during the angiographic filming sequence (penetration without perfusion)
2	Contrast opacifies the entire coronary bed distal to the stenosis, but the rate of entry and/or clearance is slower than in comparable areas not perfused by the distal vessel (partial perfusion)
3	Complete perfusion. Specifically, the antegrade flow of contrast with complete filling of the artery and its major and minor branches within 3 full cardiac cycles. Contrast also clears from the arterial segment within 3 full cardiac cycles (complete perfusion)
Thrombus grade	**Thrombus grade description**
0	No thrombus
1	Possible thrombus (mural opacities)
2	Small thrombus (size <0.5 × normal lumen diameter)
3	Medium thrombus (size 0.5–2 × normal lumen diameter)
4	Large thrombus (size >2 × normal lumen diameter)
5	Recent thrombotic occlusion (fresh thrombus with dye stasis and delayed washout)
6	Chronic total occlusion (smooth, abrupt, and with no dye stasis and brisk flow)
Rentrop grade	**Rentrop classification for collateral grading**
0	No collaterals (absent)
1	Filling of side branches of a target-occluded epicardial coronary artery via collaterals without visualization of the epicardial coronary itself
2	Partial filling of the epicardial segment via collateral arteries
3	Complete filling of the epicardial segment via collateral arteries

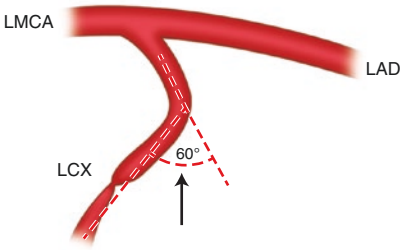
Degree of Tortuosity (Fig. 3.5)

Characteristic of ACC/AHA Type A, B, and C Lesions (Table 3.4)

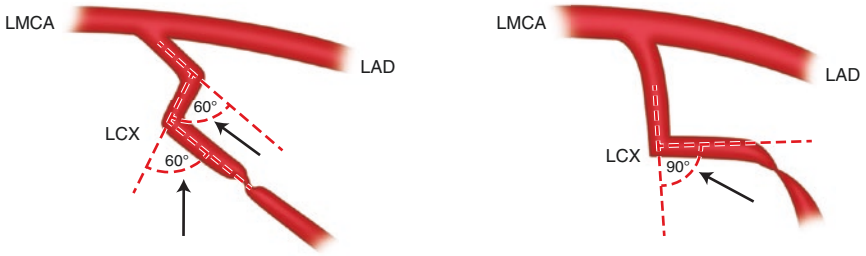
Bifurcation Lesions Classified Based on Medina Classification

- The most commonly used method to classify bifurcation lesions is the Medina Classification (Fig. 3.6) [9].

1. No tortuosity: no bend of 45–90% bend prior to lesion.



2. Moderate tortuosity: 1–2 bends of 45–90% prior to the lesion



3. Excessive or severe tortuosity: >3 bends of 45–90% prior to the lesion or 1 or more thane one 90° bend prior to the lesion

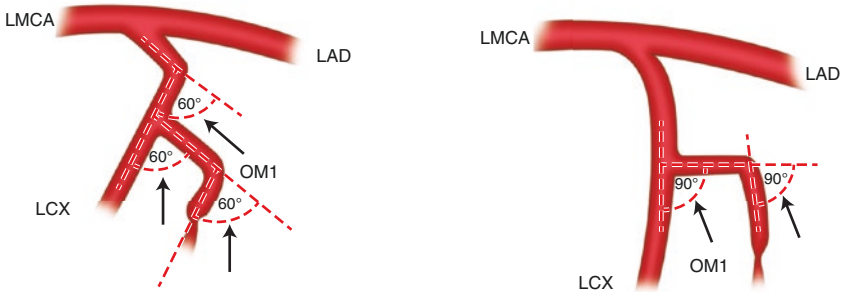


Fig. 3.5 Description of tortuosity based on severity. (Adapted from Kini et al. [7] with permission from Elsevier)

Table 3.4 ACC/AHA lesion classification

Type A	Type B	Type C
Discrete (length < 10 mm)	Tubular (length 10–20 mm)	Diffuse (length > 20 mm)
Concentric	Eccentric	–
Readily accessible	Moderate tortuosity of the proximal segment	Excessive tortuosity of the proximal segment
Non-angulated segment (<45°)	Moderately angulated (45–90°)	Extremely angulated (>90°)
Smooth contour	Irregular contour	–
Little or no calcification	Moderate or heavy calcification	Degenerated vein grafts with friable lesions
Absence of thrombus	Some thrombus present	Significant thrombus present
Non-ostial	Ostial location	–
No major side branch involved	Bifurcation lesion requiring double guidewires	Inability to protect major side branch
Less than totally occlusive	Total occlusion <3 m	Total occlusion >3 months

Adapted from Ryan et al. [8]

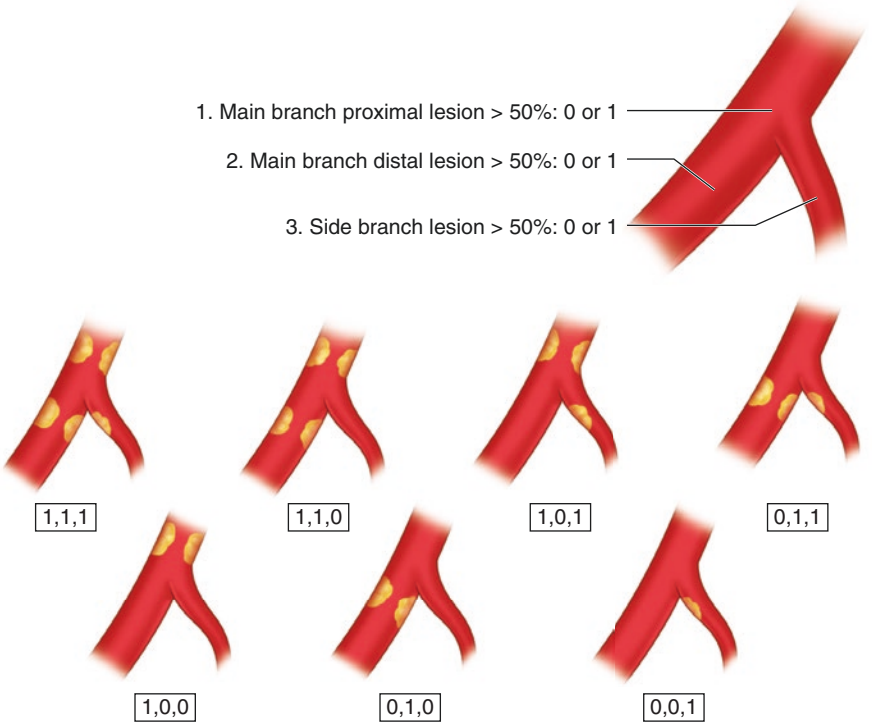


Fig. 3.6 Medina classification for coronary bifurcation lesions. (Adapted from Latib et al. [9] with permission from Elsevier)

- Medina Classification assesses plaque burden based on the presence (1) or absence (0) of stenosis in the proximal main branch (MB), distal MB, and side branch (SB).
- A bifurcation lesion is considered significant if it is >50% stenosed, the MB and SB are within 5 mm of the carina (point of bifurcation), and the SB is >2.5 mm in diameter.

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Physiological Assessment During Interventional Procedures

4

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Introduction

Visual coronary angiography stenosis severity poorly predicts the physiological significance of the stenosis. In patients with coronary artery disease, the presence of inducible myocardial ischemia is an important risk factor for an adverse clinical outcome and can be used to risk-stratify patients. As recommended by current guidelines for the treatment of coronary artery disease, the presence of myocardial ischemia should play a pivotal role in the decision-making process about coronary revascularization. There are hyperemic and non-hyperemic pressure indices to determine the physiological significance of the stenosis. In this chapter, we will discuss the hyperemic index—Fractional flow reserve [FFR]; one of the many but most commonly used non-hyperemic index—Instantaneous wave-free ratio [iFR] along with a brief description of non-invasive approach using CT-FFR.

Coronary Flow Reserve (CFR)

CFR is the term used to describe the ratio of myocardial blood flow (MBF) at stress conditions that can be supplied to the myocardium compared to the blood flow at resting conditions [1]. It is influenced not only by coronary stenosis but also by several other factors, including heart rate, coronary resistance (both epicardial and microvascular), coronary collateral circulation, and coronary vasodilatation (Fig. 4.1).

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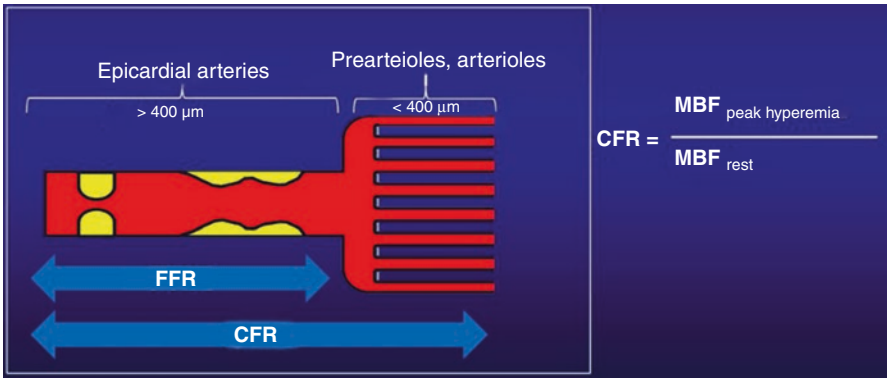


Fig. 4.1 Comparison of CFR and FFR

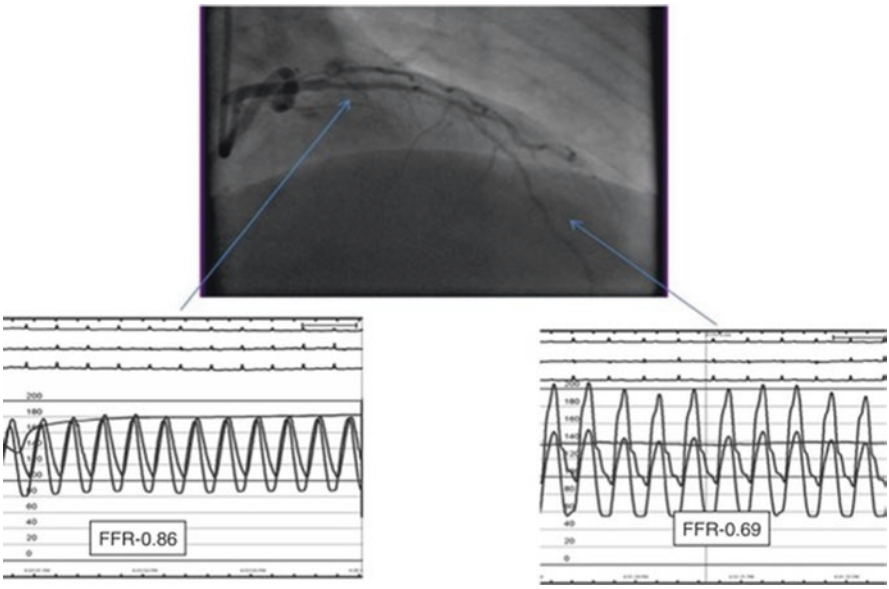


Fig. 4.2 Hemodynamic assessment of proximal and distal LAD lesion by FFR

Fractional Flow Reserve (FFR)

FFR is defined as the maximum achievable blood flow to a myocardial territory in the presence of a stenosis as a ratio to the normal maximum achievable blood flow to that same myocardial territory in the hypothetical situation the supplying vessel would be completely normal [2]. At maximum hyperemia, myocardial perfusion pressure and myocardial flow are linearly proportional, and a change in myocardial perfusion pressure results in a proportional change in myocardial flow. Therefore, the ratio of maximum stenotic and normal maximum flow can be expressed as the ratio of distal coronary pressure (Pd) and proximal aortic pressure (Pa) at maximal hyperemia (Figs. 4.1 and 4.2).

Special Characteristics of FFR

- Independent of HR/BP/LV function.
- Accounts for the contribution from collaterals.
- Relates the severity of stenosis to the myocardial mass subtended by the vessel.
- Easy to perform and interpret.
- Reproducible.
- Directly relates the myocardial blood flow.
- Excellent correlation with non-invasive tests.

Value of FFR

- In a vessel without any obstructions, the value of FFR is 1.
- Based on FAME and FAME II trials [2, 3]:
  - FFR $\leq$0.8 is considered significant and may benefit from coronary revascularization.
  - Patients with FFR > 0.8 have an excellent prognosis with medical therapy alone.
  - Only 0.2% of FFR-negative patients had an MI at 2 years follow-up.

Summary of Major FFR Trials (Table 4.1)

Drugs Used

- Adenosine
  - IV: Preferred over a central line or a large-bore peripheral line 140 mcg/kg/min infusion.
  - IC: LCA, 80–120 mcg bolus. Bolus repeated three times. Lower dose 60–80 mcg bolus for RCA.

Table 4.1 Summary of major FFR trials

Trial	Study design	N	Treatment	Results
DEFER	Multi-center randomized	325	PCI vs FFR guided deferral	No additional survival benefit with revascularization when FFR $\geq$0.75
FAME	Multi-center randomized	750	Angiography guided vs FFR guided PCI	Less MACE (death, MI or repeat revascularization) with FFR guided PCI in multivessel CAD
FAME II	Multi-center randomized	1220	FFR guided PCI + OMT vs OMT alone	Less MACE with FFR guided PCI + OMT

Key: PCI = percutaneous coronary intervention; MI = myocardial infarction; OMT = optimal medical therapy

- Papaverine IC: at least 10 mg bolus for RCA and at least 15 mg for LCA.
- Regadenoson IV: a single bolus of 400 mcg.

Role of FFR

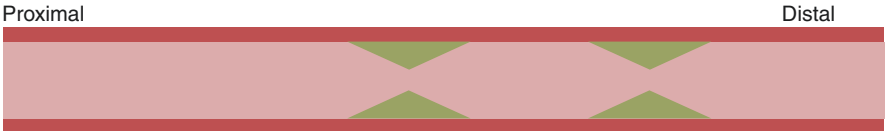
- Multivessel disease.
- Left main disease.
- Ostial lesion.
- Multiple lesions in a single vessel.
- Bifurcation.
- Evaluation of intermediate lesions (40–70% stenosis).

Steps of FFR

- Anticoagulation as per institution protocol.
- Flush the pressure wire system with saline and keep it flat on the table.
- Zero the pressure wire system and remove the FFR wire carefully.
- Pressure sensor is located at the junction between the radiolucent and the radiopaque portion of the wire.
- Advance the wire into the proximal part of the vessel so that the pressure sensor is proximal to the stenosis and remove the introducer needle. Flush the guide catheter system with saline while holding the wire and then equalize. The difference between Pa and Pd pressure tracings [offset] after equalizing to '1.00' should not be greater than 15. If the offset is greater than 15, the FFR readings are not valid.
- For LM/ostial main vessel stenosis, always disengage the guide and equalize in the aorta.
- Once equalized, reintroduce the wire introducer and advance the FFR wire distal to the lesion. Start vasodilators as per protocol.
- While removing the wire, reconfirm that both the pressures are equal once the radiopaque part of the wire is pulled back proximal to the lesion.
- Take a final angiogram to ensure there were no complications from the FFR wire.

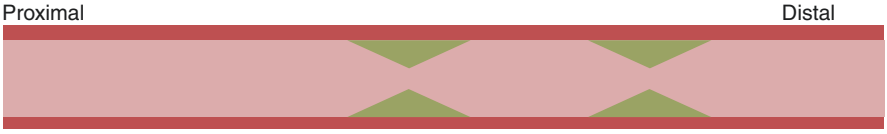
FFR of Multiple Lesions in the Same Vessel

- Consider the FFR as the FFR of the entire vessel rather than that of individual lesions.
- If FFR >0.8, none of the lesions is significant. No intervention is required.



	Lesion A		Lesion B
	Proximal FFR	FFR between Lesion A and B	Distal FFR
FFR value	1	0.7	0.6
Mean pressure	85	60	52
Mean pressure drop		25	8

Fig. 4.3 Clinical example showing two lesions and FFR readings



	Lesion A		Lesion B
	Proximal FFR	FFR between Lesion A and B	Distal FFR
FFR value	1	0.9	0.6
Mean pressure	85	76	52
Mean pressure drop		9	24

Fig. 4.4 Clinical example showing two lesions and FFR readings

- For tandem and sequential lesions, the maximum drop in mean pressure should be observed. In Fig. 4.3, FFR of the vessel with two tandem lesions is ≤0.8 and the drop in mean pressure is more for the proximal lesion than the distal lesion. Here the proximal lesion needs to be treated first. In Fig. 4.4, FFR of the vessel with two tandem lesions is ≤0.8 but the drop in mean pressure is more for the distal lesion than the proximal lesion. Here the distal lesion needs to be treated first. After the first intervention, the second lesion may be reassessed. This is true for all lesions in series in a vessel except for left main disease. If the two lesions are close enough, a single stent covering both lesions may be considered.

FFR in Left Main Disease

- FFR values (>0.80) in left main lesions are associated with excellent long-term outcomes.
- Presence of downstream disease may affect the FFR value of LM disease.
- Myocardial bed supplied by the LM artery may be larger if it supplies collaterals to an occluded RCA.
- Presence of a significant lesion in either the LAD or the LCX makes the myocardial bed smaller for the LM leading to a falsely higher FFR.
- Left main FFR alone cannot be accurately measured when there are significant downstream serial lesions.
- If the LAD and LCX are hemodynamically insignificant, the left main FFR will be accurate.
- Use smaller (6 Fr), less aggressive guides (FL, VL) for the procedure to avoid LM dissection at the site of the lesion.

FFR in SVG Disease

- The role of FFR in SVG lesions is controversial as the distal coronary pressure represents blood flow due to both SVG and a non-occluded native vessel.

FFR in Acute MI

- The role of FFR to evaluate non-culprit vessels in STEMI is controversial due to microvascular obstruction from thrombus embolization and vasospasm associated with infarct.
- After an MI, the FFR may be normal even though angiographically the obstruction is significant.
  - This may be because the infarcted territory has more scar tissue and less viable tissue, rather than a false underestimation of FFR provided maximal hyperemia is obtained.

FFR in in-Stent Restenosis

- The role of FFR has not been well defined in ISR and the lesion should be assessed by IVUS if needed.

FFR Troubleshooting

- Improper Preparation/Setup
  - Height of the fluid-filled pressure transducer. An increase or decrease in 10 cm from the mid atrial level leads to a change in aortic pressure by 10 mmHg.

  - Make sure the system is flushed with normal saline before zeroing the FFR wire.
  - Equalize the pressure wire with guide catheter pressure appropriately.
  - Flush the system with normal saline before equalizing as the presence of contrast will lead to pressure damping.
  - Remove the introducer needle from the Y connector before equalizing the pressures.
- Inadequate Hyperemia
  - Obtaining hyperemia is the single most important step in assessing lesion significance by FFR.
  - Submaximal hyperemia is indicated by an undulating FFR Pd/Pa tracing.
  - Increasing dose of adenosine to 180 mcg/kg/min has been shown to produce maximal hyperemia.
- Pressure Drift
  - Presence of FFR $\leq$0.8 with the same pressure trace in the aorta and distal coronary artery is likely due to pressure drift.
  - Absence of a ventricularized pattern of distal pressure trace rules out an abnormal FFR due to an epicardial vessel stenosis.
- Guide Catheter Issues
  - Larger guide catheter leads to lumen narrowing leading to falsely higher FFR.
  - Diagnostic catheter with narrow lumen leads to aortic pressure dampening leading to falsely higher FFR.
  - Avoid using side-hole catheters.
  - For LM/ostial, stenosis always disengage the guide and equalize with wire in the aorta.
- Whipping Artifacts
  - If the coronary wire touches the wall, a whipping artifact occurs leading to a falsely high coronary pressure.
  - This can be managed by pulling the wire slightly back or advancing it 2–3 mm.
- Difficulty in crossing the lesion
  - Use another wire as a buddy wire to help cross the lesion with the FFR wire.

Instantaneous Wave-Free Ratio

iFR Development

- The principle of iFR is based on the concept that coronary microvascular resistance is constant during the diastolic wave-free period, defined as beginning from 25% into diastole to 5 ms before the end of diastole, and that P_d/P_a measured during this period is a surrogate of coronary flow during maximal hyperemia [4]. Measurement of iFR requires the use of a pressure wire but obviates the need for adenosine. It, therefore, avoids the side effects and symptoms associated with adenosine infusion and incurs less cost.

Summary of Major iFR Trials (Table 4.2)

Steps of iFR

- Technique of performing iFR is similar to FFR without the need for inducing hyperemia.
- Cut-off value of FFR and iFR (Fig. 4.5).

iFR Co-registration and Angiography to Guide Coronary Interventions (Fig. 4.6)

- iFR-pullback system plot, align and overlay the physiological map onto the angiogram using the time-stamp data recorded during a manual pullback, to provide a rapid visualization of areas of iFR loss and an easy evaluation of which lesions cause the highest physiological impact on the ischemic burden in the presence of tandem or diffuse disease.

Table 4.2 Summary of major iFR trials

Trial	Study design	Treatment	Results
DEFINE-FLAIR	Multi-center randomized	iFR guided vs FF guided revascularization	iFR non-inferior to FRR with respect to MACE (death, MI or unplanned revascularization) at 1 year.
iFR-SWEDEHEART	Multi-center randomized	iFR guided vs FF guided revascularization	iFR non-inferior to FRR with respect to MACE (death, MI or unplanned revascularization) at 1 year.
Key: MI = myocardial infarction; OMT = optimal medical therapy			

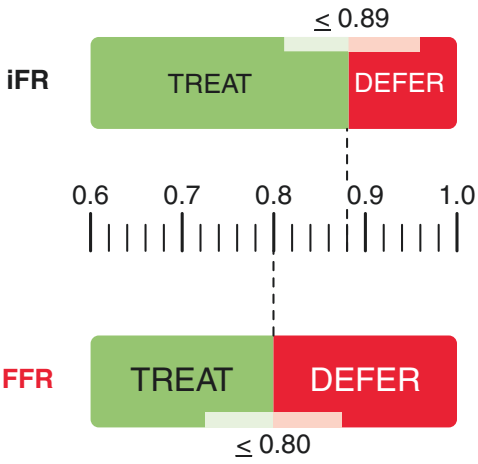


Fig. 4.5 iFR and FFR cut-off values

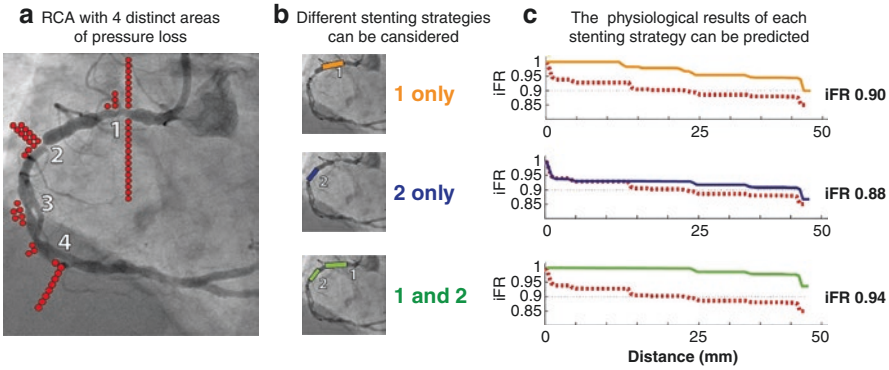


Fig. 4.6 (**a**) RCA with 4 discrete stenosis. A pressure wire is pulled back at rest from the distal vessel the proximal vessel at 0.5 mm/sec using a motorized device. The red-dots correspond to the iFR intensity plot shown in B lower panel. The numbers correspond to the four discrete physiological lesions identified on iFR-pullback. Virtual PCI can be performed on iFR-Pullback to calculate the effect of removing a stenosis. (**b**) Different stenting strategies can be considered. (**c**) On the iFR-pullback trace, a stenosis can be selected for removal, and computer algorithms will model the impact upon the rest of the vessel. Removing stenosis 1 or 2 alone will not give the vessel an iFR well above the threshold for treatment; both stenosis are required for an optimal result. (Reprinted from Nijjer et al. [4] with permission from Elsevier)

- This physiological map permits an accurate assessment of the relevance of each individual lesion in tandem stenosis, and the pattern of iFR loss allows the operator to determine whether the disease is predominantly focal or diffuse.
- The value of mapping the iFR intensity in diffusely diseased vessels enables identification of any focal areas of disease that may cause the predominant pressure loss and therefore be targeted for percutaneous intervention. The percentage contribution of pressure loss can be displayed to assist decision-making.
- "Dots" representing units of pressure loss can help identify which stenosis are most hemodynamically important. In addition, iFR intensity plotted as a function of distance can give additional information regarding the length over which the pressure drop occurs [4].
- This may assist in identifying which lesions in the vessel contribute most to pressure loss and allow operators to estimate the physiological length of a stenosis to help decide between different strategies.
- In addition, the software facilitates PCI strategies by allowing operators to perform virtual PCI: once the more physiologically relevant stenosis has been identified using the physiological map, it can be manually selected for virtual removal in order to predict the best possible post-PCI physiological result, and the functional relevance of residual disease.

Table 4.3 Summary of major CT-FFR trials

Trial	Vessels (vessels with FFR < 0.8)	Co-relation of CT-FFR with FFR	Sensitivity	Specificity
DISCOVER-FLOW	159 (58)	0.68	88	82
DeFACTO	407 (137)	0.63	89	61
NXT	468 (135)	0.82	84	86
Coenen et al	189 (80)	0.59	88	65

Ct-FFR (FFR_{CT})

CT-FFR Development

The computation of FFR is based on three main physiological assumptions:

- The baseline coronary blood flow is proportional to the volume of myocardial mass at resting condition.
- Coronary microvascular resistance at rest is inversely associated with the subtended vessel size (morphometric law).
- A predicable reduction of microcirculatory resistance is seen as response to infusion of vasodilators such as adenosine [5].

FFR_{CT} is then defined as the computed mean coronary pressure distal to a lesion divided by the mean blood pressure in the aorta under conditions of simulated maximal hyperemia.

Summary of Major CT-FFR Trials (Table 4.3)

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5 Antiplatelet and Antithrombotic Therapy in Percutaneous Coronary Interventions

Amit Hooda, Gurpreet S. Johal, and Usman Baber

Introduction

Dual antiplatelet therapy (DAPT) is the cornerstone of pharmacological therapy preventing atherothrombotic complications. Advances in drug-eluting stent technology, antiplatelet pharmacotherapy, and novel parenteral and oral anticoagulants (OAC) have led to constant evolution in periprocedural antithrombotic strategy. The following chapter outlines our cardiac catheterization laboratory's current approach to antiplatelet and anticoagulation therapy in various clinical settings based on current guidelines and expert opinion.

Oral Antiplatelet Therapy

Aspirin: All Patients

- For elective patients, with or without known stable CAD, aspirin 162 mg should be given at the time of obtaining consent.
- For ACS (acute coronary syndrome) patients, non-enteric aspirin 325 mg should be given as early as possible before PCI [1].
- Post-PCI, aspirin 81 mg daily should be continued indefinitely.

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Dual Antiplatelet Therapy

- All patients should be counseled on the necessity and concomitant risk associated with dual antiplatelet therapy (DAPT) before placement of intracoronary stents.
- For patients unable or unwilling to comply with the recommended duration of DAPT, alternative revascularization (bypass) or bare-metal stents (BMS) should be considered.

P2Y12 Antagonist Therapy

- Pre/intra-procedural loading doses for patients *not previously taking the same* $P2Y_{12}$ receptor inhibitor for ≥5 days:
  - Clopidogrel 600 mg.
  - Prasugrel 60 mg.
  - Ticagrelor 180 mg.
- Loading doses for patients *already taking the same* $P2Y_{12}$ receptor inhibitor for ≥5 days:
  - Clopidogrel 300 mg.
  - Prasugrel 30 mg.
  - Ticagrelor 90 mg.
- Platelet Reactivity Unit (PRU) should be checked in patients presenting with stent thrombosis. For clopidogrel *nonresponders* (with confirmed compliance), defined as PRU >230, load with prasugrel 60 mg or ticagrelor 180 mg.

Parenteral $P2Y_{12}$ Inhibitor – Cangrelor [2, 3]

- Only parenteral P2Y12 inhibitor.
- Onset-2 mins, offset of action-1 hour.
- Current Indications for IV Cangrelor use.
  - Bridge for elective non-cardiac surgery in a patient requiring DAPT (1-3 M or 6 M of DES, Table 5.1).
  - MI/ACS with hemodynamic instability and poor absorption status; Cardiogenic shock, Intubated.
  - ACS and SIHD PCI with multiple high-risk angio features (bifurcation, thrombus, 3+ stents, Ca+).
  - MI/ACS patient awaiting cardiac surgery or urgent non-cardiac surgery (hip fracture, GI surgery).
  - ACS patient with clinical presentation suggestive of CABG (marked ST-segment depression, known extensive CAD).

Table 5.1 Recommendation for bridging DAPT before surgery in recent PCI/stent, 0–1 M post stent placement, or 1–6 M from high-risk stent[a]

P2Y12 inhibitor	Surgery to be performed on ASA	Surgery to be performed on no antiplatelet
Clopidogrel or Ticagrelor	Stop P2Y12 inhibitor 5 days prior to surgery	Stop ASA 5 days prior to surgery.
		Stop P2Y12 inhibitor 10 days prior to surgery
	Start cangrelor[b] 3 days prior to surgery	Start cangrelor 7 days prior to surgery
Prasugrel	Stop P2Y12 inhibitor 7 days prior to surgery	Stop ASA 5 days prior to surgery.
		Stop P2Y12 inhibitor 14 days prior to surgery
	Start cangrelor 3 days prior to surgery	Start cangrelor 10 days prior to surgery

[a]LM, bifurcation with 2 stents, multivessel, long/diffuse disease
[b]Cangrelor dose: 0.75 μg/kg/min infusion without a bolus

Table 5.2 Switching to oral P2Y12 inhibitor in high-risk PCI patients on IV Cangrelor

Patient condition	Cangrelor dose	Antiplatelet to be restarted one pt. condition resolves: FOLLOW-ON ORAL DRUG
Patient unable to take PO or acute intracoronary thrombosis or cardiogenic shock or hemodynamic compromise with active consideration for LVAD escalation	30 μg/kg bolus and 4 μg/kg^{-1}/min^{-1} infusion for at least 2 h or duration PCI. Continue cangrelor at a rate of 0.75 μg/kg^{-1}/min^{-1} until patient can take PO or until shock resolves	Clopidogrel: 600 mg immediately after discontinuation of cangrelor
		Ticagrelor: 180 mg at any time during cangrelor infusion or immediately after discontinuation of cangrelor
		Prasugrel: 60 mg immediately after discontinuation of cangrelor

- Recommended dose: 30 mcg/kg IV bolus followed by 4 mcg/kg/min IV infusion, continue for at least 2 hours or the duration of the procedure, whichever is longer.
- Transition to oral P2Y12 inhibitor (Table 5.2):
  - Ticagrelor 180 mg at any time during cangrelor infusion or immediately after discontinuation.
  - Clopidogrel 600 mg or prasugrel 60 mg after cangrelor discontinuation as their antiplatelet effect will be attenuated due to competitive inhibition.

Special Considerations

- Clopidogrel is considered the default P2Y12 inhibitor of choice in patients with [4, 5]:
  - SIHD treated with PCI.
  - Concomitant OAC.
  - ACS patients in whom ticagrelor or prasugrel are contraindicated.
  - Drug intolerance/adverse effects to other P2Y12 inhibitors.
  - High bleeding risk.
- Prasugrel (contraindicated in patients with a history of TIA/CVA and avoided in patients weighing <60 kg or ≥ 75 years of age) should be preferred in the following clinical situations:
  - STEMI undergoing PCI.
  - High-risk coronary anatomy.
  - Clopidogrel allergy.
  - Clopidogrel nonresponder (PRU >230 on maintenance dose of clopidogrel).
  - Stent thrombosis on clopidogrel.
- In patients >75 years old or with history of TIA/CVA, or weighing <60 kg, ticagrelor should be preferred in the following clinical situations:
  - ACS (STEMI/NSTEMI) patients irrespective of PCI.
  - Complex PCI.
  - Clopidogrel allergy.
  - Clopidogrel nonresponder (PRU >230 on maintenance dose of clopidogrel).
  - Stent thrombosis on clopidogrel.
- For pre-liver or renal transplant patients, DES is preferred with a shorter duration of DAPT [5].
- Ticagrelor can be used as a monotherapy in selected patients with aspirin allergy or intolerance.

Duration and Switching of DAPT

- All ACS patients should receive 12 months of DAPT (Fig. 5.1).
- Non-ACS patients receiving DES should receive a minimum of 6 months of DAPT.
- Prolonged DAPT beyond 12 months may be considered in patients tolerating therapy without bleeding issues, with a complex disease requiring multiple stents, and/or significant residual CAD. Ticagrelor in a dose of 60 mg BID is the preferred P2Y12 inhibitor [6].
- Shorter DAPT <6 months should be considered in patients who are at HBR or had significant overt bleeding (Table. 5.3).
- Multiple studies with newer-generation DES have shown a shift towards shorter standard DAPT post-PCI in HBR individuals. 1–3 months of DAPT may ensure sufficient protection from stent thrombosis reducing the risk of bleeding [7, 8].

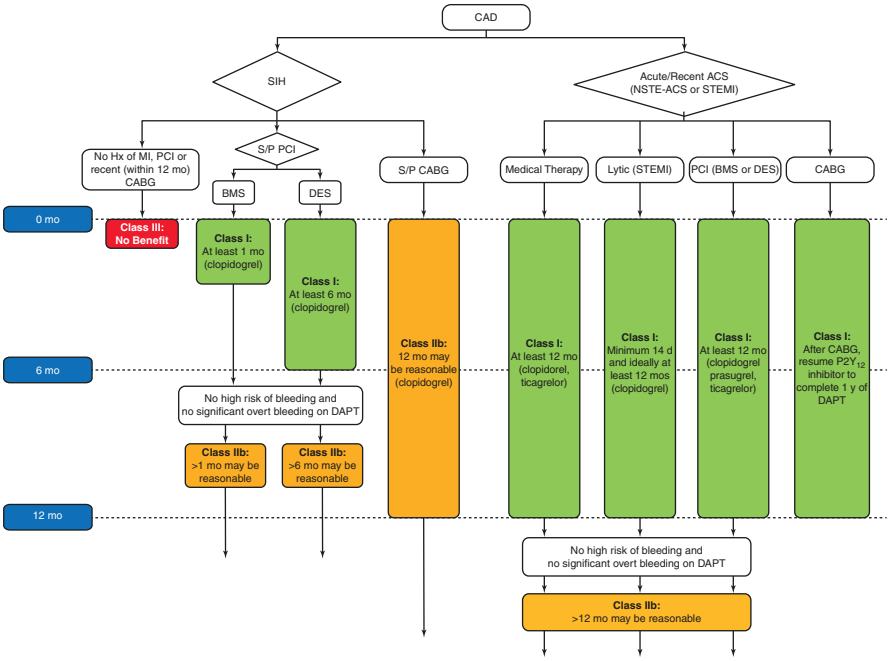


Fig. 5.1 Algorithm for current recommendations for the duration of DAPT. (Adapted from the 2016 ACC/AHA Guideline Focused Update on DAPT [4])

Table 5.3 Factors to consider when deciding the duration of DAPT

Favoring prolong DAPT	Favoring short DAPT/ HBR
Prior stent thrombosis on adequate Antiplatelet therapy	Any intracerebral bleed
Chronic kidney disease (CrCl <60 ml/min)	Clinical indication for anticoagulation
Multivessel disease in diabetics	Steroid use >30 days after PCI
≥3 stents implanted	Hospital admission for bleeding during the prior 12 months
Bifurcation with 2 stents implanted	Blood dyscrasia
Total stent length > 60 mm	History of bleeding diathesis
In-stent restenosis	Hgb <11 g/dl (or transfusion in <1 month prior)
Stenting of the last remaining patent coronary artery	Thrombocytopenia (Plt <100 K)
Multiple prior MIs	Ongoing malignancy with high bleeding Potential
CTO	Chronic alcohol abuse
	Low body weight
	ESRD
	Advanced age

Adapted from Capodanno et al. [9]

- DAPT and PRECISE-DAPT scores are helpful in decision making regarding the duration of DAPT beyond 12 months. DAPT score ≥ 2 points and PRECISE-DAPT score < 25 favors a standard or long DAPT duration and vice versa [5].
- Combined dosing for P2Y12 antagonists with aspirin 81 mg daily:
  - Clopidogrel 75 mg orally once daily.
  - Prasugrel 5 mg orally once daily (10 mg daily in patients >100 kg).
  - Ticagrelor 90 mg orally twice daily.
- Switching among different P2Y12 inhibitors, see Fig. 5.2.

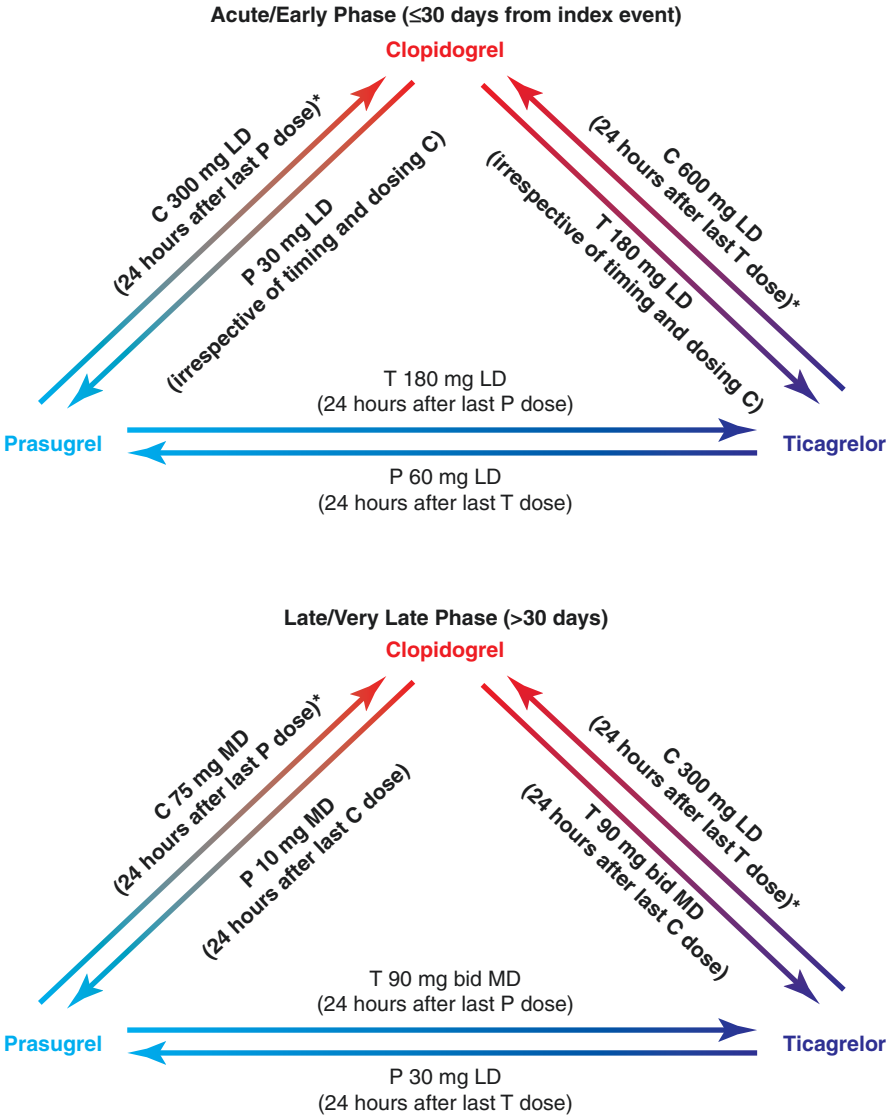


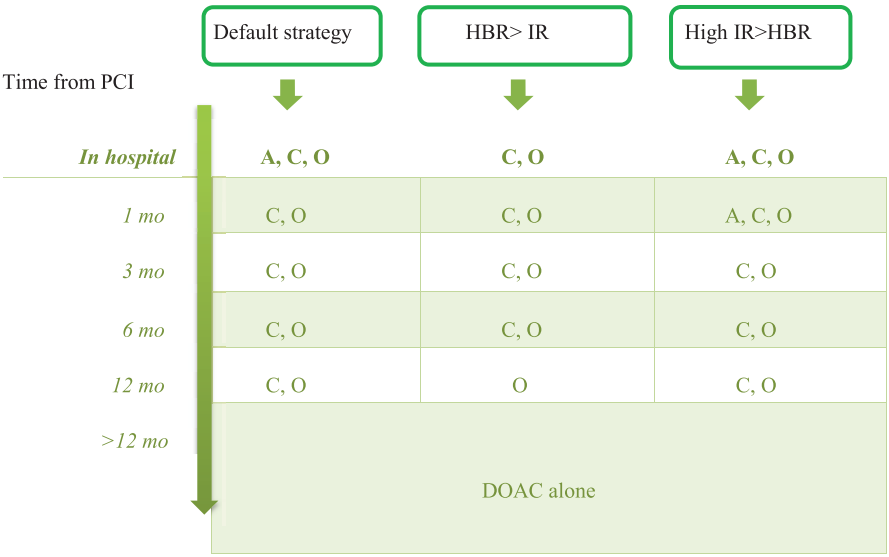
Fig. 5.2 Switching among different P2Y12 inhibitors. (Adapted from International Expert Consensus on Switching Platelet P2Y12 Receptor-Inhibiting Therapies [10])

Antiplatelet Therapy in PCI Patients Requiring Oral Anticoagulation

- Managing optimal antithrombotic therapy is a clinical conundrum. Numerous studies have shown single antiplatelet therapy (SAPT) preferably, clopidogrel in combination with DOACs provides better safety compared with a regimen with VKA and is the mainstay of therapy. Triple antithrombotic therapy (TAT) is associated with high bleeding risk and should be avoided except in rare patients at higher risk of ischemic events.
- DOACs should be preferred to VKA.
- No need to hold OAC in urgent or emergent procedures.
- Withhold DOAC 24 hours before an elective procedure, 48 hours if a patient has renal dysfunction.
- Recommended doses of DOACs:
  - Rivaroxaban 15 mg OD, 10 mg od in renal dysfunction.
  - Dabigatran 150 mg BID, 110 mg BID in age > 70 yrs.
  - Apixaban 2.5 mg BID.
- VKA (Coumadin) is indicated only in patients with [9]:
  - Moderate to severe mitral stenosis.
  - Mechanical prosthetic heart valves.
- All patients are loaded with aspirin and P2Y12 antagonist, regardless of the long-term anticoagulant requirement.
- In patients at higher risk for bleeding, clopidogrel is preferred as the initial P2Y12 antagonist of choice. If the patient is a clopidogrel nonresponder, ticagrelor is the next preferred option. Prasugrel is not recommended [9].
- For the duration of antiplatelet therapy in the setting of long-term anticoagulation, see Fig. 5.3.
- Post-PCI resumption of anticoagulant therapy:
  - Warfarin should be dosed the same day/evening due to an expected delay in achieving therapeutic INR (2–2.5).
  - DOACs should be dosed the following day unless there is a compelling indication for immediate resumption (Fig. 5.3) [11].
  - In patients with compelling indications requiring immediate anticoagulation (or re-bridge to oral anticoagulation) post-PCI, such as acute venous/pulmonary thromboembolism or mechanical valve, a heparin drip may be started without bolus 2 hours post-PCI with successful hemostasis using a closure device or 6 hours after sheath pull with successful hemostasis using manual compression or after a radial procedure.

Anticoagulation for PCI

- Administer bivalirudin bolus (0.75 mg/kg) via the intra-arterial sheath and begin continuous infusion (1.75 mg/kg/h).
- Check activated clotting time (ACT) 3 min after bolus:
  - For ACT <270, administer an additional 1/2 bolus of bivalirudin.



IR= Ischemic risk, HBR= High bleeding risk, A= Aspirin, C= Clopidogrel, O= Oral anticoagulation

Fig. 5.3 Current recommendations for the management of OAC and Antiplatelet Therapy in Patients with AF undergoing PCI. (Adapted from Capodanno et al. JACC 2019 [11])

  - For ACT 270–299, administer an additional 1/3 bolus of bivalirudin.
  - The guiding catheter may be inserted once ACT>200.
  - Intracoronary guidewire/equipment may be inserted once ACT >300.
- Post-PCI:
  - For patients naïve to clopidogrel (on therapy <1 week or receiving the first time load of 600 mg <2 hours before PCI), bivalirudin infusion should be continued for 2 hours from the time of stent implantation.
  - For patients receiving prasugrel or ticagrelor (on maintenance therapy or receiving the first time load on the table), bivalirudin infusion can be discontinued as soon as the PCI is completed.
  - For patients requiring a periprocedural glycoprotein (GP) IIb/IIIa inhibitor bolus, bivalirudin infusion can be stopped after administration of the GP IIb/IIIa bolus.

Elective Non-cardiac Surgery after PCI [5]

- Defer elective surgeries requiring discontinuation of the P2Y12 inhibitor for at least 1–3 months.
- Continue Aspirin perioperatively.
- Surgical procedures mandating interruption of DAPT perioperatively, a bridging strategy with cangrelor or GP IIb/IIIa may be considered, especially if performed within 1 month of PCI.

Role of Glycoprotein (GP) IIb/IIIa Inhibitors [13]

- GP IIb/IIIa antagonist administration for bailout purposes only.
- At the discretion of the operator, single (or double) bolus eptifibatide (single = 180 mcg/kg) may be administered in cases of edge dissection, side branch closure, slow flow, no-reflow, embolization, and large thrombus.
- For patients receiving abciximab, the platelet count should be checked 3 hours post-procedure and the following morning.
- For patients receiving eptifibatide, the platelet count should be checked 6 hours post-procedure and the following morning.
- GPIIb/IIIa infusion should be discontinued if significant thrombocytopenia develops (platelets <100 K).

Managing Thrombocytopenia Post-PCI

Platelets <20 K

- Discontinue all antiplatelet therapies.
- Transfuse 5 or more units of platelets until platelet count >20 K.
- Aspirin 81 mg and clopidogrel 75 mg can be resumed once platelet count >50 K.
- Platelet count should be checked daily until >70 K.

Platelets 20–50 K

- Discontinue GP IIb/IIIa inhibitor.
- Transfuse platelets only for active bleeding.
- Aspirin 81 mg and clopidogrel 75 mg can be resumed once platelet count >50 K.

Platelets 50–100 K

- Discontinue GP IIb/IIIa inhibitor.
- Aspirin 81 mg and clopidogrel 75 mg can be continued as long as platelet count remains >50 K.

Future Directions

Aspirin free-strategies 1–3 months post-PCI have shown encouraging results in several studies, thereby significantly reducing the incidence of bleeding events, without altering the ischemic risk [12]. Individualization of DAPT duration particularly in HBR patients appears therefore mandatory, making aspirin not necessary in several cases. Ongoing studies may potentially shift the paradigm of antiplatelet therapy after PCI in the near future.

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Patient Selection and Appropriate Use Criteria

6

Gurpreet S. Johal and Samin K. Sharma

Introduction

The American College of Cardiology, Society for Cardiovascular Angiography and Interventions, Society of Thoracic Surgeons, and American Association for Thoracic Surgery, and other societies, developed and published appropriate use criteria (AUC) for coronary revascularization initially in 2009 [1, 2]. The AUC were updated in 2012, and more recently in 2017. The primary purpose of the AUC is to provide a framework to assess clinical practice patterns, expand physician decision-making, and improve the quality of care [1]. Since the publication of the AUC for coronary revascularization in 2009, the volume of nonacute PCI has significantly decreased [3, 4]. Moreover, the proportion of nonacute PCIs classified as inappropriate has also declined [3].

AUC are either evidence-based or are expert consensus opinion (when evidence is lacking), and are approved by ACC and the American Heart Association (AHA). AUC are not intended to diminish the complexity or uncertainty of clinical decision-making, and are not to replace thoughtful clinical judgment [1].

Appropriate Use Criteria for Stable Ischemic Heart Disease

The AUC guidelines assume patients are receiving all indicated therapies for secondary prevention of cardiovascular disease with pharmacotherapy at doses that adequately control patients' symptoms or are maximally tolerated [1]. AUC does

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not evaluate all patient conditions/variables which may affect the strategies used to manage patients with CAD. Examples of such conditions/variables include [1]:

- Severe chronic kidney disease.
- Severe peripheral vascular disease.
- Known malignancies.
- Poor lung function.
- Advanced liver disease.
- Advanced dementia.
- Other comorbidities that have excluded patients from clinical trials that provide the evidence base for coronary revascularization.

In developing these AUC for coronary revascularization in patients with stable ischemic heart disease (SIHD), a panel of experts scored each indication using the following definition of appropriate use*: A coronary revascularization is appropriate care when the potential benefits, in terms of survival or health outcomes (symptoms, functional status, and/or quality of life), exceed the potential negative consequences of the treatment strategy*. The panel scored each indication on a scale from one to nine based on their level of agreement with the definition of appropriate use for that indication as follows [1]:

- Score 1 to 3: Rarely appropriate care (risks > benefit).
- Score 4 to 6: May be appropriate care (potential benefit).
- Score 7 to 9: Appropriate care (risk < benefit).

In patients with SIHD, AUC indications for coronary revascularization were developed considering several variables [1]:

- Clinical presentation/ischemic symptoms.
- Use of antianginal medications (Beta-blockers, Calcium channel blockers, Long-acting nitrates, Ranolazine).
- Results of noninvasive tests to evaluate the presence and severity of myocardial ischemia (Table 6.1).
- Presence of other confounding factors and comorbidities such as diabetes.
- Extent and complexity of anatomic coronary artery disease.
- Prior coronary artery bypass surgery.
- Invasive testing such as intravascular ultrasound (IVUS) and invasive physiology such as fractional flow reserve (FFR).

Per AUC, significant coronary stenosis in SIHD was defined as [1]:

- ≥70% luminal diameter narrowing of epicardial stenosis, measured by visual assessment in the "worst view" angiographic projection.
- ≥50% luminal diameter narrowing of left main stenosis, measured by visual assessment in the "worst view" angiographic projection.
- 40% to 70% luminal narrowing of epicardial stenosis, measured by visual assessment in the "worst view" angiographic projection with an abnormal FFR (abnormal if ≤0.80).

Refer to Figs. 6.1, 6.2, and 6.3 for a summary of AUC for SIHD.

Table 6.1 Noninvasive risk stratification

High risk (>3% annual death or MI)
1. Severe resting LV dysfunction (LVEF <35%) not readily explained by noncoronary causes
2. Resting perfusion abnormalities in ≥10% of the myocardium in patients without prior history or evidence of MI
3. Stress ECG findings including ≥2 mm of ST-segment depression at low workload or persisting into recovery, exercise-induced ST-segment elevation, or exercise-induced VT/VF
4. Severe stress-induced LV dysfunction (peak exercise LVEF <45% or drop in LVEF with stress ≥10%)
5. Stress-induced perfusion abnormalities encumbering ≥10% myocardium or stress segmental scores indicating multiple vascular territories with abnormalities
6. Stress-induced LV dilation
7. Inducible wall motion abnormality (involving >2 segments or 2 coronary beds)
8. Wall motion abnormality developing at low dose of dobutamine (≤10 mg/kg/min) or at a low heart rate (<120 beats/min)
9. CAC score > 400 Agatston units
10. Multivessel obstructive CAD (≥70% stenosis) or left main stenosis (≥50% stenosis) on CCTA
Intermediate risk (1% to 3% annual death or MI)
1. Mild/moderate resting LV dysfunction (LVEF 35% to 49%) not readily explained by noncoronary causes
2. Resting perfusion abnormalities in 5% to 9.9% of the myocardium in patients without a history or prior evidence of MI
3. ≥ 1 mm of ST-segment depression occurring with exertional symptoms
4. Stress-induced perfusion abnormalities encumbering 5% to 9.9% of the myocardium or stress segmental scores (in multiple segments) indicating 1 vascular territory with abnormalities but without LV dilation
5. Small wall motion abnormality involving 1 to 2 segments and only 1 coronary bed
6. CAC score 100 to 399 Agatston units
7. One vessel CAD with ≥70% stenosis or moderate CAD stenosis (50% to 69% stenosis) in ≥2 arteries on CCTA
Low risk (<1% annual death or MI)
1. Low-risk treadmill score (score ≥ 5) or no new ST segment changes or exercise-induced chest pain symptoms; when achieving maximal levels of exercise
2. Normal or small myocardial perfusion defect at rest or with stress encumbering <5% of the myocardium[a]
3. Normal stress or no change of limited resting wall motion abnormalities during stress
4. CAC score < 100 Agaston units
5. No coronary stenosis >50% on CCTA

[a]Although the published data are limited; patients with these findings will probably not be at low risk in the presence of either a high-risk treadmill score or severe resting LV dysfunction (LVEF <35%). CAC = coronary artery calcium; CAD = coronary artery disease; CCTA = coronary computed tomography angiography; LV = left ventricular; LVEF = left ventricular ejection fraction; and MI = myocardial infarction

Adapted from Patel et al. [1] with permission from Springer Nature

a

Appropriate Use Socre (1-9)

One-Vessel Disease

		Asymptomatic				Ischemic Symptoms			
		Not on AA Therapy or With AA Therapy		Not on AA Therapy		On 1 AA Drug (BB Preferred)		On ≥2 AA Drugs	
	Indication	PCI	CABG	PCI	CABG	PCI	CABG	PCI	CABG
No Proximal LAD or Proximal Left Dominant LCX Involvement									
1.	▪ Low-risk findings on noninvasive testing	R (2)	R (1)	R (3)	R (2)	M (4)	R (3)	A (7)	M (5)
2.	▪ Intermediate- or high-risk findings on noninvasive testing	M (4)	R (3)	M (5)	M (4)	M (6)	M (4)	A (8)	M (6)
3.	▪ No stress test performed or, if performed, results are indeterminate ▪ FFR ≤0.80*	M (4)	R (2)	M (5)	R (3)	M (6)	M (4)	A (8)	M (6)
Proximal LAD or Proximal Left Dominant LCX Involvement Present									
4.	▪ Low-risk findings on noninvasive testing	M (4)	R (3)	M (4)	M (4)	M (5)	M (5)	A (7)	A (7)
5.	▪ Intermediate- or high-risk findings on noninvasive testing	M (5)	M (5)	M (6)	M (6)	A (7)	A (7)	A (8)	A (8)
6.	▪ No stress test performed or, if performed, results are indeterminate ▪ FFR ≤0.80	M (5)	M (5)	M (6)	M (6)	M (6)	M (6)	A (8)	A (7)

b

Two-Vessel Disease

		Asymptomatic				Ischemic Symptoms			
		Not on AA Therapy or With AA Therapy		Not on AA Therapy		On 1 AA Drug (BB Preferred)		On ≥2 AA Drugs	
	Indication	PCI	CABG	PCI	CABG	PCI	CABG	PCI	CABG
No Proximal LAD Involvement									
7.	▪ Low-risk findings on noninvasive testing	R (3)	R (2)	M (4)	R (3)	M (5)	M (4)	A (7)	M (6)
8.	▪ Intermediate- or high-risk findings on noninvasive testing	M (5)	R (4)	M (6)	M (5)	M (6)	M (4)	A (8)	M (8)
9.	▪ No stress test performed or, if performed, results are indeterminate ▪ FFR ≤0.80* in both vessels	M (5)	R (4)	M (6)	M (4)	M (7)	M (5)	A (8)	M (8)
Proximal LAD Involvement and No Diabetes Present									
10.	▪ Low-risk findings on noninvasive testing	M (4)	M (4)	M (5)	M (5)	M (6)	M (6)	A (7)	A (7)
11.	▪ Intermediate- or high-risk findings on noninvasive testing	M (6)	M (6)	A (7)	A (7)	A (7)	A (7)	A (8)	A (8)
12.	▪ No stress test performed or, if performed, results are indeterminate ▪ FFR ≤0.80 in both vessels	M (6)	M (6)	M (6)	M (6)	A (7)	A (7)	A (8)	A (8)
Proximal LAD Involvement With Diabetes Present									
13.	▪ Low-risk findings on noninvasive testing	M (4)	M (5)	M (4)	M (6)	M (6)	A (7)	A (7)	A (8)
14.	▪ Intermediate- or high-risk findings on noninvasive testing	M (5)	A (7)	M (6)	A (7)	A (7)	A (8)	A (8)	A (9)
15.	▪ No stress test performed or, if performed, results are indeterminate ▪ FFR ≤0.80 in both vessels*	M (5)	M (6)	M (6)	A (7)	A (7)	A (8)	A (7)	A (8)

Fig. 6.1 (**a**) AUC for one vessel disease. (**b**) AUC for two-vessel disease. (**c**) AUC for three-vessel disease. (**d**) AUC for left main coronary artery stenosis. (Adapted from Patel et al. [1] with permission from Springer Nature). The number in parentheses next to the rating reflects the median score for that indication. *Substitution of a newer coronary pressure ratio that does not require hyperemia for FFR may be considered provided the appropriate reference values are used. A = appropriate; AA = antianginal; BB = beta-blockers; CABG = coronary artery bypass graft; FFR = fractional flow reserve; LAD = left anterior descending coronary artery; LCX = left circumflex artery; LMCA = left main coronary artery; M = may be appropriate; PCI = percutaneous coronary intervention; R = rarely appropriate; and SYNTAX = Synergy between PCI with Taxus and Cardiac Surgery trial)

c

Three-Vessel Disease

		Asymptomatic				Ischemic Symptoms			
		Not on AA Therapy or With AA Therapy		Not on AA Therapy		On 1 AA Drug (BB Preferred)		On ≥2 AA Drugs	
	Indication	PCI	CABG	PCI	CABG	PCI	CABG	PCI	CABG
Low Disease Complexity (e.g., Focal Stenoses, SYNTAX ≤22)									
16.	■ Low-risk findings on noninvasive testing ■ No diabetes	M (4)	M (5)	M (5)	M (5)	M (5)	M (6)	A (6)	A (7)
17.	■ Intermediate- or high-risk findings on noninvasive testing ■ No diabetes	M (6)	A (7)	A (7)	A (7)	A (7)	A (8)	A (8)	A (8)
18.	■ Low-risk findings on noninvasive testing ■ Diabetes present	M (4)	M (6)	M (5)	M (6)	M (6)	A (7)	A (7)	A (8)
19.	■ Intermediate- or high-risk findings on noninvasive testing ■ Diabetes present	M (6)	A (7)	M (6)	A (8)	A (7)	A (8)	A (7)	A (9)
Intermediate or High Disease Complexity (e.g. Multiple Features of Complexity as Noted Previously, SYNTAX >22)									
20.	■ Low-risk findings on noninvasive testing ■ No diabetes	M (4)	M (6)	M (4)	A (7)	M (5)	A (7)	M (6)	A (8)
21.	■ Intermediate- or high-risk findings on noninvasive testing ■ No diabetes	M (5)	A (7)	M (6)	A (7)	M (6)	A (8)	M (6)	A (9)
22.	■ Low-risk findings on noninvasive testing ■ Diabetes present	M (4)	A (7)	M (4)	A (7)	M (5)	A (8)	M (6)	A (9)
23.	■ Intermediate- or high-risk findings on noninvasive testing ■ Diabetes present	M (4)	A (8)	M (5)	A (8)	M (5)	A (8)	M (6)	A (9)

d

Left Main Disease

		Asymptomatic				Ischemic Symptoms			
		Not on AA Therapy or With AA Therapy		Not on AA Therapy		On 1 AA Drug (BB Preferred)		On ≥2 AA Drugs	
	Indication	PCI	CABG	PCI	CABG	PCI	CABG	PCI	CABG
24.	■ Isolated LMCA disease ■ Ostial or midshaft stenosis	M (6)	A (8)	A (7)	A (8)	A (7)	A (9)	A (7)	A (9)
25.	■ Isolated LMCA disease ■ Bifurcation involvement	M (5)	A (8)	M (5)	A (8)	M (5)	A (9)	M (6)	A (9)
26.	■ LMCA disease ■ Ostial or midshaft stenosis ■ Concurrent multivessel disease ■ Low disease burden (e.g., 1-2 additional forcal stenoses, SYNTAX socre ≤22)	M (6)	A (8)	M (6)	A (9)	A (7)	A (9)	A (7)	A (9)
27.	■ Ostial or midshaft stenosis ■ Concurrent multivessel disease ■ Intermediate or high disease burden (e.g., 1-2 additional bifurcation stenosis, long stenoses, SYNTAX socre >22)	M (4)	A (9)	M (4)	A (9)	M (4)	A (9)	M (4)	A (9)
28.	■ LMCA disease ■ Bifurcation involvement ■ Low disease burden in other vessels (e.g., 1-2 additional forcal stenosis, SYNTAX socre ≤22)	M (4)	A (8)	M (5)	A (8)	M (5)	A (9)	M (6)	A (9)
29.	■ LMCA disease ■ Bifurcation involvement ■ Intermediate or high disease burden in other vessels (e.g., 1-2 additional bifurcation stenosis, long stenoses, SYNTAX socre >22)	R (3)	A (8)	R (3)	A (9)	R (3)	A (9)	R (3)	A (9)

Fig. 6.1 (continued)

a

		Asymptomatic				Ischemic Symptoms			
		Not on AA Therapy or With AA Therapy		Not on AA Therapy		On 1 AA Drug (BB Preferred)		On ≥2 AA Drugs	
Indication		PCI	CABG	PCI	CABG	PCI	CABG	PCI	CABG
Stenosis Supplying 1 Territory Disease (Bypass Graft or Native Artery) to Territory Other Than Anterior									
30.	■ Low-risk findings on noninvasive testing	R (3)	R (1)	R (3)	R (2)	M (6)	R (3)	A (7)	M (4)
31.	■ Intermediate- or high-risk findings on noninvasive testing	M (5)	R (3)	M (5)	R (3)	A (7)	M (4)	A (8)	M (5)
32.	■ No stress test performed or, if performed, the results are indeterminate ■ FFR of stenosis ≤0.80*	M (4)	R (3)	M (4)	R (3)	M (6)	M (4)	A (8)	M (5)
Stenoses Supplying 2 Territories (Bypass Graft or Native Artery, Either 2 Separate Vessels or Sequential Graft Supplying 2 Territories) Not Including Anterior Territory									
33.	■ Low-risk findings on noninvasive testing	R (3)	R (2)	M (4)	R (3)	M (6)	R (3)	A (7)	M (5)
34.	■ Intermediate- or high-risk findings on noninvasive testing	M (5)	R (3)	M (5)	M (4)	A (7)	M (5)	A (8)	M (6)

b

		Asymptomatic				Ischemic Symptoms			
		Not on AA Therapy or With AA Therapy		Not on AA Therapy		On 1 AA Drug (BB Preferred)		On ≥2 AA Drugs	
Indication		PCI	CABG	PCI	CABG	PCI	CABG	PCI	CABG
Stenosis Supplying-1 Territory Disease (Bypass Graft or Native Artery)-Anterior (LAD) Territory									
35.	■ Low-risk findings on noninvasive testing	M (4)	R (3)	M (5)	R (3)	M (6)	M (4)	A (7)	M (5)
36.	■ Intermediate- or high-risk findings on noninvasive testing	M (6)	M (4)	M (6)	R (4)	A (7)	M (5)	A (8)	M (6)
37.	■ No stress test performed or, if performed, the results are indeterminate ■ FFR of stenosis ≤0.80*	M (5)	M (4)	M (6)	R (4)	A (7)	M (5)	A (8)	M (6)
Stenoses Supplying 2 Territories (Bypass Graft or Native Artery, Either 2 Separate Vessels or Sequential Graft Supplying 2 Territories) LAD Plus Other Territory									
38.	■ Low-risk findings on noninvasive testing	M (5)	M (4)	M (6)	M (4)	A (7)	M (5)	A (7)	M (6)
39.	■ Intermediate- or high-risk findings on noninvasive testing	M (6)	M (5)	A (7)	M (6)	A (7)	A (7)	A (8)	A (8)
Stenoses Supplying 3 Territories (Bypass Graft or Native Arteries, Separate Vessels, Sequential Grafts, or Combination Thereof) LAD Plus 2 Other Territories									
40.	■ Low-risk findings on noninvasive testing	M (5)	M (5)	M (6)	M (5)	M (6)	M (6)	A (7)	A (7)
41.	■ Intermediate- or high-risk findings on noninvasive testing	A (7)	A (7)	A (7)	A (7)	A (7)	A (7)	A (8)	A (8)

Fig. 6.2 (**a**) AUC for an internal mammary artery to left anterior descending artery patent and without significant stenosis. (**b**) AUC for an internal mammary artery to the left anterior descending artery, not patent. (Adapted from Patel et al. [1] with permission from Springer Nature). The number in parentheses next to the rating reflects the median score for that indication. *Substitution of a newer coronary pressure ratio that does not require hyperemia for FFR may be considered provided the appropriate reference values are used. A = appropriate; AA = Antianginal; BB = beta-blockers; CABG = coronary artery bypass graft; FFR = fractional flow reserve; IMA = internal mammary artery; LAD = left anterior descending coronary artery; M = may be appropriate; PCI = percutaneous coronary intervention; and R = rarely appropriate)

		Asymptomatic				Ischemic Symptoms			
		Not on AA Therapy or With AA Therapy		Not on AA Therapy		On 1 AA Drug (BB Preferred)		On ≥2 AA Drugs	
	Indication	PCI	CABG	PCI	CABG	PCI	CABG	PCI	CABG
Patients Undergoing Renal Transplantation, No Diabetes									
42.	▪ One- or two-vessel CAD, no proximal LAD inclvement, with low-risk noninvasice findings	R (3)	R (2)	M (4)	R (3)	M (6)	M (4)	A (7)	M (5)
43.	▪ One- or two-vessel CAD, no proximal LAD inclvement, with intermediate- or high-risk noninvasice findings	M (5)	M (4)	M (6)	M (4)	A (7)	M (5)	A (8)	M (6)
44.	▪ One- or two-vessel CAD, including proximal LAD, with low-risk noninvasice findings	M (5)	M (4)	M (6)	M (5)	M (6)	M (6)	A (8)	A (7)
45.	▪ One- or two-vessel CAD, including proximal LAD, with intermediate- or high-risk noninvasice findings	M (6)	M (6)	A (7)	A (7)	A (7)	A (7)	A (8)	A (8)
46.	▪ Left main and/or three-vesse; disease, with intermediate- or high-risk noninvasive findings (e.g., SYNTAX ≤22)	M (6)	A (7)	A (7)	A (7)	A (7)	A (7)	A (8)	A (8)
47.	▪ Left main and/or three-vesse; disease, with intermediate- or high-risk noninvasive findings (e.g., SYNTAX >22)	M (5)	A (7)	M (6)	A (8)	M (6)	A (8)	M (6)	A (9)
Patients Undergoing Renal Transplantation, Diabetes Present									
48.	▪ One- or two-vessel CAD, no proximal LAD inclvement, with low-risk noninvasice findings	R (3)	R (3)	M (4)	R (3)	M (5)	M (4)	A (7)	M (6)
49.	▪ One- or two-vessel CAD, no proximal LAD inclvement, with intermediate- or high-risk noninvasice findings	M (5)	M (4)	M (5)	M (4)	A (6)	M (5)	A (7)	A (7)
50.	▪ One- or two-vessel CAD, including proximal LAD, with low-risk noninvasice findings	M (5)	M (5)	M (5)	M (6)	M (5)	A (7)	A (7)	A (7)
51.	▪ One- or two-vessel CAD, including proximal LAD, with intermediate- or high-risk noninvasice findings	M (6)	M (6)	M (6)	A (7)	M (6)	A (7)	A (7)	A (8)
52.	▪ Left main and/or three-vesse; disease, with intermediate- or high-risk noninvasive findings (e.g., SYNTAX ≤22)	M (6)	A (8)	M (6)	A (8)	M (6)	A (8)	A (7)	A (9)
53.	▪ Left main and/or three-vesse; disease, with intermediate- or high-risk noninvasive findings (e.g., SYNTAX >22)	M (5)	A (8)	M (5)	A (8)	M (5)	A (9)	M (5)	A (9)
Patients Who Will Undergo a Percutaneous Valve Procedure (TAVR, MitraClip, Others)									
54.	▪ One- or two-vessel CAD, no proximal LAD inclvement, with low-risk noninvasice findings	M (4)		M (4)		M (6)		A (8)	
55.	▪ One- or two-vessel CAD, no proximal LAD inclvement, with intermediate- or high-risk noninvasice findings	A (7)		A (7)		A (7)		A (8)	
56.	▪ One- or two-vessel CAD, including proximal LAD, with low-risk noninvasice findings	M (6)		M (6)		A (7)		A (8)	
57.	▪ One- or two-vessel CAD, including proximal LAD, with intermediate- or high-risk noninvasice findings	A (7)		A (7)		A (8)		A (9)	
58.	▪ Left main and/or three-vesse; disease, with intermediate- or high-risk noninvasive findings (e.g., SYNTAX ≤22)	A (8)		A (8)		A (8)		A (9)	
59.	▪ Left main and/or three-vesse; disease, with intermediate- or high-risk noninvasive findings (e.g., SYNTAX >22)	A (7)		A (7)		A (8)		A (8)	

Fig. 6.3 AUC for stable ischemic heart disease undergoing procedures for which coronary revascularization may be considered. (Adapted from Patel et al. [1] with permission from Springer Nature). The number in parentheses next to the rating reflects the median score for that indication. A = appropriate; AA = Antianginal; BB = beta-blockers; CABG = coronary artery bypass graft; CAD = coronary artery disease; LAD = left anterior descending coronary artery; M = may be appropriate; PCI = percutaneous coronary intervention; R = rarely appropriate; SYNTAX = Synergy between PCI with Taxus and Cardiac Surgery trial; and TAVR = transcatheter aortic valve replacement)

Appropriate Use Criteria for Acute Coronary Syndrome

In developing these AUC for coronary revascularization in patients with acute coronary syndrome (ACS), a panel of experts scored each indication using the following definition of appropriate use*: A coronary revascularization or antianginal therapeutic strategy is appropriate care when the potential benefits, in terms of survival or health outcomes (symptoms, functional status, and/or quality of life) exceed the potential negative consequences of the treatment strategy*. The panel scored each indication on a scale from one to nine based on their level of agreement with the definition of appropriate use for that indication as follows [2]:

- Score 1 to 3: Rarely appropriate care (risks > benefit).
- Score 4 to 6: May be appropriate care (potential benefit).
- Score 7 to 9: Appropriate care (risk < benefit).

In patients with ACS, AUC indications for coronary revascularization were developed considering several variables [2]:

- Clinical presentation (STEMI, NSTEMI, or other ACS).
- Time from onset of symptoms.
- Presence of other complicating factors (severe heart failure or cardiogenic shock, hemodynamic or electrical instability, presence of left ventricular dysfunction, persistent or recurring ischemic symptoms).
- Prior treatment with fibrinolytics.
- Predicted risk as estimated by the Thrombolysis In Myocardial infarction score.
- Relevant comorbidities.
- Extent of anatomic disease in the culprit and non-culprit arteries.

Determining the significance of coronary stenosis in ACS includes not only the percent luminal diameter narrowing but also the angiographic appearance of the stenosis and distal flow pattern. Per AUC, coronary stenosis in ACS was defined as follows [2]:

- Severe:
  - ≥70% luminal diameter narrowing of epicardial stenosis, measured by visual assessment in the "worst view" angiographic projection.
  - ≥50% luminal diameter narrowing of the left main artery, measured by visual assessment in the "worst view" angiographic projection.
- Intermediate:
  - ≥50% and <70% diameter narrowing of epicardial stenosis, measured by visual assessment in the "worst view" angiographic projection.

Refer to Fig. 6.4 for a summary of AUC for ACS.

a

	Indication	Appropriate Use Score (1-9)
Revascularization of the Presumed Culprit Artery by PCI (Primary PCI)		
1.	■ Less than or equal to 12 hours from onset of symptoms	A (9)
2.	■ Onset of symptoms within the prior 12-24 hours AND ■ Severe HF, Persistent ischemic symptoms, or hemodynamic or electrical instability present	A (8)
3.	■ Onset of symptoms within the prior 12-24 hours AND ■ Stable without severe HF, persistent ischemic symptoms, or hemodynamic or electrical instability	M (6)
Successful Treatment of the Culprit Artery by Primary PCI Followed by Immediate Revascularization of 1 or More Nonculprit Arteries During the Same Procedure		
4.	■ Cardiogenic shock persisting after PCI of the presumed culprit artery ■ PCI or CABG of 1 or more additional vessels	A (8)
5.	■ Stable patient immediately following PCI of the presumed culprit artery ■ One or more additional severe stenoses	M (6)
6.	■ Stable patient immediately following PCI of the presumed culprit artery ■ One or more additional intermediate (50%-70%) stenoses	M (4)

Fig. 6.4 (**a**) AUC for STEMI immediate revascularization by PCI. (**b**) AUC for STEMI initial treatment by fibrinolytic therapy. (**c**) AUC for STEMI revascularization of the non-culprit artery (D) AUC for NSTEMI/Unstable angina. (Adapted from Patel et al. [2] with permission from Springer Nature). The number in parenthesis next to the rating reflects the median score for that indication. A = appropriate; CABG = coronary artery bypass graft; FFR = fractional flow reserve; HF = heart failure; M = may be appropriate; NSTEMI = non–ST-segment elevation myocardial infarction; PCI = percutaneous coronary intervention; R = rarely appropriate; STEMI = ST-segment elevation myocardial infarction; TIMI = Thrombolysis In Myocardial Infarction)

b

Indication	Appropriate Use Score (1-9)
PCI of the Presumed Culprit Artery After Fibrinolsis	
7. ■ Evidence of failed reperfusion after fibrinolysis (e.g., failure of ST-segment resloution, presence of acute severe HF, ongoing myocardial ischemia, or unstable ventriculer arrhythmias)	A (9)
8. ■ Stable after fibrinolysis AND ■ Asymptomatic (no HF, myocardial ischemia, or unstable ventricular arrhythmias) AND ■ PCI performed 3-24 hours after fibrinolytic therapy	A (7)
9. ■ Stable after fibrinolysis AND ■ Asymptomatic (no HF, myocardial ischemia, or unstable ventricular arrhythmias) AND ■ PCI >24 hours after onset of STEMI	M (5)

c

Indication	Appropriate Use Score (1-9)
Successful Treatment of the Culprit Artery by Primary PCI or Fibrinolysis Revascularization of 1 or More Nonculprit Arteries During the Same Hospitalization	
Revascularization by PCI or CABG	
10. ■ Spontaneous or easily provoked symptoms of myocardial ischemia ■ One or more additional severe stenoses	A (8)
11. ■ Asymptomatic ■ Findings of ischemia on noninvasive testing ■ One or more additional severe stenoses	A (7)
12. ■ Asymptomatic (no additional testing performed) ■ One or more additional severe stenoses	M (6)
13. ■ Asymptomatic (no additional testing performed) ■ One or more additional intermediate stenoses	R (3)
14. ■ Asymptomatic ■ One or more additional intermediate (50%-70%) stenoses ■ FFR performed and ≤0.80	A (7)

d

Indication	Appropriate Use Score (1-9)
Revascularization by PCI or CABG	
15. ■ Evidence of cardiogenic shock ■ Immediate revascularization of 1 or more coronary arteries	A (9)
16. ■ Patient stabilized ■ Intermediate- OR high-risk features for clinical events (e.g., TIMI score 3-4) ■ Revascularization of 1 or more coronary arteries	A (7)
17. ■ Patient stabilized after presentation ■ Low-risk features for clinical events (e.g., TIMI score ≤2) ■ Revascularization of 1 or more coronary arteries	M (5)

Fig. 6.4 (continued)

Pearls

- More than one treatment may be considered "Appropriate," "May Be Appropriate," or "Rarely Appropriate" for any clinical indication and a shared decision approach should be undertaken to determine which treatment is suitable.
- Rating of "appropriate care" does not mandate that a revascularization procedure be performed; similarly, a rating of "rarely appropriate care" should not prevent a revascularization procedure from being performed.
- If a clinician decides not to follow the AUC rating, the provider should document case-specific details that are not accounted for, and the rationale for choosing an alternative treatment plan.
- AUC only covers clinical scenarios where the culprit artery and additional non-culprit arteries are treated at the time of primary PCI, or later during the initial hospitalization.
- Consider a heart team evaluation when faced with complex clinical scenarios or patients with severe CAD.

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Guide Catheter Selection

7

Gurpreet S. Johal, Amit Hooda, and Samin K. Sharma

Introduction

Optimal guide support is essential for successful interventions. An ideal guide catheter has an atraumatic soft tip, excellent torque control, adequate support, and low surface frictional resistance that allows devices to track with ease.

Guide Catheters

Guiding Catheter Layers

- The outer layer is polyurethane or polyethylene, and is responsible for overall stiffness.
- The middle layer is composed of a wire matrix and is responsible for torque generation.
- The inner layer is composed of Teflon for the smooth passage of balloons, stents, and devices (Fig. 7.1).

Guide Selection

- The guide catheter is usually firmly supported against the aortic wall opposite to the coronary sinus from which the artery arises.

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https://doi.org/10.1007/978-3-030-68538-6_7

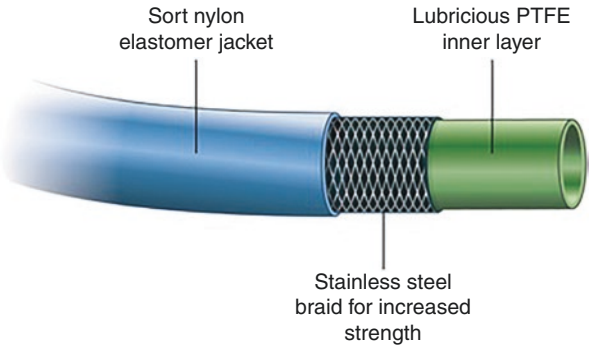


Fig. 7.1 Guiding catheter layers

- Selection is dependent on:
  - Side holes.
  - French sizes (Fr).
  - Length.
  - Type of curve (anatomy based: size of the aortic root, ostial origin, and takeoff).
  - Support.
  - Ostial origin of the coronary (normal or anomalous origin of coronary vessels).

Step One: Side Holes

- Side holes prevent ventricularization (fall of diastolic pressure) or dampening (fall of both systolic and diastolic pressure) caused by engagement of guide in significant ostial lesions, misalignment of guides, during coronary spasm, or when a large Fr guide is used for engagement of a smaller coronary artery (Fig. 7.2).
- Refer to Table 7.1 for the use, advantages, and disadvantages of side holes.

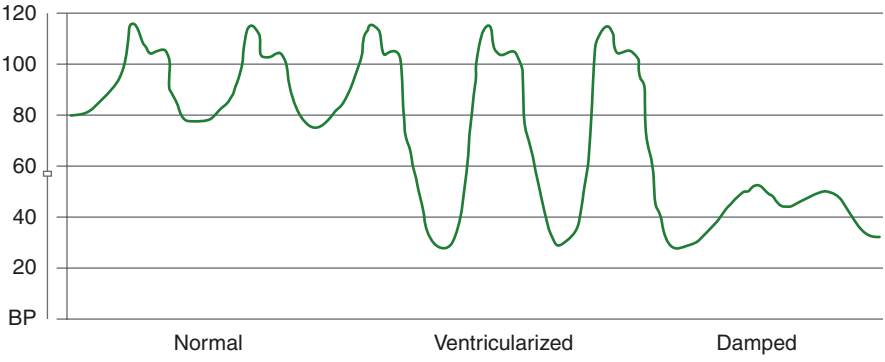


Fig. 7.2 Ventricularization and damping

Table 7.1 Side holes in guide catheters: use, advantages, and disadvantages

Advantages	Disadvantages
Maintains coronary artery perfusion	A false sense of security as it monitors aortic, not coronary pressure
	Suboptimal opacification of the coronary artery
	Increased contrast usage

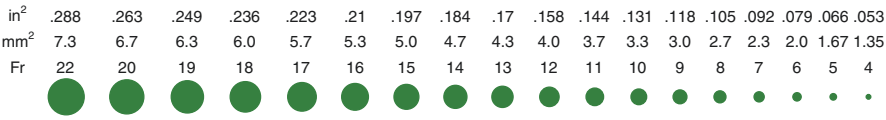


Fig. 7.3 French sizes

Table 7.2 Selecting an appropriately sized guide catheter for an interventional procedure

Guide catheter size (Fr)	Description
6 Fr	Usually sufficient for most interventions, two-stent strategy for bifurcation lesions [DK Crush technique, Culotte technique], bailout stent technique [TAP technique, Reverse Crush (internal) technique], including orbital atherectomy or rotational atherectomy burr <2 mm
7 Fr	Two-stent strategy for bifurcation lesions [Minicrush technique, modified T technique, SKS technique, V technique], or rotational atherectomy burr of 2 mm
8 Fr	Rotational atherectomy burr of 2.15 or 2.25 mm

Step Two: French Size

- Ideally, use the smallest diameter catheter feasible to minimize the risk of arterial damage (Fig. 7.3).
- Larger French catheters have the advantage of improved opacification, better guide support, and allow for pressure monitoring but increase the risk of ostial trauma, vascular complications, and contrast nephropathy.
- Refer to Table 7.2 for selecting the appropriate size of a guide catheter for a particular intervention.

Step Three: Guide Length

- Regular 100 cm guides will suffice for most coronary interventions.
- Long saphenous vein graft (SVG) or internal mammary artery (IMA) grafts interventions may require the use of short 80 or 90 cm guides.

Step Four: Guide Catheter Support

- Support is either passive or active.
  - Passive support: Depends on the guide design, stiffness, and its backup support against the opposite aortic wall or aortocoronary sinus.
  - Active support: Manipulation of the guide to conform to the aortic root or deep engagement of the guide into the coronary arteries provides active support for interventions.
- Different guide catheters provide different levels of support. The most commonly used guide catheters are the Judkins (JL/JR), Amplatz (AL/AR), Voda (VL), Internal Mammary (IM), and Extra Backup (EBU (Medtronic) or XB (Cordis)). When selecting the appropriate guide catheter, it is important to remember the level of support it provides depends on several factors, including which coronary vessel is being intervened on (guide catheters listed in order of more support to less support):
  - Left coronary artery: EBU/XB > VL > JL.
  - Right coronary artery: AL > AR/IM > JR.

Pearls

- Other maneuvers to improve guide catheter support.
  - Use a longer sheath (when performing a femoral procedure).
  - Use a larger-sized guide catheter.
  - Use a buddy wire technique.
  - Use an anchoring balloon.
  - Use a guide support extension catheter/mother-in-child techniques (i.e. Guideliner, Guidezilla, or Telescope).

Step Five: Catheter Curve

- Select a curve style that is optimal for a given anatomical configuration (Table 7.3).

Table 7.3 Size of the aortic root

Size	Left coronary artery	Right coronary artery
Normal (3.5–4 cm)	VL 3.5	IM, FR, AR2, AL 0.75
Large (>4 cm)	VL 4, VL 4.5	IM, AL 1, AL 2
Small (<3.5 cm)	VL 3	IM, FR

Step Six: Knowledge of Vascular Anatomy/Ostial Origin of Coronary Arteries

- LM usually arises anterior, inferior, and leftward from the left coronary sinus.
- LAD usually arises anteriorly and superiorly from the LM.
- LCX usually arises posteriorly and inferiorly from the LM.
- RCA usually arises anteriorly from the right aortic cusp.
- SVGs usually arise anteriorly from the aorta.

Coronary Ostial Origin and Orientation

Classification of Coronary Ostial Origin and Orientation

- Coronary ostial 'origin' is classified as (Fig. 7.4 and Table 7.4):
  - Normal, High, or low.
  - Anterior or posterior.

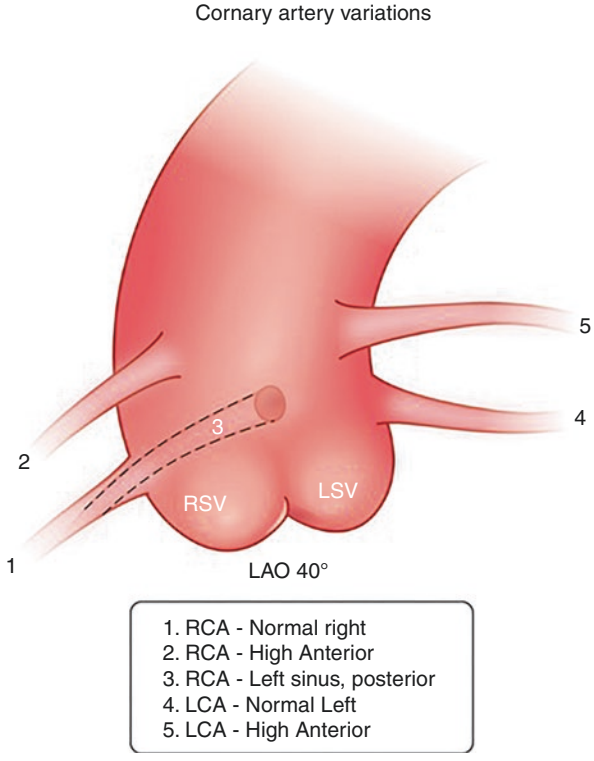


Fig. 7.4 Ostial origins of the coronary arteries

Table 7.4 Guide selection depending on the origin of the coronary ostia

Origin	Left coronary artery	Right coronary artery
Anterior	–	NoTo, 3DRC, AR2 or AL0.75 (dilated aortic root)
Shepherd's crook	–	SCR, HS, IMA
Short left main	FL, FCL, VL3	–
Long left main	VL4	–

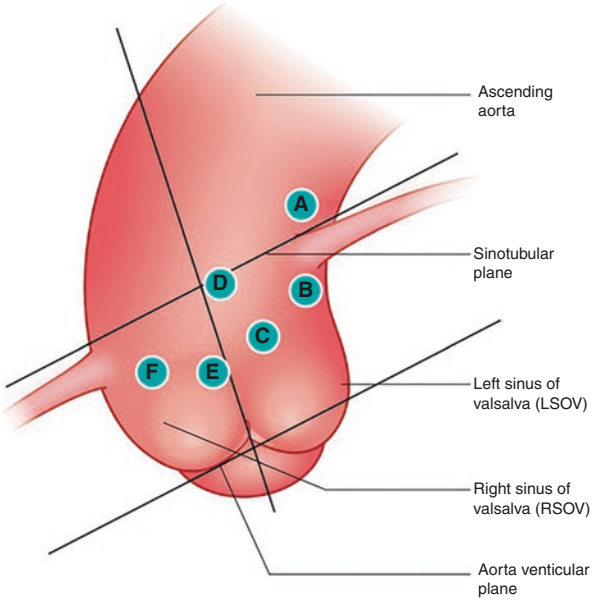


Fig. 7.5 Anomalous RCA origins

- Coronary ostial 'orientation' is classified as (Fig. 7.4 and Table 7.4):
  - Superior, inferior, or horizontal.
  - Shepherd's crook (RCAs only).

Catheter Selection for Anomalous Right Coronary Artery

Anomalous RCA Catheter Selection

- In the LAO view, draw an imaginary line at the upper edge of the bulge that marks the plane dividing the aortic sinuses from the ascending aorta.
- A similar line drawn along the lower edge of this bulge divides the aortic cusps from the ventricular outflow tract.
- Finally, a line is drawn along the long axis of the ascending aorta intersecting the sinus aortic and aorto-ventricular planes perpendicularly.
- The origin of the anomalous vessel is described based on its location relative to these landmarks (Fig. 7.5 and Table 7.5).

Table 7.5 Catheter selection for anomalous right coronary arteries

Type	Origin	First choice	Second choice
From left cusp			
A	From the aorta above the sinotubular plane	FL3	FCL3
B	Just below the ostium of the left coronary artery	FCL 3	FCL 3.5,VL3.5
C	Below the sinotubular plane between the midline and the origin of the left coronary artery	VL 3.5	FCL3.5
D	Origin near the midline	AL1	AL 0.75, AL2, AL3
From right cusp			
E	Origin near the midline	AR2	AR 1
F	Anterior origin	3D RC	AR2, AR1

Different Guide Catheters Available

Types of Guide Catheters

- Refer to Fig. 7.6 for the different guide catheter shapes, sizing, and unique characteristics.

Pearls

- Our first go-to guide for coronary interventions is as follows:
  - Left coronary intervention: VL.
  - Right coronary intervention: IM (ostial or proximal RCA) and AL (mid to distal RCA, or RPDA).
  - SVG intervention: IM, AL0.75, AL1, or AR2.

Left coronary guides

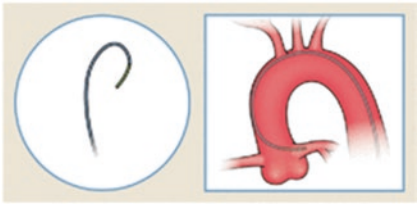
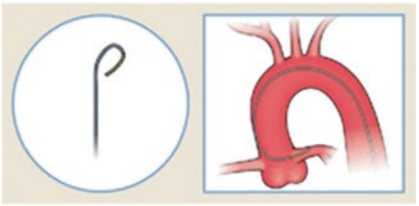
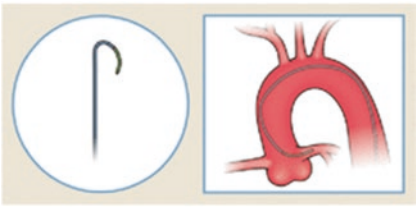
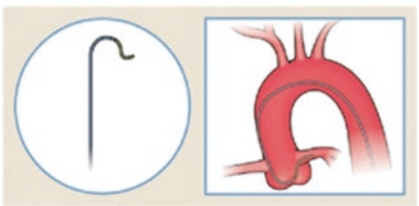
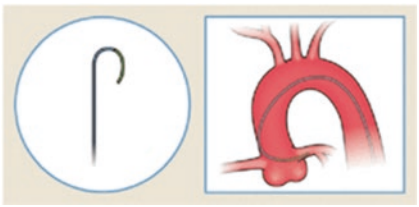
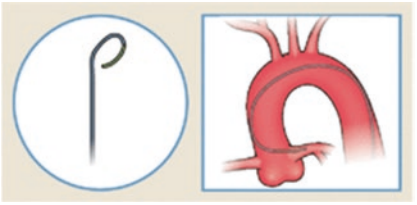
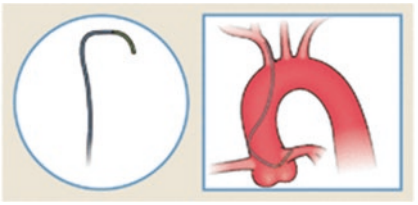
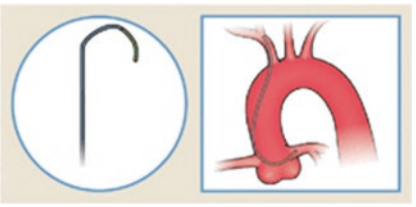
Name	Curve	Sizing/unique characteristics	Comparable to available in:
Left back up curve		– Downsize by a half size from JL curve – Provides large area of support on the contralateral wall. Designed for excellent support and control when engaging the LCA.	Comparable to: XB / XBLAD / EBU Available in: Convey guide Runway guide
CLS up curve		– Downsize by a half size from JL curve – Provides large area of support on the contralateral wall directly opposite the ostium.	Comparable to: XB / XBLAD / EBU Available in: Convey guide Mach1 guide Runway guide Wiseguide
Judkins left curve (FL)		– Standard sizing	Comparable to: JL/FL Available in: Convey guide Mach1 guide Runway guide
Amlatz left curve (AL)		– Standard sizing – Also used for RCA interventions, particularly high anterior RCA	Comparable to: JL/FL Available in: Expo diagnostic Impulse diagnostic Convey guide Mach1 guide Runway guide Wiseguide
Q curve		– Standard sizing – Designed to sub-select LAD	Available in: Mach1 guide Runway guide Wiseguide

Fig. 7.6 Guide catheter shapes, sizing, and unique characteristics

Voda left curve		– Downsize by a half size from JL curve – Backup support from opposite aortic wall. – Excellent for difficult LCA anatomy (tortuous, calcified, total occlusion)	Available in: Mach1 guide Convey guide Runway guide
TIG curve		– Standard sizing	Comparable to: Tiger Available in: Convey guide
Radial back up curve (RB)		– One size fits all	Comparable to: Radial brachial MRADIAL Available in: Convey guide

Right coronary guides

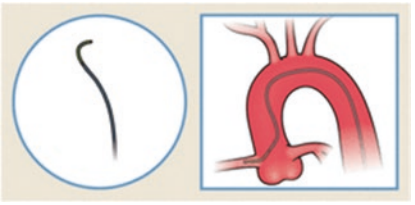
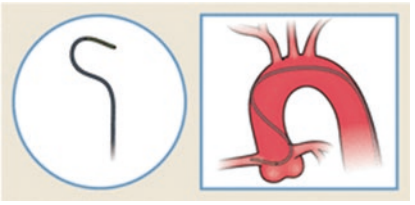
Name	Curve	Sizing/unique characteristics	Comparable to available in:
Femoral right curve (FR)		– Standard sizing	Comparable to: JR Available in: Expo diagnostic Impulse diagnostic Convey guide Runway guide Wiseguide Mach guide
Allright curve (ART)		– Downsize by 1/2 form JR curve – Contralateral support for most RCAs with normal to superior take off	Comparable to: XBRCA / RBU Available in: Convey guide Mach1 guide Wiseguide Runway guide

Fig. 7.6 (continued)

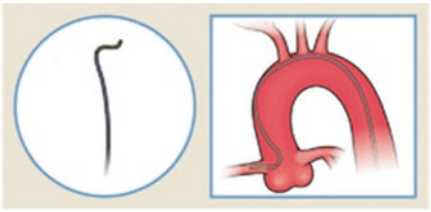
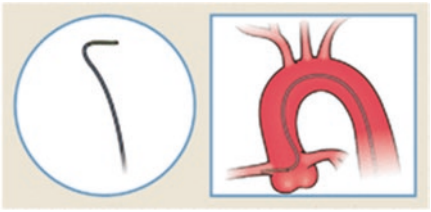
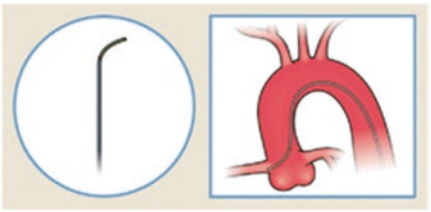
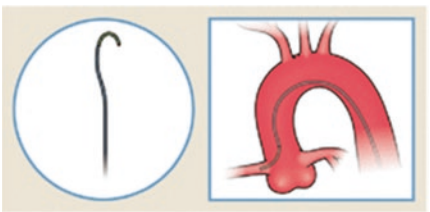
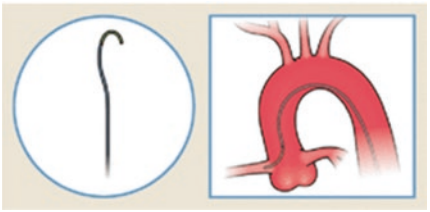
Amplatz right curve (AR)		– Standard sizing – Vein graft to the RCA with interior take off – Interventions to RCA vein grafts (especially inferior take off)	Available in: Expo diagnostic Impulse diagnostic Convey guide Mach1 guide Runway guide Wiseguide
Hockey Stick curve (HS)		– One size fits all – Interventions to vein graft to LCA	Comparable to: H-stick / HS1 Available in: Convey guide Mach1 guide Runway guide
Multi purpose curve (MP)		– Standard sizing – Vein graft interventions to the LCA with inferior or horizontal take off	Comparable to: MPA / MBF Available in: Expo diagnostic Impulse diagnostic Convey guide Mach1 guide Runway guide Wiseguide
Right Coronary Shepherd's Crook Curve (RC)		– Standard sizing – Excellent support for difficult Shepherd's Crook RCA anatomy	Comparable to: SCR Available in: Convey guide Mach1 guide Runway guide Wiseguide
Williams Right Posterior Curve (WRP)		– One size fits all – No torque right. Minimal catheter manipulation required. – Three dimensional for added support	Available in: Expo diagnostic Runway guide

Fig. 7.6 (continued)

Bypass graft guides

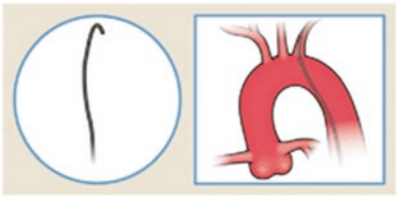
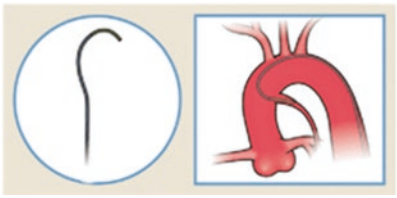
Name	Curve	Sizing/unique characteristics	
Internal Mammary Curve (IM)		– One size fits all – Incorporates a large radius primary curve designed to ease device passage. – Provides contralateral support from the subclavian artery	Available in: Expo diagnostic Impulse diagnostic Convey guide Mach1 guide Runway guide Wiseguide
Left Coronary Bypass Curve (LCB)		– One size fits all – Similar to JR and HS. – Useful for vein grafts to the LCA with horizontal or slightly superior take off	Available in: Expo diagnostic Convey guide Mach1 guide Runway guide Wiseguide

Fig. 7.6 (continued)

Guidewire Properties and Selection

8

Rohit Malhotra, Gurpreet S. Johal, and Samin K. Sharma

Introduction

The inherent properties of support and steerability of a wire to deliver coronary devices through various coronary anatomies are paramount for coronary interventions.

A wire that can support and enable easy steerage of coronary devices through tortuous and calcified coronary anatomies is key to a successful coronary intervention. The basic construct of the coronary guidewire imparts it the following key properties:

- Trackability through a vessel.
- Lesion access.
- Atraumatic lesion crossing.
- Support to deliver complex interventional devices.

While there are numerous guidewires available, the key characteristics of the guidewire structure and design remain consistent across manufacturers.

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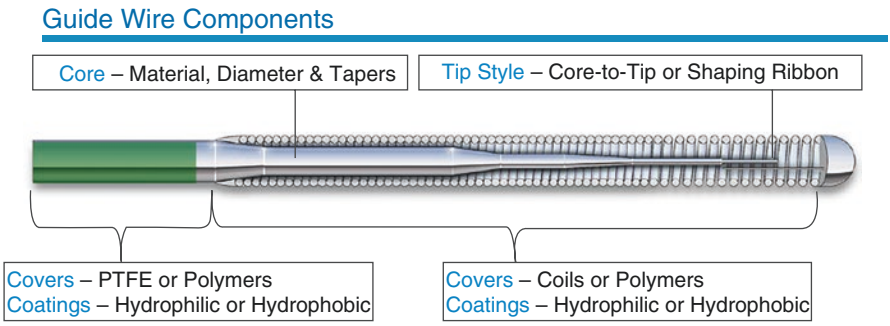

Fig. 8.1 Guidewire structure. (Courtesy of Abbott Vascular. ©2014 Abbott. All Rights Reserved)

Structure of the Guidewire

There are three main components of the guidewire (Fig. 8.1):

- Core.
- Tip.
- Covering.

Core

- The Core is the innermost part of the guidewire and it extends through the shaft of the wire from the proximal to the distal end, where it begins to taper. The core material affects the steering, trackability, as well as flexibility, and support; on the other hand, the diameter of the core influences the flexibility, support, and torque.
  - Core Material:
    The two most common core materials are Stainless Steel and Nitinol (a super-elastic alloy of nickel and titanium with excellent resilience and kink resistance).
    - Stainless steel (SS):
      - Strengths: provide optimal support, force and torque transmission, and good shapeability.
      - Weakness: more susceptible to kinking.
    - Nitinol (NT):
      - Strengths: It provides excellent flexibility and steerability and is more durable than stainless steel. Due to its ability to resist deformation, it is often used for the treatment of multiple lesions and tortuous anatomy.
      - Weakness: less torqueability than stainless steel.
  - Core diameter:
    The diameter of the core influences the performance of the wire. A wider bore improves support and allows for 1:1 torque response, while narrower diameters have the opposite effect and enhance the flexibility of the wire.

- Core taper:
  The core of the guidewire is tapered along its length. The taper may be continuous or segmental and variable in length. Shorter tapers or smaller numbers of widely spaced gradual tapers, enhance the support and transmission of push force, while longer tapers and larger numbers of more segmental tapers enhance the flexibility.

Tip

- The tip refers to the distal end of the guidewire. There are two styles of guidewire tips, namely core to tip tapers and shaping ribbons.
  - Core to tip design:
    The most commonly used wires have a one-piece core where the core extends to the tip with a variable taper. These wires are engineered to possess precise steering and tip control and a soft tip.
  - Two-piece or shaping ribbon:
    The core stops just before the distal tip. A shaping ribbon (a small piece of metal) bridges the gap between the end of the core and the distal tip, which allows for a very flexible, soft, atraumatic tip with easy shapability, and good shape retention. However, compared to other designs these wires have less reliable torque control and a higher likelihood to prolapse.

Surface Coating

- To maintain the overall diameter of 0.014 inches, all tapered cores and guidewire tips have a specific surface coating. The tip could be covered with coils (spring-tip guidewires) or polymer (polymer-tip guidewires).
  - Coils:
    A stainless steel coil is the outermost covering of a wire. In an outer coil design, coils are placed over the tapered core and tip of the wire as opposed to the tip-coil design (shaping ribbon) where the tip alone is covered with coils. These coils add flexibility to the distal part of the wire as well as support, steerability, trackability, and visibility. Visibility of the wire tip is provided by radiopaque platinum coils which are usually placed at the distal tip (generally 2–3 cm long). Coils placed on the working length of the wire are referred to as intermediate coils.
  - Covers:
    Some wires have polymer or plastic covering over the tapered wire core instead of outer coils. The use of polymer or plastic covering provides the wire with lubricity and enables smooth tracking through tortuous and calcified anatomy.
  - Coating:
    The coating refers to the outer covering that keeps the overall diameter consistent and influences the wire performance. The type and length of the coating may vary and are most often applied to the distal 30 cm of the wire. Two types of coatings are used:

- Hydrophilic coatings attract water and are applied along the entire length of the wire including the tip coils. When dry, the coating is solid, thin, and non-slippery solid. Upon contact with liquids, the coating becomes a slippery "waxlike" surface that reduces friction and increases trackability. While providing for a lubricous low-friction motion inside the vessel, these wires should be utilized judiciously as they carry the risk of subintimal penetration, dissection, and perforation of the coronary artery.
- Hydrophobic coatings are silicone-based coatings that repel water. It is applied to the working length wire except for the distal tip. The silicone coating has higher-friction, is more stable inside the vessel, and is not activated by liquids.

Classification of Guidewires

All guidewires can be classified based on three core properties of each wire; these are:

- Tip flexibility.
- Device support.
- Coating type.

Given the high number of guidewires, each interventional cardiologist needs to familiarize themselves with guidewires based on the lesion characteristics being tackled. Accordingly, we would recommend the following 4 basic types of guidewires:

- Workhorse.
- First-line finesse.
- Extra Support.
- Specialty.

The desirable characteristics of each guidewire are as below (Table 8.1).

Table 8.1 Basic classification of guidewires

WORKHORSE	FIRST LINE FINESSE	EXTRA SUPPORT	SPECIALTY
• Safe/ atraumatic tip • 1:1 torque transmission • Shape retention • Durability • Moderate support	• 1:1 torque transmission • Trackability through tortuos lesions • Lubricity with tactile feedback • Moderate support	• Gentle Tip • Extra support to facilitate device delivery to distal/ tortuos segments.	• Variable tip load/ tapers for crossability. • Moderate support. • Safe/ atraumatic tips

Selection of Guidewires

The selection of a guidewire should be primarily determined by vessel and lesion characteristics, and device properties. For example, a highly tortuous/ calcified segment may require a more lubricious guidewire with a highly durable tip. Similarly, distal vessel access/ device delivery might require additional support hence an appropriate guidewire must be considered.

While there are many guidewires available from different vendors, each with similar/ overlapping properties, it is imperative to familiarize oneself with 1–2 guidewires needed for various lesion types. Also, given the overlap in guidewire properties, most successful, high-volume cath labs have a finite number of guidewires available on the shelf, which helps increase provider familiarity and improve the chances of successful lesion interventions.

Guidewires for Standard Lesion Morphology

- A standard lesion is defined by the absence of complex characteristics (i.e., heavy calcification, tortuous segments, and total occlusions). A "workhorse or frontline" wire is most suitable for standard lesions (Table 8.2). The workhorse wire, which accounts for about 70% of all coronary wires used, is a floppy wire with an atraumatic tip, which provides low to moderate support.
- The preferred workhorse wire at the Mount Sinai Cardiac Catheterization Laboratory is currently the Terumo Runthrough NS wire. Unlike its stainless steel wire predecessors (Balance Middleweight and Prowater wires), it has a unique core design with the main shaft core of stainless steel and a distal core of nitinol alloy which extends into a nitinol shaping ribbon. The distal tip is hydrophilic coated. These properties give it a better steerability, which allows for easier navigation through difficult angulated lesions.

Table 8.2 Workhorse wires in the Mount Sinai Cath Lab

Guidewire	Tip load (g)	Radiopaque length (cm)	Tip diameter	Polymer cover	Coating
Runthrough NS	1	3	0.014	Full	Hydrophilic
Balance middleweight wire	0.8	3	0.014	None	Hydrophilic
Whisper ES	1.2	3	0.014	Full	Hydrophilic

Guidewires for Chronic Total Occlusions

- For the more complex lesions, particularly chronic total occlusions (CTO) , a stiffer wire with increasing support may be required (Tables 8.3 and 8.4).

Table 8.3 CTO wires (by name)

Guidewire	Tip load	Radiopaque length	Tip diameter	Polymer cover	Coating
Fielder	3.7	3	0.014	Full	Hydrophilic
Fielder FC	1.6	3	0.014	Full	Hydrophilic
Fielder XT	1.2	16	0.009	Full	Hydrophilic
MiracleBros 3	3.9	11	0.0125	None	Hydrophobic
MiracleBros 4.5	4.4	11	0.0125	None	Hydrophobic
MiracleBros 6	8.8	11	0.0125	None	Hydrophobic
MiracleBros 12	13	11	0.0125	None	Hydrophobic
Confianza 9	8.6	20	0.009	None	Hydrophobic
Confianza PRO 9	9.3	20	0.009	None	Hybrid
Confianza PRO 12	12.4	20	0.009	None	Hybrid
PROGRESS 40	4.8	3	0.012	Tip only	Hydrophilic
PROGRESS 80	9.7	3	0.012	Tip only	Hydrophilic
PROGRESS 120	13.9	3	0.012	Tip only	Hydrophilic
PROGRESS 140 T	12.5	3	0.0105	Tip only	Hydrophilic
PROGRESS 200 T	13.3	3	0.009	Tip only	Hydrophilic
HT PILOT 200	4.1	3	0.014	Full	Hydrophilic
HT PILOT 150	2.7	3	0.014	Full	Hydrophilic
HT PILOT 50	1.5	3	0.014	Full	Hydrophilic

Table 8.4 CTO wires (by tip load)

Guidewire	Tip load	Radiopaque length	Tip diameter	Polymer cover	Coating
Fielder XT	1.2	16	0.009	Full	Hydrophilic
HT PILOT 50	1.5	3	0.014	Full	Hydrophilic
FIELDER FC	1.6	3	0.014	Full	Hydrophilic
HT PILOT 150	2.7	3	0.014	Full	Hydrophilic
Fielder	3.7	3	0.014	Full	Hydrophilic
MiracleBros 3	3.9	11	0.0125	None	Hydrophobic
HT PILOT 200	4.1	3	0.014	Full	Hydrophilic
MiracleBros 4.5	4.4	11	0.0125	None	Hydrophobic
PROGRESS 40	4.8	3	0.012	Tip only	Hydrophilic
Confianza 9	8.6	20	0.009	None	Hydrophobic
MiracleBros 6	8.8	11	0.0125	None	Hydrophobic
Confianza PRO 9	9.3	20	0.009	None	Hybrid
PROGRESS 80	9.7	3	0.012	Tip only	Hydrophilic
Confianza PRO 12	12.4	20	0.009	None	Hybrid
PROGRESS 140 T	12.5	3	0.0105	Tip only	Hydrophilic
MiracleBros 12	13	11	0.0125	None	Hydrophobic
PROGRESS 200 T	13.3	3	0.009	Tip only	Hydrophilic
PROGRESS 120	13.9	3	0.012	Tip only	Hydrophilic

Technical Tips

Guidewire Manipulation

- Guidewire manipulation to successfully cross lesions involves the following steps:
  - Successful engagement of the target vessel:
    An appropriate tip shape helps with vessel engagement; a mis-shapen tip can also hamper proper engagement. Shaping a guidewire for successful vessel engagement is described below.
  - Engagement of target lesion:
    Lesion engagement/crossing is most successfully achieved by torquing the guidewire once the tip engages the lesion. Based on lesion characteristics guidewire choice may differ, but fine wire movements are primarily similar.
  - Crossing the lesion with ample distal purchase for successful device delivery:
    Distal vessel access after lesion crossing helps in successful device delivery. It is important to have the stiff/ non-tapered shaft of a guidewire across a lesion, which can be angiographically seen after the entire radio-opaque tip has crossed a lesion. In rare instances, especially with highly angulated proximal lesions or very distal lesions, device delivery could be attempted over the flexible tip/ radio-opaque wire segments with utmost caution.

Shaping the Wire Tip

- Shape the tip using the shaping needle that comes with the wire or the introducer needle. Usually, a simple J curve with a distal bend that approximates the vessel diameter will allow the guidewire to track through the vessel. Careful manipulation of the guidewire during shaping is warranted to avoid damaging the structure and integrity of the guidewire (Fig. 8.2).

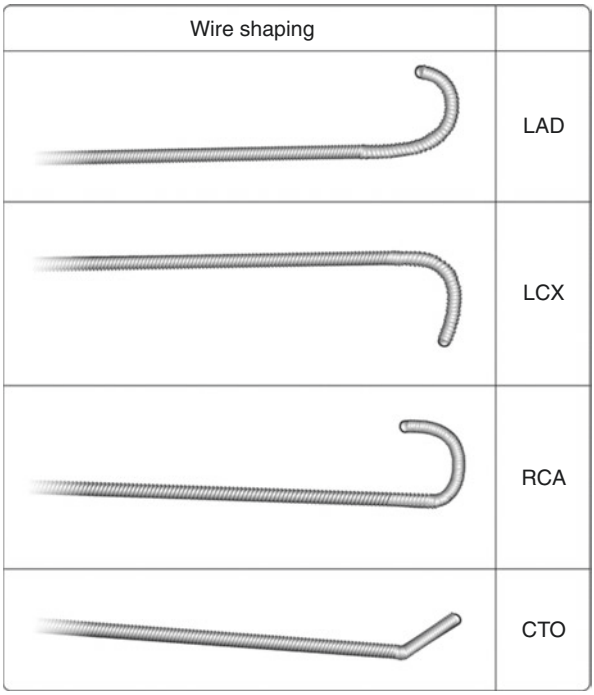

Fig. 8.2 Shaping of the guidewire

Steering

- When steering the wire through the vessel, the wire should be gently advanced and passed smoothly through the stenosis. Forceful manipulation can disrupt plaque, causing thrombus formation and acute occlusion. While advancing the guidewire, a repeated rotation of 180° in clockwise and counterclockwise directions will reduce the likelihood of sub-selection of unwanted small branches. One should avoid a complete 360° rotation as this may result in tip fracture or entanglement with a second wire. The wire tip should be placed as distally as possible, so the stiff part of the wire is across the lesion where the stent and other interventional devices are to be tracked.

Wiring the Left Anterior Descending Artery (LAD)

- The best initial view to enter the LAD is the left anterior oblique (LAO) caudal view (spider view). Once the wire has been advanced to the proximal LAD, change to the right anterior oblique (RAO) cranial view so the wire can be traversed to mid and then distal LAD.

Wiring the Left Circumflex Artery (LCx)

- For LCx interventions, a broader tip is warranted to successfully enter the LCX and a smaller curve to cross the obtuse marginal. Wiring of the circumflex can be performed in the Antero-posterior (AP) caudal or RAO Caudal views.

Wiring the Right Coronary Artery (RCA)

- If the origin of the RCA is relatively normal, a conventional soft wire with good steerability to avoid side branches is chosen first. Initial wiring can be performed in the LAO view, using the LAO cranial view for distal RCA and Right Posterior Descending Artery (RPDA).

Better Torque Control

- When a wire is more difficult to manipulate after it passes through too many curves, advancing the balloon catheter to near the wire tip will improve wire support, torque control, and steerability. Other options include the use of stiffer wire or hydrophilic wires, which are very sleek and kink resistant. However, given their often hydrophilic coatings, they provide little tactile feedback resulting in subintimal tracking and/ or distal perforation if inadvertently advanced into a small and short branch.

Pearl

- While manipulating hydrophilic wires, carefully observe the distal tip to avoid inadvertent migration and perforation.

Wire Prolapsing

- When navigating a curve to enter an artery (e.g., from the LM into the LCX), a floppy wire may keep prolapsing into an unintended 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). 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 the wire. When navigating the LM to enter a sharp bifurcation of the LCX, perform the following maneuver.

- Apply clockwise torque on the guide so its tip will point toward the LCX ostium, especially if the LM is short.
- 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 this is unsuccessful, then remove the wire and shape the tip to conform to 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 toward the left of the patient and counterclockwise if the tip was pointing toward 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 (Fig. 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 RAO or LAO cranial view. Here the wire should move downward. Any stray to the left will point to the diagonals and the right will point to the septals.

Crossing a Stent

- If a stent needs to be re-crossed, 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. If there is subtle resistance, then 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.

Fundamentals of Intracoronary Imaging

9

Rohit Malhotra, Yuliya Vengrenyuk, and Annapoorna Kini

Introduction

Intravascular imaging is an important tool in the arsenal of each interventional cardiologist. A multitude of imaging modalities are available to help understand coronary arterial and plaque morphology. While angiography provides a two-dimensional image of a three-dimensional structure, intravascular imaging enhances understanding by providing detailed cross-sectional images. The imaging modalities used often include intravascular ultrasound (IVUS) and Optical Coherence Tomography (OCT). Near-infrared spectroscopy (NIRS) is also available, although not widely used.

While IVUS imaging is based on ultrasound wave reflection, OCT is based on infrared light transmission and reflection through different tissues. Each of these two modalities has utility in diagnostic coronary angiography and percutaneous interventions, both for stable coronary artery disease (CAD) and in cases of acute coronary syndrome (ACS).

Intravascular Ultrasound (IVUS)

IVUS imaging technique has been available in clinical practice for a long time and is the most widely used and studied technique for intravascular imaging. First commercially available in the USA in the 1980's, IVUS is increasingly being used in day-to-day practice [1].

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A. Kini, S. K. Sharma (eds.), *Practical Manual of Interventional Cardiology*,
https://doi.org/10.1007/978-3-030-68538-6_9

Principle of IVUS Imaging

- IVUS catheters use the principles of ultrasonic sound wave [20–45 Megahertz (MHz)], transmission and reflection. Sound waves, produced by a piezoelectric crystal in the IVUS catheter, propagate through the different tissues and are reflected according to the acoustic densities of each one of them.
- Imaging with IVUS technique requires a special catheter to be inserted into the coronary arteries. There are two kinds of IVUS catheters available in the USA currently.
  - Phased Array Transducers:
    Consist of multiple transducers arranged circumferentially that are activated sequentially to produce an image. For example, Eagle Eye imaging system from Philips Healthcare (Andover, Massachusetts, USA).
  - Single array (Mechanical) transducers:
    Consist of a single transducer that rotates 360° to create images of the targeted vessel segment. For example: Opticross imaging catheters (Boston Scientific, Marlborough, Massachusetts, USA) and Kodama HD imaging catheter (Acist Medical Imaging, Eden Praire, Minnesota, USA).
- The above-mentioned imaging systems use similar principles of image production and have similar axillary and longitudinal resolutions. Proprietary post-processing software is marketed by each individual company provides further improvement in image quality and definition.

Indications

- IVUS imaging can be used in cases of acute coronary syndrome and stable coronary artery disease. Current guidelines recommend IVUS use in both pre-procedure planning and post-percutaneous intervention (PCI) stent optimization.
- Pre-procedure planning:
  - Reference vessel sizing proximal and distal to the lesion.
  - Lesion characterization: Lesion complexity (e.g., plaque burden, calcification), and length, especially in bifurcations lesions.
  - To assess the need for lesion preparation/ modification.
  - Left main sizing, including assessing the significance of left main lesions. Class IIa, Level of Evidence: B [2]. Left main lumen area of 5.5 mm^2 and non-left main area of 4 mm^2 are considered significant for intervention.
  - Assessment of graft vasculopathy in transplanted hearts.

  - Assessment of Lesions with in-stent restenosis (ISR) needing evaluation for a mechanism of ISR. Class IIa, Level of Evidence: B [2].
  - Integral to performing no-contrast or ultra-low contrast percutaneous interventions in people with renal insufficiency [3].
- Post-percutaneous intervention (PCI):
  - Optimizing stent placement by assessing for under expansion and malapposition.
  - Assess proximal and distal edge for geographical miss and dissection.
  - To assess for minimal stent area (MSA) post-implantation. MSA > 6 mm^2 in non-left main vessels and MSA > 8.5 mm^2 in left main lesions is considered ideal.

Image Characteristics

- IVUS imaging has a resolution of 100–200 μm. It can help discriminate between the lumen–intima interface and, to a lesser degree, the interface between the media and intima.
- The image is acquired as a gray scale (Fig. 9.1).
- Virtual histology (VH) imaging incorporation and ChromaFlow (Eagle Eye catheter, Philips) can help with additional data, however, their interpretation is more specialized and can require advanced imaging experience.

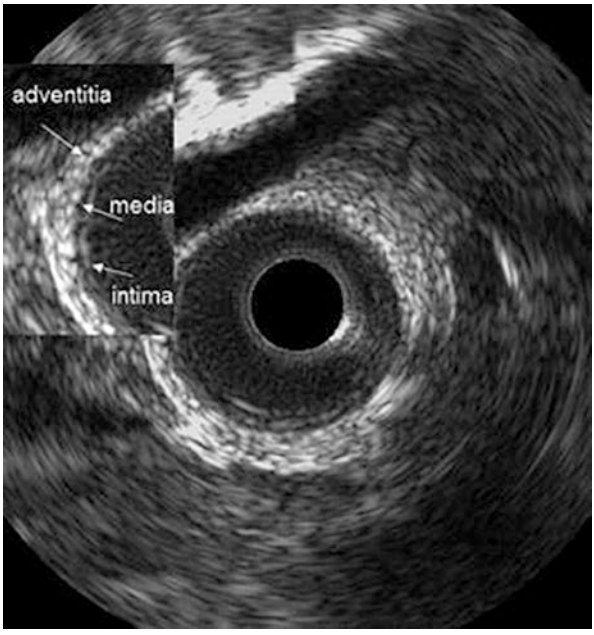

Fig. 9.1 Normal coronary artery

Image Acquisition

- Accurate IVUS image acquisition is essential for correct interpretation. Steps of IVUS imaging are as below.
  - Flush and prepare the IVUS catheter and connect to the imaging console.
  - Coronary artery engagement with the guide catheter. The guide catheter should be ≥5 French (Fr.)
  - Wiring of the desired coronary artery with a 0.014″ guidewire.
  - Flush and prepare the IVUS catheter with heparinized saline/ sterile normal saline.
  - Deliver the IVUS catheter past the targeted area of imaging. Most IVUS catheters are based on a 0.014″ monorail system.
  - Perform manual or mechanical pull back at a consistent speed of 1 m/ sec for imaging.
  - Live images are stored for post-processing and interpretation using vendor-specified software.
  - Remove the IVUS catheter and flush the guide catheter to remove any air from the "Y connector."

Practical Considerations

- Sheath Size: ≥5 French.
- Anticoagulation: Therapeutic with Heparin or bivalirudin for ACT >250 s.
- Administer 100–200 μg of intracoronary nitroglycerin prior to imaging.
- Saline flushing may be needed to clear the vessel of artifact if using a mechanical IVUS catheter.

Limitations

- Catheter deliverability across tortuous and calcified/ stenotic vessels.
- Attenuation across calcified vessel segments.
- Near field imaging is difficult with phased array transducers requiring image processing to clear the ring artifact.
- Inter-observer variability in image interpretation.

Image Characteristic

- IVUS images are gray scale. Various intracoronary plaques are as demonstrated below (Figs. 9.2, 9.3, and 9.4). Reference vessel diameter is measured by considering the average of proximal and distal reference as measured from media to media or lumen-to-lumen.

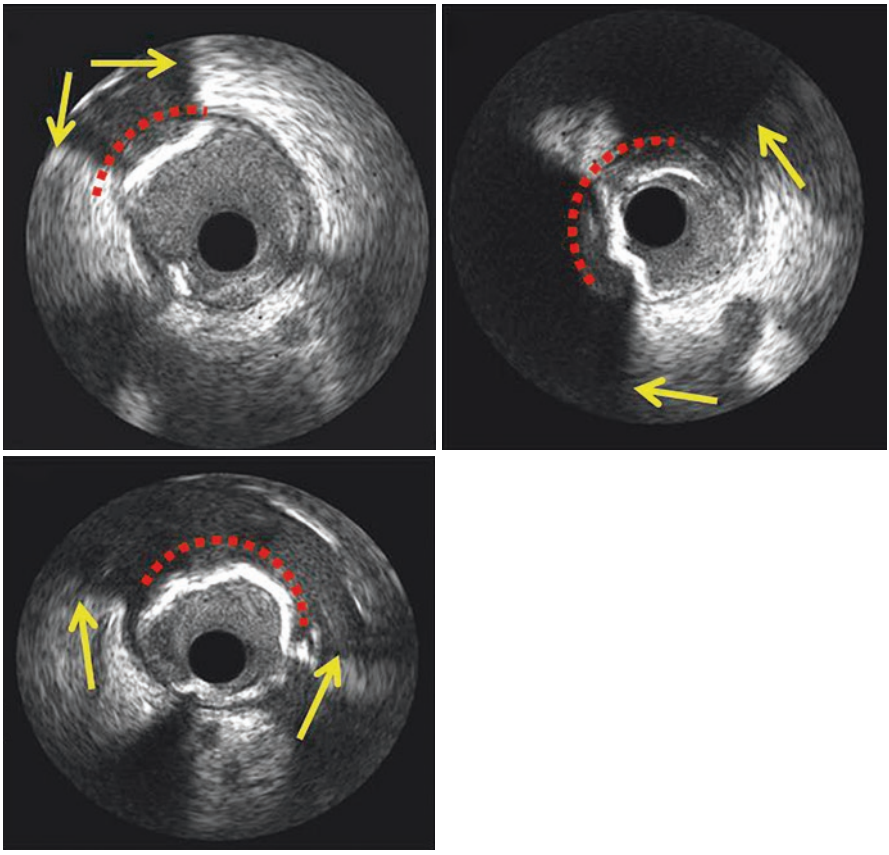

Fig. 9.2 Calcified plaque (hyperechoic) on IVUS (as demarcated by the red dotted line) with tissue attenuation (yellow arrow) beyond calcified segments

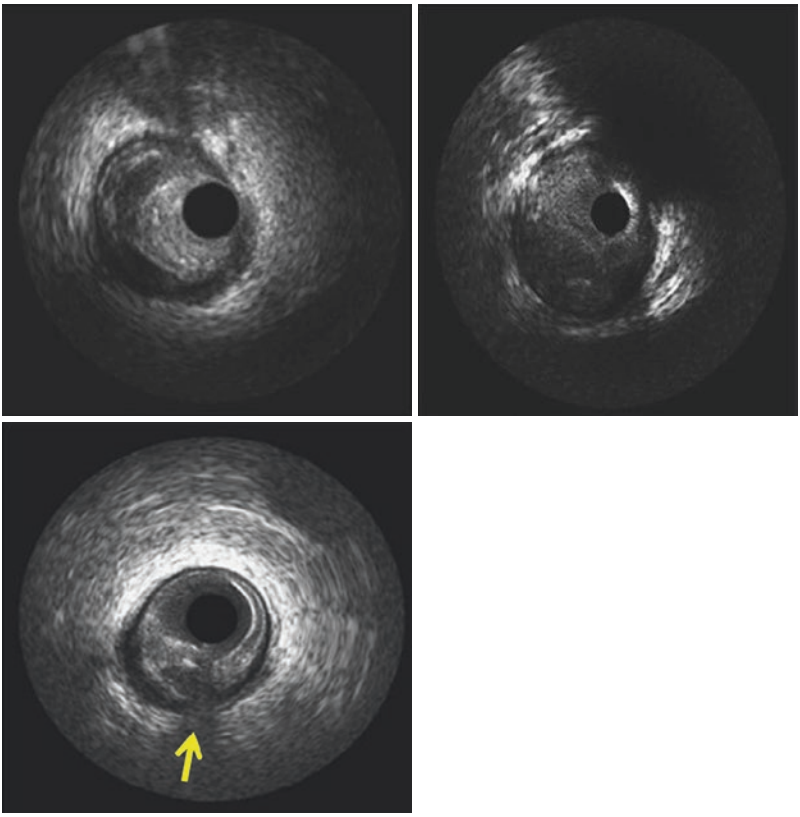

Fig. 9.3 Lipid pool (echo lucent area within plaque) on IVUS

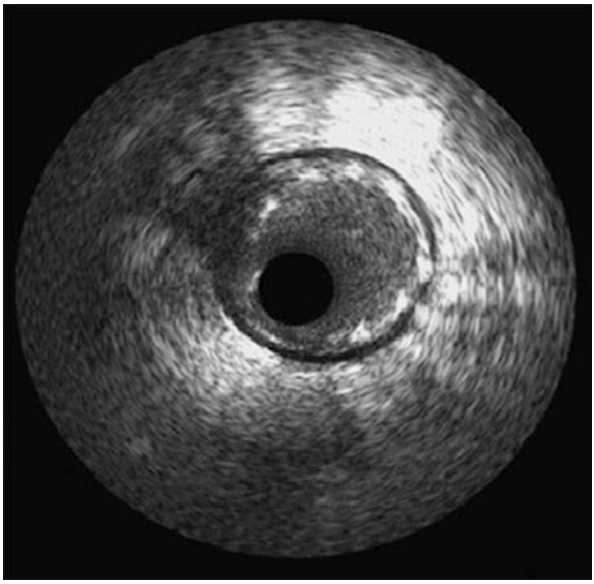

Fig. 9.4 Well-apposed stent struts in a stented coronary artery

Optical Coherence Tomography (OCT)

Optical coherence tomography (OCT) is an intravascular imaging technology for evaluating the cross-sectional and three-dimensional (3D) microstructure of blood vessels at a resolution of approximately 10 μm. It has been commercially available in the USA more recently and is gaining wider acceptance clinically. Abbott Vascular, Minneapolis, Minnesota, manufactures the current catheter (DragonFly OPTIS).

Principles of OCT Imaging

- The OCT catheter utilizes near-infrared light for image acquisition. The lag time of optical echoes reflected or backscattered from subsurface structures in biological tissues helps in discriminating different coronary structures. OCT light source is in the near-infrared (NIR) range, typically with wavelengths of approximately 1.3 μm.
- It has a resolution of nearly 10–20 μm, but tissue penetration is only 1–2 mm due to the short wavelength.

Indications

- OCT and IVUS have overlapping indications in terms of coronary interventions. Some specific circumstances where we prefer OCT imaging to IVUS include plaque characterization and accurate assessment of post-PCI dissections.
- Assessment of plaque morphology (e.g., plaque erosion in ACS) (Figs. 9.5 and 9.6).

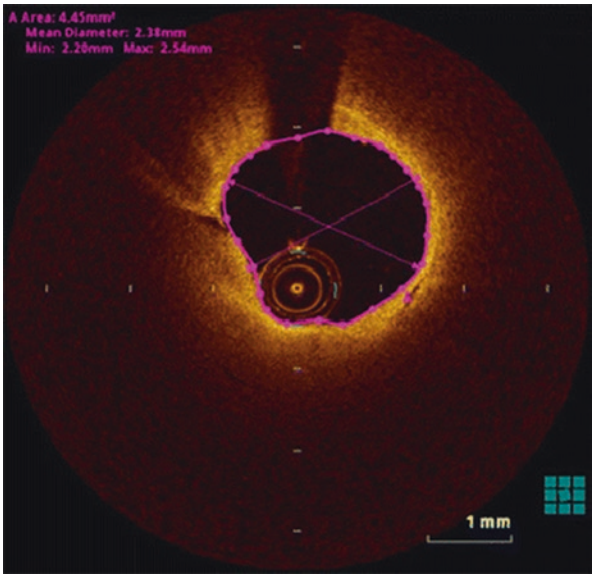

Fig. 9.5 OCT of an LAD lesion. Note the OCT catheter at 7 o'clock

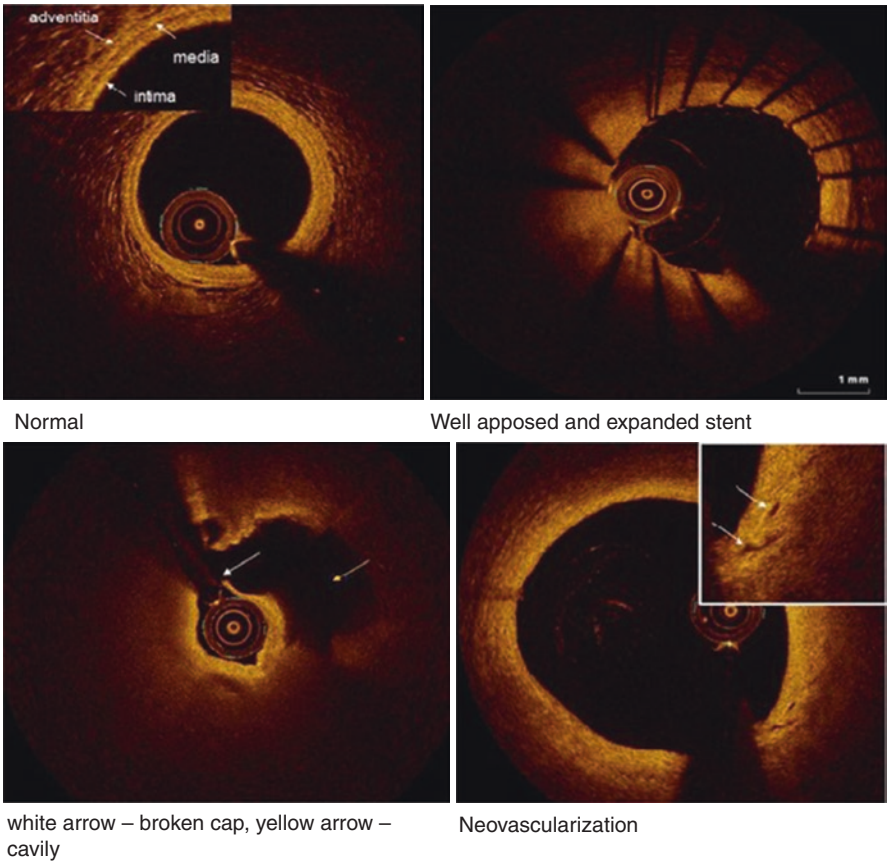

Fig. 9.6 (**a**) OCT cross section of normal vessel. (**b**) OCT of a well-apposed stent. (**c**) Ruptured plaque as seen by OCT. (**d**) Neovascularization of plaque

- Detection of edge dissections (Fig. 9.6). Major dissection defined as dissections >60° or ≥ 3 mm in length) [4].
- Post-PCI stent optimization by exclusion stent malapposition. (Stent malapposition defined as a lumen to stent distance ≥200 μm) [4].

Image Characteristics

- OCT images have a higher resolution than any other intracoronary imaging modality which allows for excellent tissue characterization as well as plaque detection. Therefore, OCT is the only imaging modality used to study/ characterize plaque erosion as an etiology of acute coronary syndromes.
  - Resolution of 10–20 μm.
  - 3-D reconstruction by proprietary software provides real-time visualization of the lumen along different planes.

  - Computer-generated 'rendering' of stent helps identify areas of malapposition that are highlighted as yellow.
  - Automatic measurements of lumen and stent area, with special focus on the minimal stent/ lumen areas.
  - The target vessel needs to be cleared of blood to acquire images using OCT.
  - Universally used automatic pullback at about 25 mm/sec allows for accurate lesion length assessment.

Image Acquisition

- Like any imaging, optimal image acquisition is the first step to accurate image interpretation. To avoid excessive contrast administration with repeated imaging attempts, a protocol based approach to OCT imaging is paramount.
  - Initiate with a sheath size of ≥6 French.
  - Therapeutic anticoagulation with heparin or bivalirudin for ACT >250 s.
  - Engage the target vessel and wire the lesion with a 0.014″ coronary guidewire.
  - Flush and prepare the OCT catheter with attached 3 cc syringe using undiluted contrast to purge air from the catheter.
  - Set the power injector according to the manufacturers' instructions (typically at least 3 milliliters/ second (mL/sec) for a total volume of 12 mL for RCA and 4 mL/sec for a total volume of 14 mL for left coronaries with a pressure of no more than 450 PSI). Consider using a 20 mL syringe in case of manual injection.
  - Check catheter position and blood clearance from the vessel with a test injection.
  - Once assured of position, activate the imaging catheter (preferably with auto-pullback) and inject 100% contrast through the power injector.
  - Remove the imaging catheter from the coronary artery once the imaging pull back is complete.

Equipment and Practical Aspects

- Sheath Size: ≥6 French. Avoid upsizing sheaths after therapeutic anticoagulation.
- Administer 100–200 μg of intracoronary nitroglycerin prior to imaging.
- The 4 P's of successful OCT image acquisition are co-axial catheter positioning, setting up of Power injector, Purging the target vessel of blood, and enabling automated Pull back.
- The length of the vessel acquired is about 75 mm, ensure the catheter is across the target lesion/ segment to be imaged.
- Consider balloon predilation across tight stenosis for catheter delivery.

Limitations

- Tissue penetration is limited, limiting clinical utility to non-left main lesions.

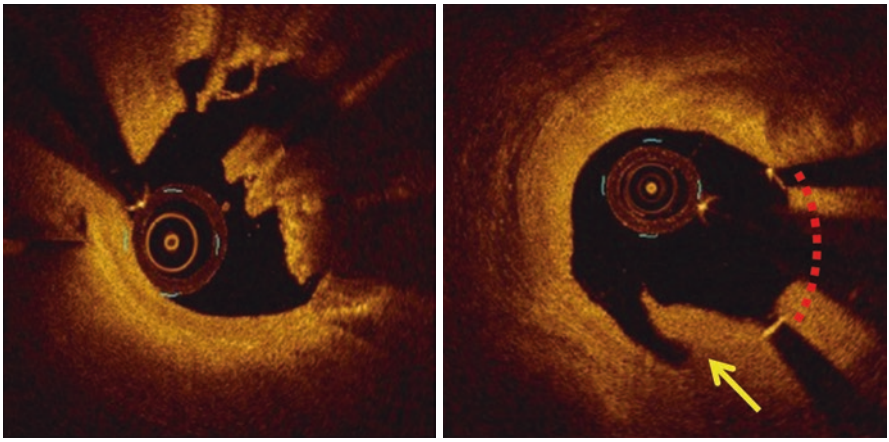

Fig. 9.7 (**a**) OCT showing ruptured plaque with red thrombus, distal attenuation behind thrombus. (**b**) OCT images of stent edge dissection

- Left main and ostial right coronary arterial lesions are not satisfactorily imaged as the blood cannot be cleared adequately with contrast and tissue penetration is limited.
- Catheter deliverability across tortuous and calcified/stenotic vessels.
- Attenuation across lipid-rich plaques.

Image Characteristics

- OCT images are generally crisp and well defined, especially compared to IVUS images. (Fig. 9.5). Some examples of various OCT findings are as below (Figs. 9.6 and 9.7).

Comparing OCT vs IVUS

Intravascular imaging is now considered obligatory for most complex coronary cases, with proven mortality benefits in CTO and long lesions [5, 6].

- The inherent differences in physical principles of either imaging modality (Table 9.1) provide a certain uniqueness to each technique's utility in clinical practice.
- IVUS has also been shown to increase the lumen gain/ post-dilation balloon sizing in various studies especially for complex lesions [5, 6].
- High definition IVUS imaging with mechanical pull-back provides good quality images that help in the optimization of percutaneous interventions, including lesion length, calcification depth, and angle.

- Each imaging modality has some specific scenarios where they are preferred as in Table 9.2.
- An intimal thickness of >0.5 mm in proximal left anterior descending (LAD) is considered a marker for increased rates of major adverse cardiovascular outcomes [7].
- OCT has a superior longitudinal and axial resolution.
- There are well-defined objective definitions of stent malapposition (>200 microns) and major dissection (>60 degrees/ 3 mm) which can further decrease inter-observer variability in image interpretation [4].
- Long-term follow-up for clinical outcomes is lacking but outcomes up to 24 months show decreased MACE rates with either imaging modality [10].
- While there is significant, inter-observer variability in vessel sizing based on angiography alone, such disagreements can be overcome with intracoronary imaging [11].
- It is of paramount importance that each practicing interventional cardiologist is familiar with the practical aspects of both imaging modalities, some of which are listed in Table 9.3.

Table 9.1 Physical characteristics of OCT versus IVUS

	OCT	IVUS
Radiation type	Light	Ultrasound
Wavelength, μm	1.3	35–80
Frequency	190 THz	20–45 THz
Resolution, μm	10–40	100–150
Penetration depth, mm	1–3	4–10
Field-of-view diameter, mm	15	7–10
Frame rate, frames/s	100	30
Pullback speed, mm/s	20	0.5–1
Plaque characterization	Yes	Yes
Fibrous cap measurement	Yes	No
Vessel remodeling	No	Yes
Blood removal required	Yes	No

Table 9.2 Specific scenarios for utility of imaging modalities [3, 7–9, 12]

	OCT	IVUS
No/low contrast PCI	No	Yes
Transplant vasculopathy	No	Yes
Acute stent thrombosis	No	Yes
Delayed in-stent restenosis	Yes	Yes

Table 9.3 Practical aspects of imaging for the interventionalist

	Description
ACT	>250 s before IVUS/ OCT catheter insertion
IVUS	Intimal thickness > 0.5 mm on IVUS consistent with transplant coronary vasculopathy. Left main MLA >6 mm^2, safe to defer stenting.
OCT	Stent malapposition with ≥200 μm distance between vessel wall and stent. Major dissection defined as a dissection >60° or ≥ 2 mm in length.

Near-Infrared Spectroscopy (NIRS): Intravascular Ultrasound (IVUS) Imaging

The imaging system utilizes diffuse reflectance near-infrared spectroscopy (NIRS) , a classic method of analytical chemistry, to characterize the plaque for lipid content [13]. Diffuse reflectance spectroscopy requires scattering and absorption at different wavelengths of the light by the tissue.

How It Works

- The combination of scattering and absorption of near-infrared light by organic molecules in the arterial wall and plaque produces a unique chemical signature.
- An algorithm analyzes the detected signal for signs of cholesterol and provides automated lipid core-containing plaques (LCP) detection without the need for manual image processing (Fig. 9.8).
- In contrast to OCT, NIRS can image through blood, as it does not need light to be directly reflected back to the detector [13].

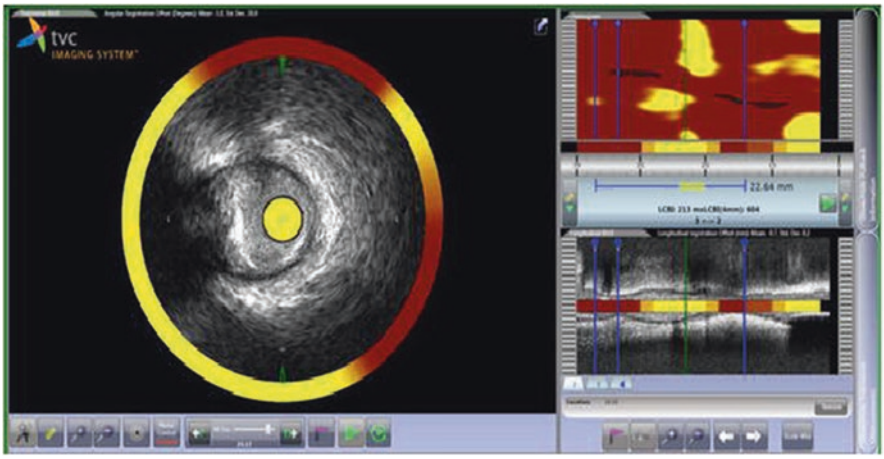

Fig. 9.8 TVC Composite™ view of co-registered near-infrared spectroscopy lipid core plaque with intravascular ultrasound. The chemogram displays low probability of lipid as red and high probability as yellow. The lipid core burden index (LCBI) indicates the amount of lipid in the scanned artery on a 0–1000 scale

Table 9.4 Equipment TVC insight catheter

	Unit
Minimum guide catheter	6 French (2 mm)
Maximum guidewire	0.014 (0.36)
Catheter crossing tip profile	3.2 French (1.1 mm)
Maximum imaging depth	16 mm
Catheter working length	120 mm
Operating frequency	40 MHz

Equipment

- See Table 9.4 for equipment for TVC Insight catheter.

Indications

- The Infraredx TVC Imaging SystemTM is intended for the detection of LCPs using NIRS and IVUS examination of the coronary arteries.
- The combination of NIRS with IVUS in a single catheter combines the benefit of NIRS with the benefits of IVUS.

Limitations

- NIRS–IVUS does not allow detection of non-superficial LCPs and LCPs in large vessels (more than 6 mm diameter) cannot visualize neovascularization.

NIRS–IVUS Numbers to Remember

- Patient with stable coronary artery disease who undergoes PCI of lesions with large lipid core plaques (maxLCBI4 mm ≥500 by NIRS) is associated with a 50% risk of periprocedural MI and a maxLCBI4 mm >400 in the culprit segment is considered significant with STEMI [13, 14]. Statin therapy has been shown to decrease LCBI in lipid-rich plaque demonstrated by NIRS imaging [15].
- Multimodality imaging: correlation of OCT and NIRS–IVUS imaging.
- Combined utilization of OCT and NIRS–IVUS allows to characterize the microstructural features of plaque morphology, such as fibrous cap thickness and neovascularization (OCT) and plaque lipid content (NIRS), and perform robust quantitative measurements of the lumen, vessel, and plaque area (grayscale IVUS) in the same lesion (Table 9.5). Figure 9.9 shows an example of multimodality images of a thin-cap fibroatheroma lesion.

Table 9.5 Comparison of three intravascular imaging modalities for the detection of coronary lipid core plaque

	IVUS	OCT	NIRS–IVUS
Hybrid intravascular imaging	No	No	Yes
Axial resolution, um	200	10	100
Imaging through blood	++	–	++
Need for blood column clearance during image acquisition	No	Yes	No
Imaging through stents	No	Yes	Yes
Imaging through calcium	No	Yes	Yes for NIRS, no for IVUS
Imaging neovascularization	No	Yes	No
Detection of non-superficial LCPs	Yes	No	No
Evaluation of LCP cap thickness	+	++	*
Detection of thrombus	–	+	*
Expansive remodeling	++	–	++
Need for manual image processing for LCP detection	Yes	Yes	No

++ Excellent, + good, – impossible, * potential under investigation, *VHIVUS* virtual histology intravascular ultrasound, *OCT* optical coherence tomography, *NIRS* near-infrared spectroscopy, *LCP* lipid core plaque

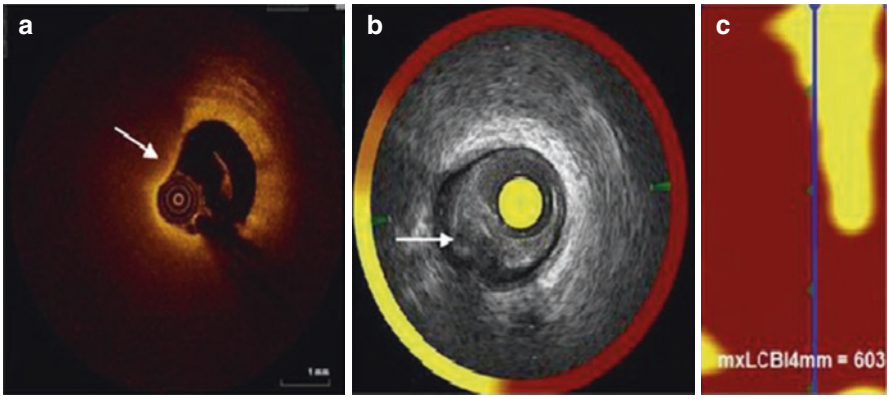

Fig. 9.9 (**a**) OCT image of a thin-cap fibroatheroma (TCFA) lesion with a 60 μm fibrous cap (*arrow*) overlying a large lipid core. (**b**) The lipid pool (*arrow*) is shown in the corresponding IVUS image. (**c**) NIRS chemogram quantifies the lipid content of the lesion

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Basics of Intracoronary Devices

10

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Introduction

In the present era of interventional cardiology, with the introduction of newer and more advanced devices every year, there are various intracoronary devices apart from stents and balloons, which have found their place in the catheterization laboratory. This chapter outlines basic coronary devices and their usage.

Balloons

Presently, the rapid exchange (Rx) coronary balloon (Monorail catheter) is the standard construction used for routine coronary intervention. It is also called single operator exchange (SOE) catheters and replaced over the wire balloon (OTW) as the routine used balloons.

- Compliant/semi-compliant balloons.
- Noncompliant balloons.
- Cutting balloon (Wolverine™, Flextome™).
- Scoring balloon (AngioSculpt™).
- Chocolate XD® PTCA Balloon.

Compliant/Semi-Compliant Balloon

- Used for predilation, opening the stent struts, and CTO dilatation.

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- Advantage: better crossing profile than a noncompliant balloon.
- The Sapphire II PRO 1.0 mm is the smallest, FDA-cleared coronary balloon on the US market.

Noncompliant Balloon

Used for post-dilation and predilation of a calcified lesion with or without atherectomy (balloon should be 1:1 to vessel diameter to achieve complete expansion).

- Advantage: high-pressure lesion or stent dilatation with little change in balloon reference diameter without "dog bone" effect at the edges.

Cutting Balloon (Wolverine™ Flextome™)

- Noncompliant balloon.
- Microblades or atherotomes over its surface that cuts the plaque (Fig. 10.1).

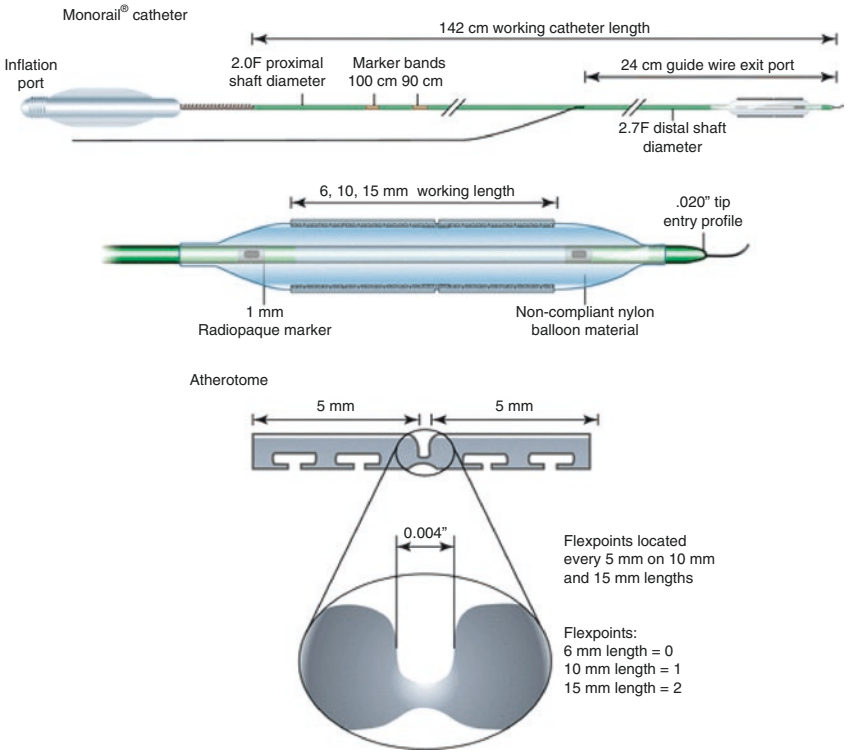

Fig. 10.1 Flextome™ cutting balloon: blades mounted on a noncompliant balloon. Flex points are located every 5 mm on the balloon. Boston Scientific Corporation or its affiliates. All rights reserved (Used with permission of Boston Scientific Corporation)

- Uses:
  - Fibrocalcified lesions.
  - Ostial lesions.
  - In-stent restenosis.
- How to use.
  - Sizing of cutting balloon: For native 1:1 sizing and upper inflation, the limit is 8 atm. For ISR lesion, sizing a quarter size more than the reference vessel and inflation at 10–12 atm can be done.
  - Rapid exchange or over the wire system.
  - Two/three cuts should be done as a balloon is deflated and pulled into the guide after the first inflation, and then re-advanced for the second/third cut/inflation.

Scoring Balloon (AngioSculpt™)

- High-pressure balloon with nitinol wire wrapped around, which focus dilation forces.
- Use is similar to Flextome™, but inflation at higher pressure (up to 22 atm) can be achieved with AngioSculpt™.

Chocolate XD® PTCA Balloon Catheter

- Semi-compliant balloon with nitinol constraining structure.
- Reduces the strain and trauma induced on the vessel walls during inflation through the use of 'pillows' without cutting or scoring 'grooves': Stress relief, plaque modification.

Super High-Pressure Balloon (OPN NC)

- Unique two layers of construction providing the highest (up to 35 atm) rated burst pressure (RBP).

Coronary Stents

A coronary stent is a mesh of metal or bio-absorbable polymer, non-coated, or coated with an antiproliferative drug, which is delivered inside the coronary to treat stenotic lesions.

There are several different types of stents available:

- Bare metal stents.
- Drug-eluting stents.

  - First-generation: Cypher™, Taxus™.
  - Second–third generation: Xience™, Promus™, Synergy™, Orsiro™, and Resolute™.
  - Bio-absorbable Stents (currently not available in the USA).

Components of Stent

- Metal Mesh:
  - Design: open-cell/closed cell.
  - Tensile strength of the stent.
  - Radial strength.
  - Longitudinal foreshortening.
- Polymer:
  - Carries the drug as it has a drug reservoir and allows for controlled drug release over time.
  - Permanent vs biodegradable polymer (like Synergy™ or Orsiro™ stent).
  - Non-polymeric drug release stents, the bioactive substance is directly attached to the stent surface (like BioFreedom™ biolimus A9-eluting stent).
- Drug:
  - Lipophilic agents like paclitaxel or limus analogs.
  - Inhibits neointimal proliferation to prevent restenosis.

Guide Catheter Extension

Prototypes

- Guideliner™, Telescope™, Guidezilla™, and Trapliner™ are different support catheter designed based on the '*mother and child*' concept (Fig. 10.2).

Components

- Distal rapid exchange 25 cm extension segment (13 cm in the Trapliner™):
  - Middle stainless steel braid to provide support.
  - Outer polymer-coated material to provide lubricity and prevent ostial damage.
- Trapliner™ has a balloon for trapping the wire.
- Collar: proximal end of a rapid exchange segment (away from tip).
- 125 cm stainless steel push rod.
- Sizes: 5.5, 6, 7, and 8 F in Guideliner™, and 6, 7, and 8 F for Guidezilla™ and trapliner™.

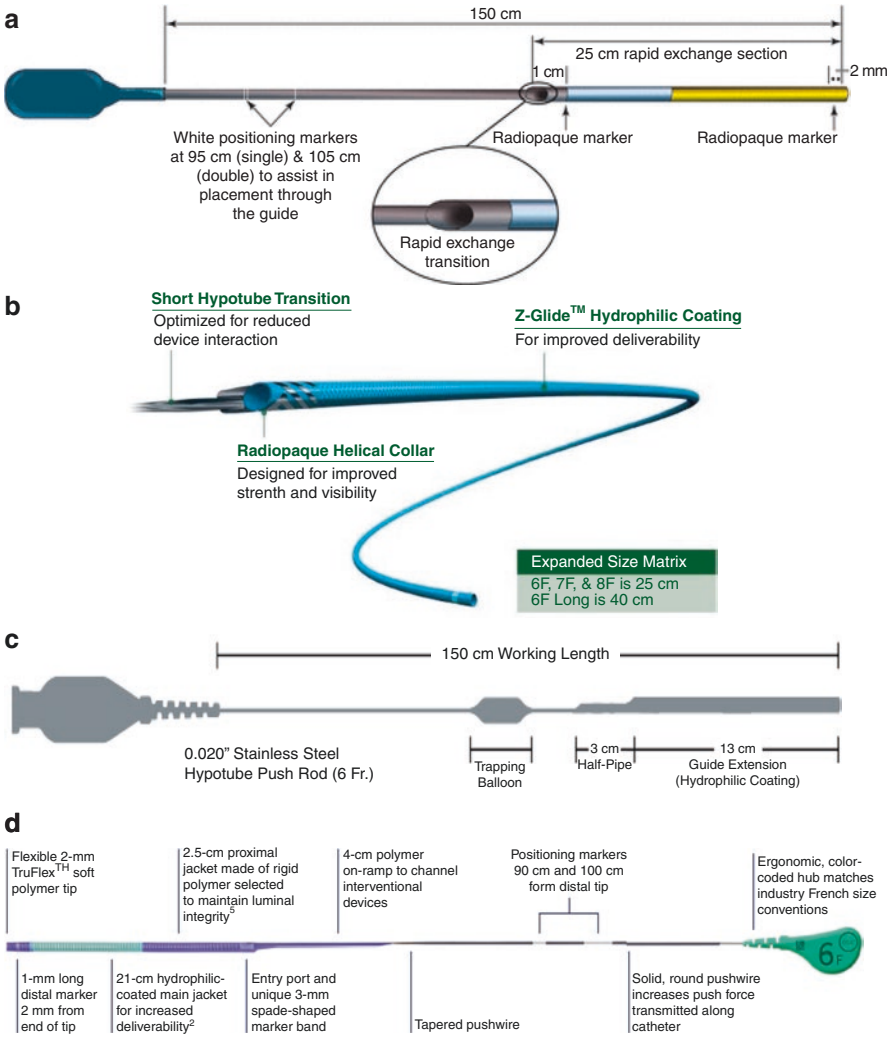

Fig. 10.2 Guide extension provides support for complex interventions. (**a**) Guideliner™. (**b**) Guidezilla™. (**c**) Trapliner™. (**d**) Telescope™

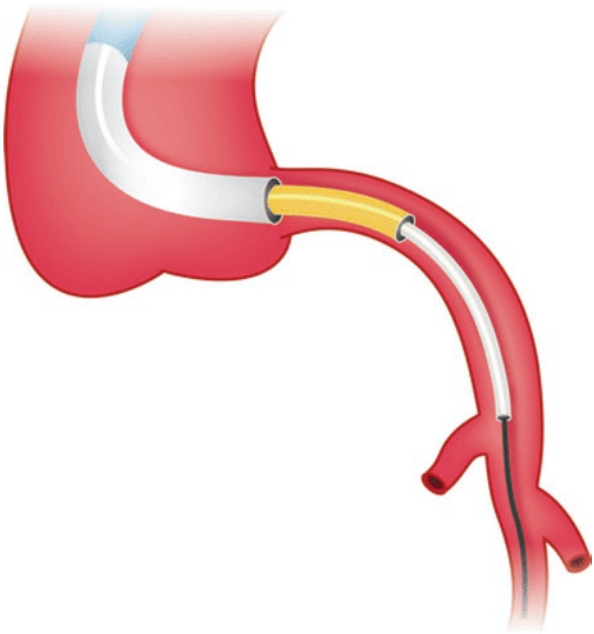

Fig. 10.3 Guideliner™: 'mother and child' guideliner extending beyond guide catheter into the coronary

Use

- Deep seating of guide (Fig. 10.3).
- For extra backup support.
- Distal delivery of devices.
- For coaxial alignment.

Technical Tips

- Flush the device from the tip.
- Select the device appropriate to guide (6 F vs 7 F).
- Advance over a wire into the guide.
- Advance a balloon into the coronary.
- Advance the Guideliner™ over the balloon shaft deep into the vessel. This technique prevents Guideliner™-induced dissection. Balloon anchoring can facilitate advancement as well.
- A useful technique for challenging cases is to use repeated balloon anchoring by inflating a balloon at guide extension tip at low atmospheres and gradual advancement over balloon while deflating. It creates a smooth transition at the tip of the guide extension and prevents vessel injury.
- The length of Guideliner™ inside the coronary is directly proportional to the amount of support.
- Avoid contrast injection (particularly with high pressure) when the Guideliner™ is deep in the coronary, especially when the pressure is damped.

Thrombus Aspiration Catheter

Use

- STEMI in the setting of primary PCI.
- Vein graft intervention with large thrombus.
- Coronary ectasia/aneurysm with thrombus and requiring intervention.
- For drug delivery in the distal vessel.

Types

- Mechanical:
  - AngioJet™: 4 F and 5 F.
  - Penumbra™ Indigo System.
- Manual.
  - Export™.
  - Pronto Device™.
  - Diver CE™.
  - Fetch Catheter™.

Pronto™

- Manual device aspirate by negative suction with a 30 cc locking syringe (Fig. 10.4) which is available in different sizes (Table 10.1).

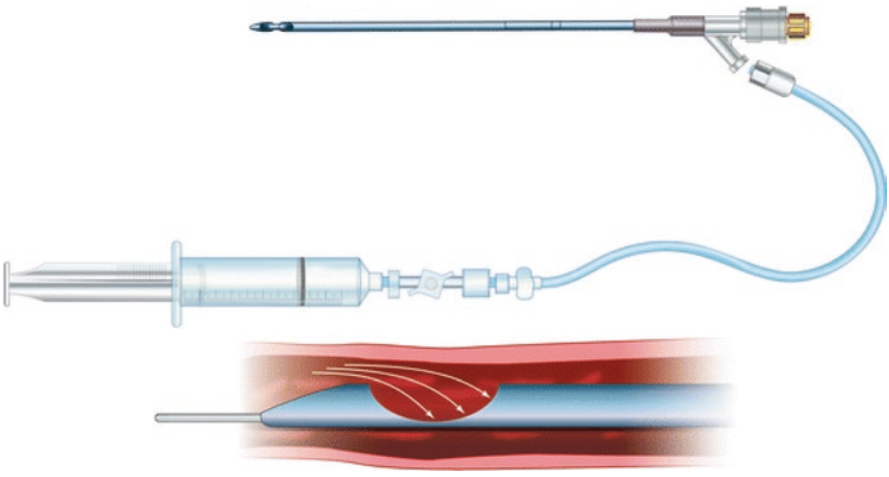

Fig. 10.4 Thrombus aspiration catheter 30 ml locking aspiration syringe. Two-way stopcock. Y-connector. The yellow hub is the stylet

Table 10.1 Sizes of Pronto™

Size	Guide compatibility	Sheath compatibility	Guidewire	Minimum vessel size	Working length
5.5 F	6 F	5 F	0.0014″	1.75 mm	138 cm
6.0 F	6 F	5 F	0.0014″	2.00 mm	138 cm
7.0 F	7 F	6 F	0.0014″	2.25 mm	138 cm
8.0 F	8 F	7 F	0.0014″	2.50 mm	138 cm
Pronto LP[b]					
4.0 F	6 F	6 F	0.0014″	1.5 mm	140 cm

For peripheral use where guide catheter is not used
[p]Reloaded with a stylet in a lumen different from aspiration to provide kink resistance and pushability. Rapid exchange length of 20 cm. Most commonly used in coronary interventions

- Kink resistant.
- Distal 18 cm hydrophilic coating.
- Unique distal tip:
  - Tapered entry: protect distal vessel and navigation.
  - Self-centering: prevents vessel adhesion while thrombus extraction.
  - Sloped extraction lumen: to aspirate large thrombus.
- Aspiration syringe is connected to the back end of the manual aspiration device, the stopcock is locked at the hub, while the syringe piston is pulled negatively and locked, thereby creating a negative pressure inside the syringe.
- The device is advanced distal to the thrombus.
- The device is preloaded with a stylet to reinforce the proximal shaft during delivery and it should be removed prior to aspiration.
- Opening the stopcock at the hub allows the creation of a vacuum within the catheter, so blood and thrombus is sucked back into the syringe.
- The catheter is pulled back in a steady and slow motion. Multiple runs are performed as required.
- After the catheter is removed, the back end of the guide is opened and a generous amount of bleed back is allowed.
- The syringe and catheter aspirate are then examined for any thrombus.

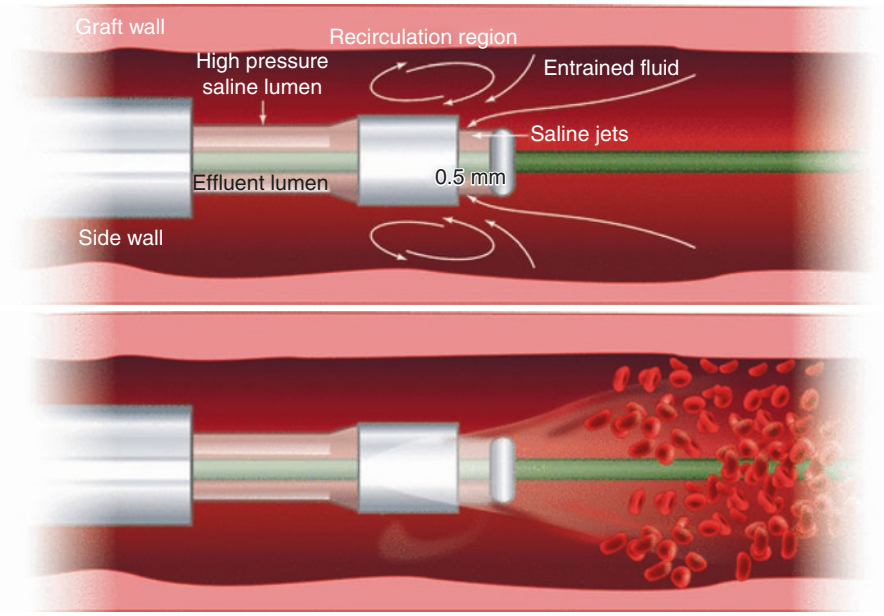

Fig. 10.5 AngioJet™ catheter. Separate lumen for saline injection and aspiration creating a low-pressure area

AngioJet™

- Angiojet devices pulverize the thrombus with a jet of saline and aspirate through other ports by the mechanism of the Venturi–Bernoulli effect.
- Select the device (Fig. 10.5):
  - 4 F AngioJet™ requires a 6 F guide.
  - 5 F AngioJet™ is useful in vessels >5 mm and SVG interventions, and needs a 7 F guide.
- Prime it outside the body by dipping in saline.
- Advance it until the proximal part of the lesion.
- AngioJet™ is switched to aspirate from proximal to distal.
- Disadvantages:
  - Difficult to deliver in tortuous vessels, distal segment, and calcified vessels.
  - Risk of Injury to a vessel like dissection.
  - Migration of thrombus.
  - Stent deformation (proximal) if an attempt is made to pass beyond the stent.

Fig. 10.6 Penumbra aspiration catheter delivering consistent suction throughout the aspiration cycle

Penumbra Indigo System with CAT RX Aspiration Catheter

- Delivers consistent suction throughout the aspiration cycle to maximize the efficiency of thrombus collection (Fig. 10.6).

Rotational/Orbital Atherectomy/Laser/Lithoplasty System (Shock-Wave™)

- See Chap. 22.

Covered Stents

JOSTENT GraftMaster™

- Two stainless steel stents and in between expandable PTFE material (Fig. 10.7).
- Over the wire system and rapid exchange system.
- Approved by FDA as a humanitarian device.
- Sizes: 2.8, 3, 3.5, 4, 4.5, and 4.8 mm in 13, 18, 23, and 27 mm length.
- Use:
  - Coronary perforation.
  - Coronary aneurysm requiring treatment.
  - Coronary artery fistula requiring treatment.
- Technical tips:
  - Consider rewiring with an extra support wire like Mailman™ or Iron Man™.
  - 300 cm coronary wire for OTW system.
  - Indicated for use in vessels ≥2.75 cm.
  - Anticipate difficulty in delivery.
  - Side branch closure if present at the site of rupture.
  - Difficult delivery into distal, tortuous, or calcified vessels.
  - Higher incidence of ISR and thrombosis.
  - Recommended guide catheter:
    2.8–4.0 mm: 6 F (min. I.D.** 0.068″).
    4.5–4.8 mm: 7 F (min. I.D.** 0.074″).

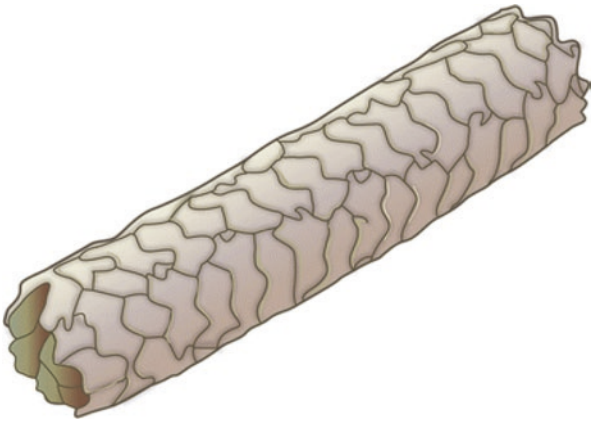

Fig. 10.7 PTFE mesh in between two laser-cut stainless steel

Fig. 10.8 A polytetrafluoroethylene-covered single layer stent graft

PK Papyrus™ Covered Coronary Stent

- Approved by FDA as a humanitarian device 2019 (Fig. 10.8).
- Cobalt-chromium metal alloy and covered with a polyurethane membrane.
- Greater bending flexibility and a smaller crossing profile compared to the traditional sandwich design stent.
- Recommended guide catheter:
  - 2.5–4.0 mm: 5 F (min. I.D.** 0.056″).
  - 4.5–5.0 mm: 6 F (min. I.D.** 0.070″).

Embolic Protection Devices (EPD)

Types (Fig. 10.9)

- Proximal Protection Devices:
  - Mainly used for carotid interventions and not currently used for SVGs.
  - Prototype: Parodi™ system, MOMA™ device.
- Distal Occlusion Devices:
  - Prototype: PercuSurge GuardWire™, TriActive™ system.
- Distal Filter Devices:
  - Prototypes: SpiderFX™, FilterWire EZ™.
  - Advantages:
    Maintains distal perfusion while trapping particles >100 μm.
    Possibility of contrast imaging during operation.
  - Disadvantages:
    High crossing profile.

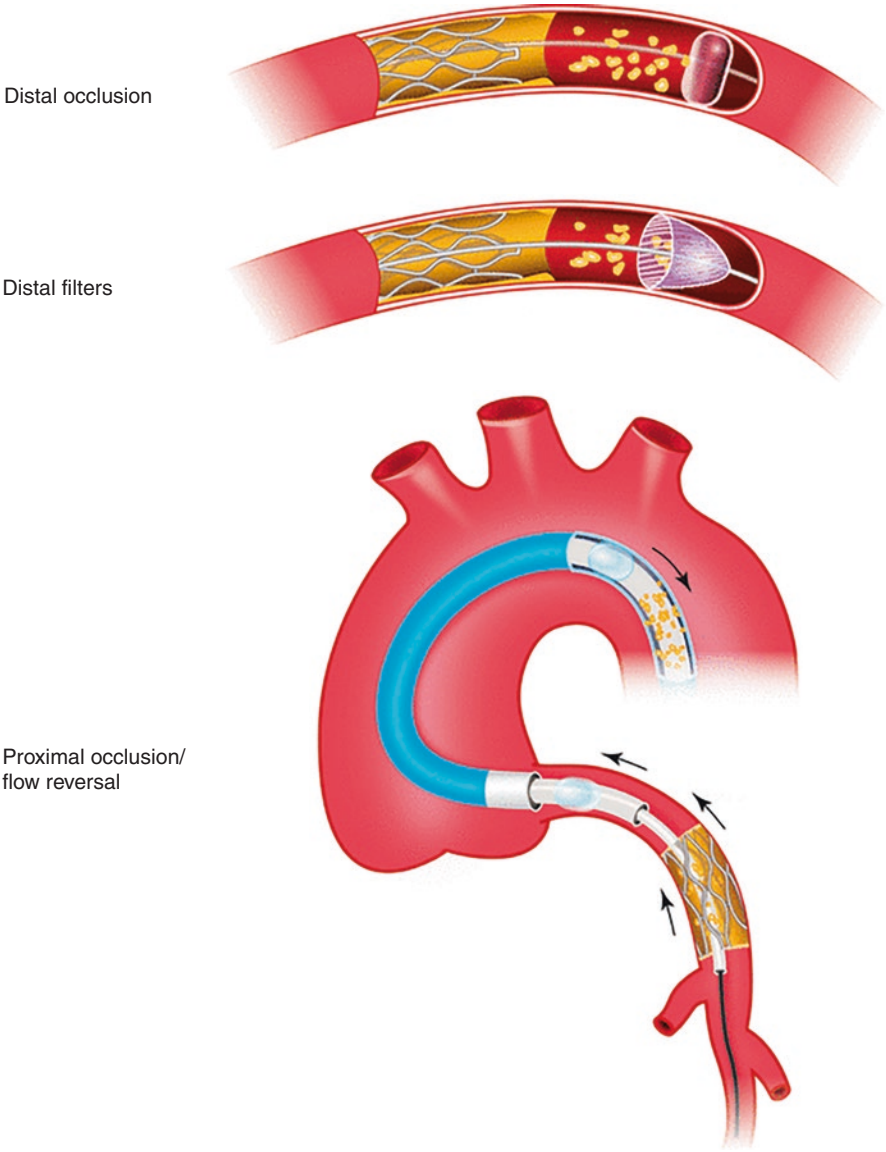

Fig. 10.9 Embolic protection devices: (a) distal occlusion balloon. Debris is aspirated with an aspiration catheter before deflating the balloon. (b) Filter which traps the embolic debris. The filter is retrieved post-procedure by ensheathing it carefully so as not to embolize from the filter: partial capture or complete capture. (c) Proximal occlusion device. Useful when the distal landing zone is not present. Mainly used for carotid interventions. The balloon is occluded proximal to the lesion and flow is reversed post-procedure

Difficulty in crossing the stenosis.
Difficulty in advancing a retrieval catheter across the stented segment.
There is a possibility that emboli pass through filter pores or an incompletely opposed filter.

PercuSurge GuardWire™

- 0.014″ nitinol distal wire (guard wire).
- Occlusion balloon 3.5 cm proximal to the terminal end of the wire.
- Steps:
  - Balloon is inflated to 2–5 atm depending on vessel size.
  - Stent is deployed over the guard wire.
  - Material is aspirated using an Export™ or Pronto™ catheter (2–3 runs).
  - Distal balloon is then deflated.
- Advantages:
  - Low crossing profile (0.026–0.033″).
  - Traps small and large particles and soluble particles.
- Limitations:
  - Complete occlusion of antegrade flow.
  - Single guidewire.
  - Collection of material in fornices of the balloon.

SpiderFx™ Filter

- Sizes: 3–7 mm. Select size 1 mm greater than the vessel diameter and deploy 3 cm distal to the lesion (Fig. 10.10).
- 320/190 cm snap wire that moves independently of the filter longitudinally.
- Nitinol mesh for complete vessel apposition.
- Steps:
  - Deployed and retrieved through a dual-ended catheter.
  - Compatible with 6 F guide and 0.014–0.018″ wire.
  - Crossing profile 3.2 F.

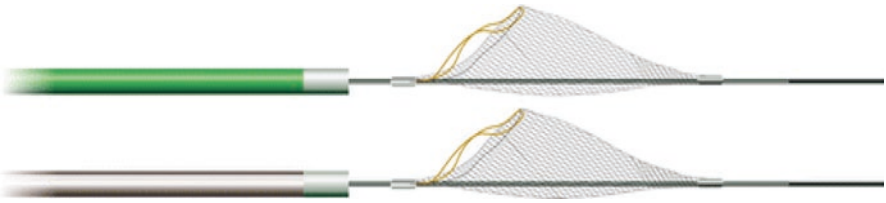

Fig. 10.10 Spider filter: unsheathe the filter after stabilizing at the desired position

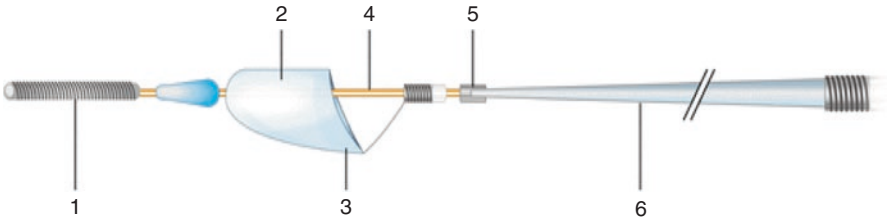

Fig. 10.11 (*1*) Radiopaque tip (2 and 3 cm for two systems, respectively). (*2*) Filter. (*3*) Radiopaque nitinol loop. (*4*) Spinner tube. (*5*) Proximal loop. (*6*) PTFE-coated wire (0.014″)

- Filter is withdrawn inside the sheath (transparent part in the mid-segment of the sheath) by ensuring that it is immersed in saline the whole time.
- Flush from the distal end.
- Advanced as a monorail over the coronary wire that was used to cross the lesion. Park it in a normal segment well beyond the lesion. The coronary wire is removed, and the capture wire is advanced until distal markers align. The SpiderFx™ catheter is then removed thereby deploying the filter.
- After completion of the intervention, the filter is retrieved by sheathing the device (blue end recovering catheter) and removing the whole system.

FilterWire EZ System™

- Components:
  - Wire.
  - Delivery sheath.
  - Retrieval sheath.
  - Torquer, peel-away introducer, and valve dilator.
- Sizes:
  - Two sizes for the following range of coronary artery sizes with a wire length of 190 or 300 cm [2.25–3.5 and 3.5–5.5 mm].
- Landing zone:
  - Landing zone is the distance from the distal edge of the lesion to the tip of the radiopaque part (Fig. 10.11). The filter can be deployed in the normal segment of the vessel.
    For 2.25–3.5 mm system: ≥2.5 mm.
    For 3.5–5.5 mm system: ≥3 mm.

Intravascular Brachytherapy (IVBT)

IVBT

- Indication: to reduce the recurrence of in-stent restenosis (ISR).

- Mechanism: locally applied radiation can block or attenuate local tissue proliferation causing ISR.
- Effects of brachytherapy:
  - Delayed recurrent restenosis.
  - Late stent thrombosis because of prolonged inhibition of intimal formation.
  - Late vessel stent separation because of positive remodeling.
  - Geographical miss: reactive hyperproliferation at the edges because of less radiation dose.
  - Types of brachytherapy: both gamma (low energy/high penetration) using 192-Ir and beta sources (high energy/low penetration) are effective, but only the beta source is clinically available.
- FDA-approved brachytherapy systems:
  - Gamma: Cordis Checkmate (192-Ir)™ (not commercially available).
  - Beta: Guidant Galileo (32-P)™ (not commercially available) and (Novoste Beta-Cath (90-Sr-Y)™.

Novoste Beta-Cath™

- Clinically used in the USA.
- Guide selection: 6 F.
- Wire: 0.014″.
- Distal 1 cm rapid exchange length.
- Selection of device and steps:
  - Appropriate source train length (30 mm, 40 mm, or 60 mm) is selected based on the injury length and desired margin (Fig. 10.12).
- Balloon predilation of ISR is done.
- No stent is placed when IVBT is planned because of an increased risk of stent thrombosis.

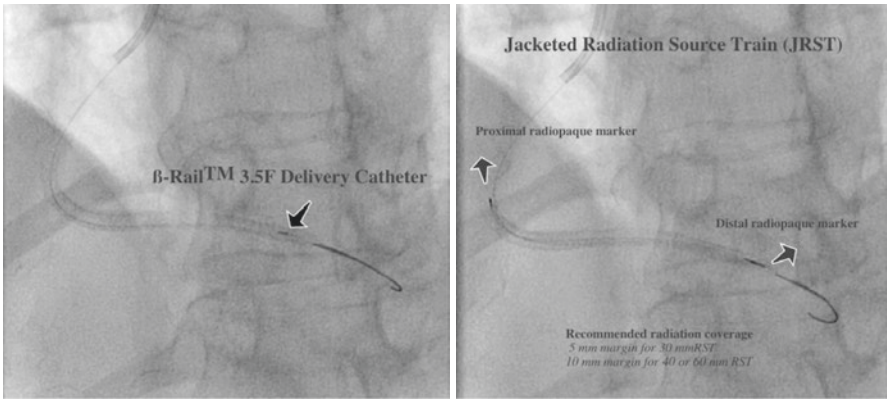

Fig. 10.12 B-Rail delivery catheter and radiation source train

- Dual antiplatelet is continued for at least 3 years.
- For a long lesion, serial delivery of catheters with a 1 mm overlap is recommended. We recommend adequate anticoagulation with bivalirudin (to an ACT of >300) and an additional dose of heparin IV just before delivery of the radiation dose.

Microcatheters for Standard Use

There are plenty of commercially available micro/ support catheters on the market (Caravel™, Teleport™, Finecross™, Turnpike™, etc.). These catheters are useful because they provide support (especially during CTO interventions), and facilitates wire exchanges.

Corsair Catheter™

- Hydrophilic-coated distal 60 cm (Fig. 10.13).
- Tungsten braiding +10 elliptical stainless steel braids.
- Outer diameter is 2.8 F and tapers to 1.3 F.
- Length is 135 cm for antegrade and 150 cm for retrograde.
- Characteristics:
  - Excellent pushability, flexibility, and torqueability.
  - Kink-resistant tip.
  - Helpful in crossing microchannels in CTO.

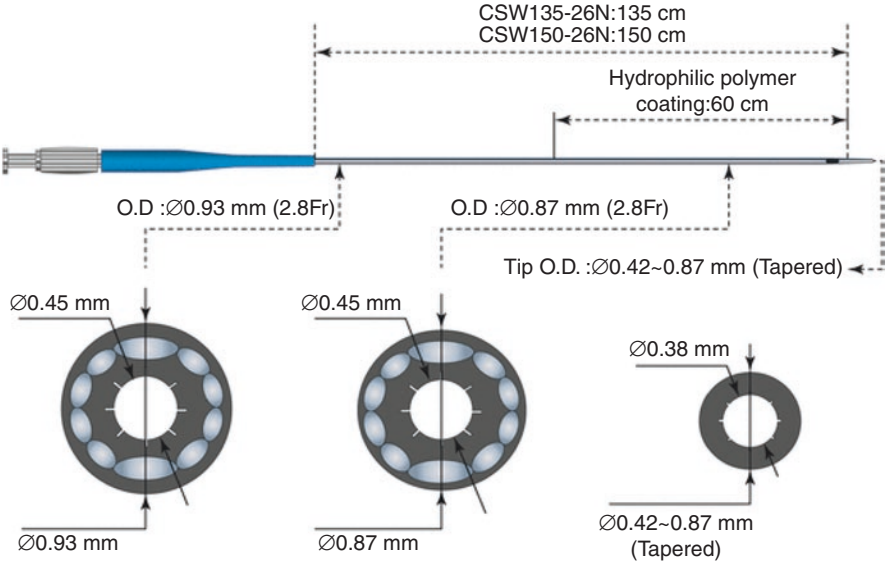

Fig. 10.13 Images of Corsair microcatheter illustrating tapered tip and hydrophilic nature

Fig. 10.14 Twin-pass catheter. The second lumen (over the wire) can be used for the second wire or delivering of contrast or drug distally

- Used for retrograde CTO PCI.
- Provides support to the guidewire and useful for guidewire exchange.
- Can be rotated in either direction (better torque transmission with counter clock move).

Twin-Pass™ Catheter (the Only Dual-Lumen Microcatheter Available in the US)

- Double-lumen exchange catheter (Fig. 10.14).
- One rapid exchange lumen and one over the wire.
- Hydrophilic coating present.
- Length is 140 cm.
- 3 F system.
- Guide compatibility:
  - ≥5 F for 0.014″ system which is most commonly used for coronary intervention.
- Use:
  - Flush both lumens before use.
  - To deliver drugs distally into coronary circulation.
  - To image distally with contrast.
  - To exchange wire while keeping one wire in place.
  - Advance a guidewire into a side branch.

Microcatheters for Difficult Wiring of Side Branches or Angulated Lesions

Venture™ Catheter

- Over-the-wire system or monorail (Fig. 10.15).
- Deflectable tip, which can be controlled by rotating a knob on the device.
- How to use:
  - Advance the wire beyond the origin of the side branch into the main vessel.
  - Advance a Venture catheter over the wire beyond the side branch to be entered.
  - Pull back the wire inside the Venture™ catheter.
  - Rotate the knob to orient the tip of the Venture™ catheter to the angle of side branch origin.

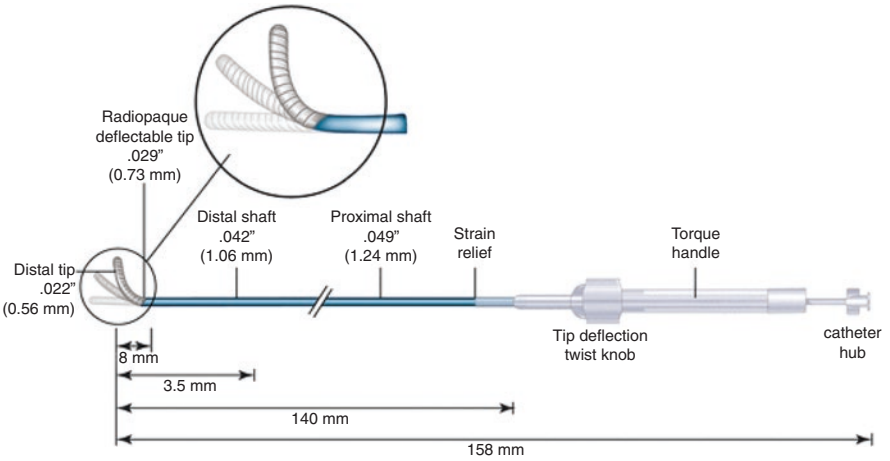

Fig. 10.15 Venture catheter: a deflectable tip that helps in directing the wire in angulated side branches. The tip can be deflected by rotating the knob

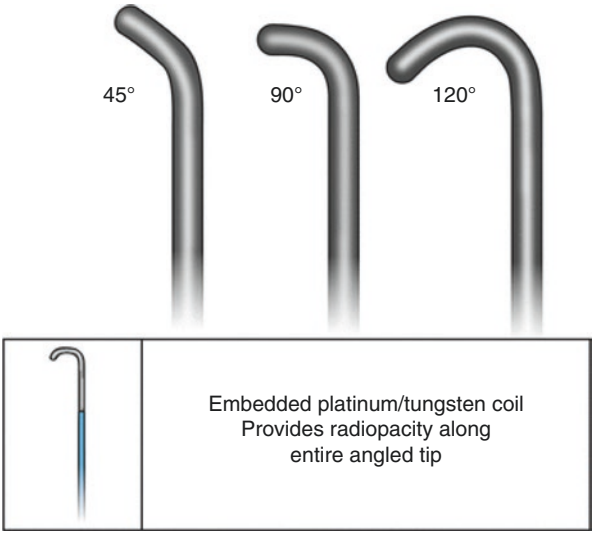

Fig. 10.16 SuperCross catheter: preshaped microcatheter to wire angulated side branches

  - Pull back the catheter until the ostium of the side branch.
  - Once the Venture™ catheter is at the origin of the side branch, advance the wire.
- Complications:
  - Dissection of the main vessel.
  - Dissection of the side branch.

SuperCross™ Catheter

- Has prefixed tip angles. The most commonly used angles are 90° and 120° (Fig. 10.16).

- Rapid exchange system.
- Selected depending on the angle of the side branch.
- How to use:
  - Advance the wire beyond the side branch origin in the main vessel.
  - Advance the SuperCross catheter on the wire, beyond the side branch.
  - Pull back the wire and the tip of SuperCross will assume its preshaped angle.
  - Pull back the SuperCross and orient it to the origin of the side branch.
  - Advance the wire into the side branch.
- Complications:
  - Dissection of the main vessel.
  - Dissection of the side branch.

SwiftNINJA™ Catheter

- A straight tip catheter that articulates up to 180° in opposing directions (very similar to Venture™ catheter) (Fig. 10.17).

Fig. 10.17 Swiftninja catheter: deflectable tip up to 180 in opposing directions, which helps in directing the wire in angulated side branches. The tip can be deflected by rotating the steering dial

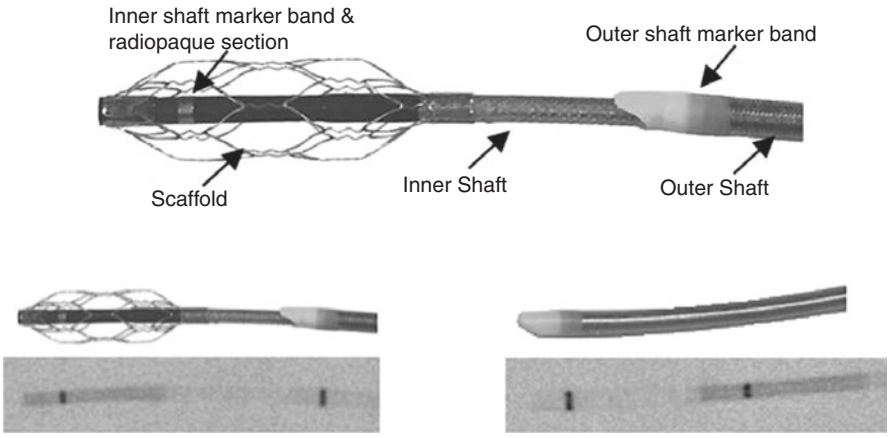

Fig. 10.18 Centercross catheter is a re-sheathable self-expanding scaffold to maximize support

Microcatheters for Further Support by Anchoring

Microcatheters: For Anchoring Support

- Prototypes: Multicross™, CenterCross™, Prodigy™, and NovaCross™.
- A self-expanding scaffold anchors our catheter near the proximal cap, designed to provide support for accurate centering and staying true lumen (Fig. 10.18).
- Amplifies tip penetration force to 5x that of a microcatheter and 0.014″ guidewire.
- How to use:
  - Use the radiopaque distal sections of the catheter to assess the location of the catheter tip.
  - To deploy the scaffold, slowly pull back on the thumb lever/prime port, while keeping the handle fixed.
  - Minimum guide catheter 7 F.

Microcatheters: CTO Catheters

CROSSBOSS™ Coronary CTO Crossing Catheter

- Blunt-tipped catheter to pass the occlusion and for subintimal entry or the true lumen.
- Facilitated Antegrade Steering Technique in Chronic Total Occlusions.

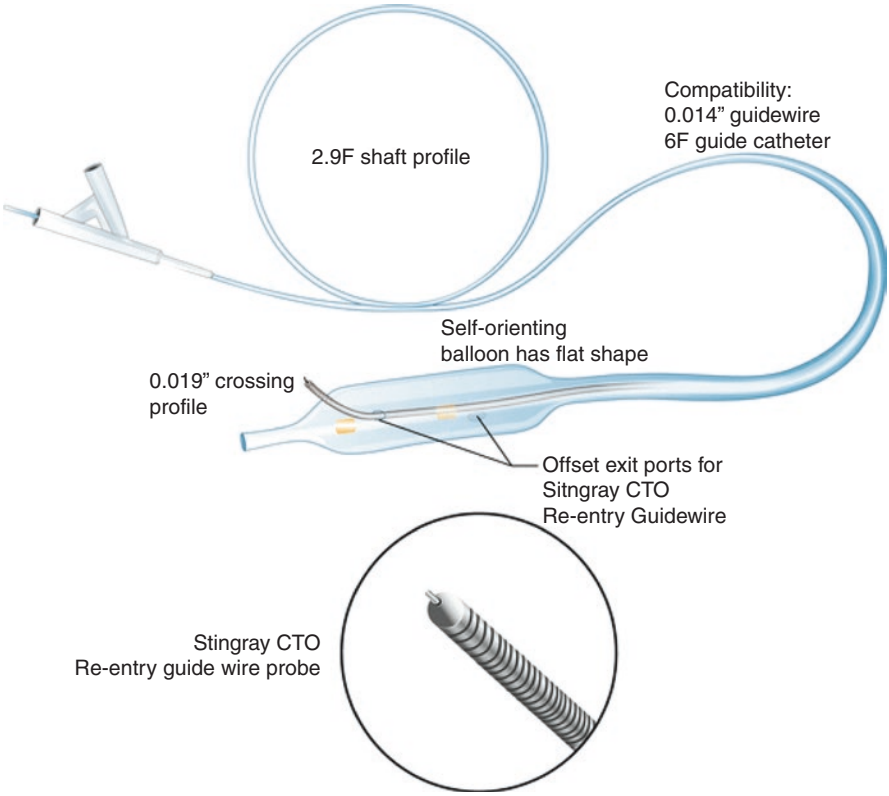

Fig. 10.19 Stingray catheter with a self-orienting flat balloon and a re-entry port

Stingray™ Coronary CTO Re-Entry System

- Flat-shaped balloon with side exit holes (Fig. 10.19).
- Guidewire: a small-diameter wire with an angled and sharpened tip for re-entry into the true lumen.
- Inflate Stingray™ balloon up to 4 atm in subintimal space, and attempt re-entry into the true lumen with a guidewire.

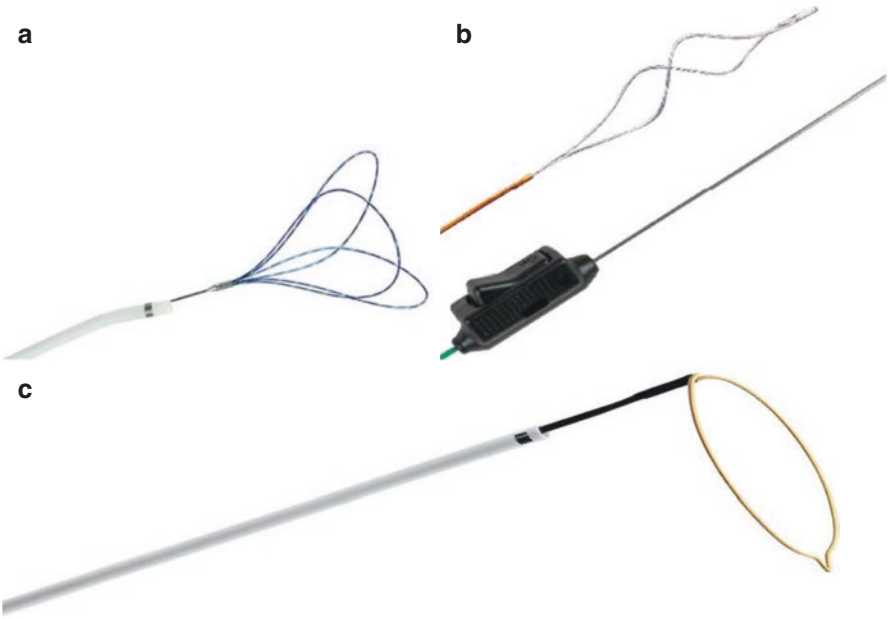

Fig. 10.20 Three different models of snare have been depicted: (**a**) EN snare. (**b**) Microelite snare. (**c**) Goose neck snare

Snares

Prototypes

- Micro Elite™ Snare.
- Goose Neck™ Snare.
- En Snare™ (Multiple loops) (Fig. 10.20).

Use

- Capture retrograde wire in CTO.
- Retrieve foreign objects intracoronary.

11 Hemodynamic Assessment: Right Heart Catheterization, Pulmonary Hypertension, Left-to-Right Shunt, Constriction, and Restriction

Tarun Jain, Annapoorna Kini, and Matthew Tomey

Introduction

Hemodynamic assessment is a critical component of cardiac catheterization. Although advances in cardiovascular imaging today offer many tools for noninvasive hemodynamic assessment, invasive hemodynamic assessment in the cardiac catheterization laboratory remains essential for accurate diagnosis and measurement, particularly when noninvasive imaging data are of suboptimal quality or indeterminate interpretation.

Right Heart Catheterization

Indications [1]

- Evaluation of valvular heart disease.
- Diagnosis of pulmonary hypertension and evaluation of treatment responsiveness.
- Evaluation of left-to-right shunt.
- Differentiation of constrictive and restrictive physiology.
- Evaluation of complex congenital heart disease (outside the scope of this text).

Contraindications and Cautions [1]

- Contraindications: vegetation, tumor, or thrombus in the right heart; mechanical prosthesis in the tricuspid or pulmonary position.

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https://doi.org/10.1007/978-3-030-68538-6_11

- Cautions: coagulopathy, severe pulmonary arterial hypertension, pre-existing left bundle branch block, passive-fixation pacing lead (e.g., coronary sinus lead), implanted pulmonary arterial pressure monitor (CardioMEMS).

Equipment

- 7–8 Fr sheath access kit.
- Multi-lumen pulmonary artery (PA) catheter (alternatively, wedge catheter with 5–6 Fr sheath).

Access

- 7–8 Fr femoral or internal jugular vein; 5–7 Fr antecubital (medial preferred).

Fluoroscopic Views

- Straight anterior-posterior (AP)

Steps for Standard Right Heart Catheterization

- Insert the PA catheter into the sheath. Advance the PA catheter to its 20 cm marker and inflate the balloon. A leading 0.035″ J-tipped wire, a 0.018″ Platinum Plus, a Swan wire, or a 0.014″ support wire (for antecubital approach, such as the Grand Slam or Iron Man) may be used if needed. Be cognizant that the catheter can sometimes exit into smaller venous branches prior to reaching 20 cm, requiring caution in balloon inflation.
- Advance the PA catheter into the right atrium (RA). Flush the system and zero the pressure transducer. Record the RA pressure.
- Advance the catheter into the right ventricle (RV) and record the pressure.
- Advance the catheter into the PA. From the internal jugular approach, a catheter with a C-shaped curvature will often progress easily from the RV to the PA with forward advancement. From the femoral approach, transmission of clockwise torque with a gentle backward motion will usually direct the catheter tip cephalad toward the RV outflow tract and allow for advancement into the PA.

Troubleshooting

- Variations in right heart anatomy and tricuspid regurgitation can contribute to difficulty in accessing the PA, particularly from the femoral approach. Several techniques can be tried:
  - *Deep inspiration*: Elongation of the mediastinum and negative intrathoracic pressure during a deep inspiratory effort can facilitate PA access.

  - *Looping*: Within the RA, direct the catheter toward the lateral wall of the RA, looping the catheter down and then across the tricuspid valve into the RV. The cephalad trajectory of the catheter across the tricuspid valve will often direct it toward the RV outflow tract.
  - *Stiffen the catheter*: As time within the body prolongs, the warm temperature of blood will soften the catheter. Techniques to stiffen the catheter—maintaining its shape and enhancing 'pushability'—include insertion of a wire (kept entirely within the catheter) or removal of the catheter from the body, flushing it, and bathing it in cold saline before reinsertion and reattempt. When the initial catheter used is a smaller caliber catheter (e.g., a 5 Fr Wedge catheter), upsizing to a larger catheter can also improve pushability.
  - *Reshaping*: In cases of RA enlargement, the radius of the preformed curve of the PA catheter may be too small, predisposing to undesired looping in the RA. Bending the curve outside the body to enlarge the radius of the catheter curve can overcome this issue.
  - *Wiring*: A wire (dedicated Swan wire; Platinum Plus; stiff coronary guidewire) can also be advanced beyond the tip of the PA catheter to carefully engage the PA, permitting subsequent tracking of the catheter into the PA. Care is required in this technique to avoid wire perforation of the RV or distal PA branches as well as arrhythmogenesis, noting the characteristic irritability of the RV outflow tract. A gentle curve on the wire tip may facilitate both safety and directability.
- An appropriate pulmonary capillary wedge pressure (PCWP) tracing should be recorded with an appropriate transition to PA waveform when the balloon is deflated.
  - Of note, stored forward tension in the catheter is released upon balloon deflation, causing a variable degree of additional forward advancement in the catheter tip. This phenomenon often obligates retraction of the catheter by a few centimeters after completion of PCWP recording to avoid remaining in a wedged or partially wedged position.
  - Conversely, this phenomenon can be exploited to help further advance the PA catheter when initial advancement does not obtain a PCWP waveform.

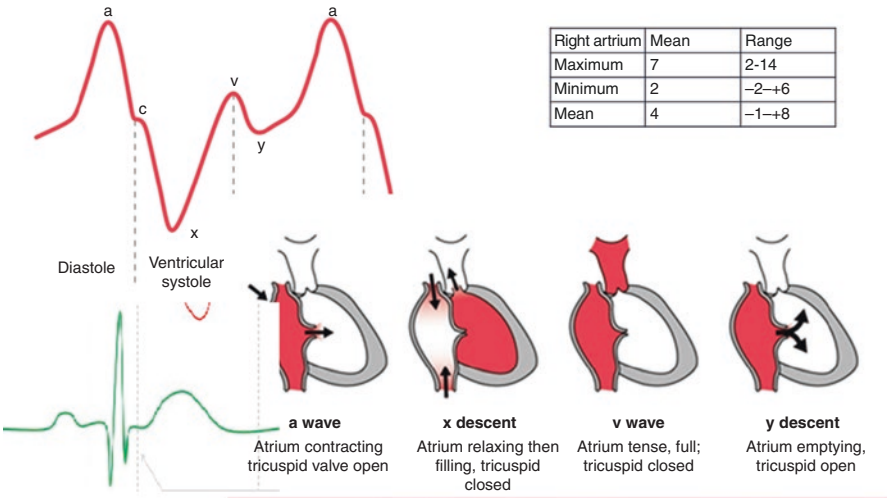
    In this technique, the balloon is subsequently slowly reinflated with continuous attention to the waveform, ceasing further inflation once an appropriate PCWP waveform is obtained; this may require less than the full 1.5 cc of air in the syringe.

    If an appropriate waveform still cannot be obtained, the catheter can be retracted with an attempt to position the catheter in a different PA branch.
  - The PCWP is reported as the mean a-wave pressure at end-expiration.
- Aspirate blood from the PA to permit measurement of oxygen saturation. For Fick estimation of cardiac output (CO), systemic arterial oxygen saturation may be measured directly (if arterial access is present) or derived from pulse oximetry.
- If using a PA catheter connect the cable for thermodilution measurement of cardiac output and firmly inject 10 cc of saline in the proximal port (repeat two to three times). Thermodilution method may be omitted if severe tricuspid/pulmonic regurgitation is present.
- If severe pulmonary hypertension is present that is unexplained by pulmonary venous hypertension (normal PCWP), consider vasoreactivity testing (see below).

- If PA saturation is >75% on repeated measurement and is not otherwise well explained (e.g., by a high cardiac output state), an oxygen saturation run should be performed for detection and quantification of a left-to-right shunt (see below).

Understanding Standard and Normal Right Heart Pressure Tracings (Table 11.1)

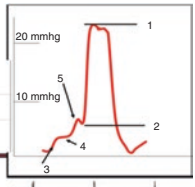
Right Atrial Pressure



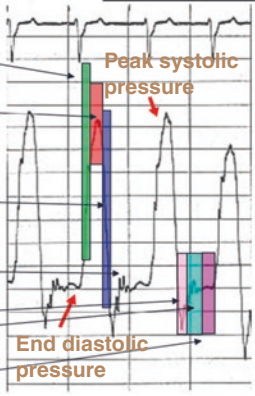
Right artrium	Mean	Range
Maximum	7	2-14
Minimum	2	–2–+6
Mean	4	–1–+8

Right Ventricular Pressure

Right ventricle	Mean	Range
Systolic	24	15-28
End-diastolic	4	0–8

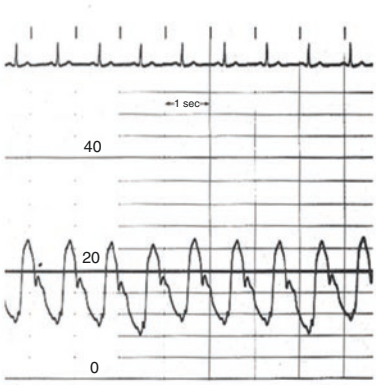


- Systole
  - Isovolumetric contraction
    - From TV closure to PV opening
  - Ejection
    - From PV opening to PV closure
- Diastole
  - Isovolumetric relaxation
    - From PV closure to TV opening
  - Filling
    - From TV opening to TV closure
    - Early Rapid Phase [3]
    - Slow Phase [4]
    - Atrial Contraction (“a” wave) [5]



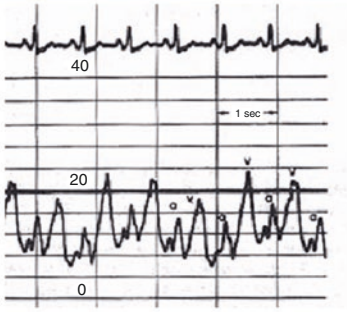
Pulmonary Artery Pressure

- Biphasic tracing
  - Systole
  - Diastole



Pulmonary artery	Mean	Range
Systolic	24	15-28
Diastolic	10	5-16
Mean	16	10-22

Pulmonary Capillary Wedge Pressure



- "a" wave
  - Atrial systole
- "c" wave
  - Protrusion of MV into LA
- "x" descent
  - Relaxation of LA
  - Downward pulling of mitral annulus by LV contraction
- "v" wave
  - LV contraction
  - Height related to atrial compliance & amount of blood return
  - Higher than a wave
- "y" descent
  - MV opening and LA emptying into LV

Pulmonary artery wedge ("PC")	Mean	
Maximum	16	9-23
Minimum	6	1-12
Mean	9	6-15

Left Ventricular Pressure

- Systole
  - – Isovolumetric contraction
    - From MV closure to AoV opening
  - – Ejection
    - From AoV opening to AoV closure
- Diastole
  - – Isovolumetric relaxation
    - From AoV closure to MV opening
  - – Filling
    - From MV opening to MV closure
    - Early Rapid Phase
    - Slow Phase
    - Atrial Contraction ("a" wave")

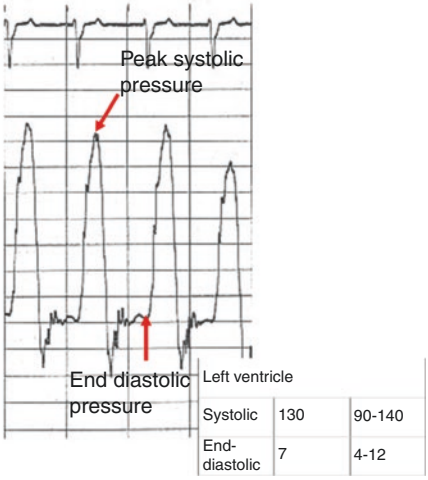


Table 11.1 Suggested guidelines for various hemodynamic pressures

Variable	Degree of severity	Value
Pulmonary systolic pressure	Normal	<30 mmHg
	Mild hypertension	31–50 mmHg
	Moderate hypertension	51–70 mmHg
	Severe hypertension	>70 mmHg
Pulmonary mean pressure	Mild hypertension	25–34 mmHg
	Moderate hypertension	35–54 mmHg
	Severe hypertension	>45 mmHg
Pulmonary capillary wedge pressure	Normal	<15 mmHg
	Increased	15–22 mmHg
	Markedly increased	>22 mmHg
Cardiac index	Normal	2.5–4.2 L/min/m^2
	Increased	>4.2 L/min/m^2
	Decreased	1.8–2.5 L/min/m^2
	Extremely low	<1.8 L/min/m^2
Pulmonary vascular resistance index	Normal	<5 Wood
	Mildly increased	5–-7 Wood units
	Moderately increased	8–14 Wood units
	Severely increased	>14 Wood units

Steps for Evaluation of Pulmonary Hypertension

- Perform standard right heart catheterization as described above.
- Calculate the transpulmonary gradient (TPG) and pulmonary vascular resistance (PVR).
  - – *Transpulmonary gradient (TPG) = Mean PAP – PCWP*
  - – *Pulmonary vascular resistance (PVR) (Woods) = TPG (mmHg)/CO (L/min)*

  - *Pulmonary vascular resistance index (PVRI) (Woods Units) = PVR/body surface area*
- If there is doubt regarding the accuracy of the wedge pressure, a simultaneous left ventricular end-diastolic pressure should be measured via left heart catheterization.
- If pulmonary arterial hypertension is present, defined by mean PA pressure > 25 mmHg and a PVR >3 Wood units, and PCWP (and/or LVEDP) <15 mmHg, then acute vasodilator testing should be performed [2].
- For vasoreactivity testing, we administer inhaled nitric oxide at a dose of 40 parts per million (ppm) for 5 min, with continuous hemodynamic monitoring [3, 4].
  - Responders (or reactivity) to vasodilator testing (for purposes of initiating calcium channel blocker therapy) are defined as demonstrating a decrease in mean PAP by 10 mmHg to a mean PAP less than 40 mmHg without a decrease in cardiac output.

Steps for Evaluation of Left-to-Right Shunt

- Perform standard right heart catheterization as described above.
  - Oxygen saturation can be measured during the advancement or pullback of the catheter.
  - When a left-to-right shunt is not clinically suspected, a screening PA oxygen saturation should be measured; if the value is >75% and unexplained by a high cardiac output state and/or AV fistula, a complete saturation run should be performed.
- Oxygen saturation should be obtained from the pulmonary artery, right ventricle, right atrium, inferior vena cava, and superior vena cava. A difference between two chambers exceeding 5–7% is considered significant [5].
- Calculation of shunt fraction:
  - Use the difference in oxygen saturation to estimate the ratio of flow across pulmonic (Q_p) and systemic circulatory (Q_s) beds:

$$\mathrm{Qs} = \frac{\mathrm{VO_2}}{\text{Systemic AV O}_2\text{ difference}}$$

$$\mathrm{Qp} = \frac{\mathrm{VO_2}}{\text{Pulmonary VA O}_2\text{ difference}}$$

$$\mathrm{Qp/Qs} = \frac{\text{systemic AV O}_2\text{ difference}}{\text{pulmonary VA O}_2\text{ difference}}$$

$$\mathrm{Qp:Qs} = \frac{\mathrm{SAO_2 - MVO_2}}{\mathrm{PVO_2 - PAO_2}}$$

$$\mathrm{MVO_2} = \frac{[3\mathrm{SVC} + 1\mathrm{IVC}]}{4}$$

- With few exceptions, arterial saturation can be used as a surrogate for pulmonary venous saturation.
- Mixed venous saturation is calculated by
  - *Flamm equation as (3 SVC saturations + 1 IVC saturation)/4.*
  - Otherwise, it is assumed equivalent to the saturation from the chamber proximal to the suspected defect (saturation before 'step up'), and the IVC saturation can generally be excluded due to variability in measurement related to streaming and relative contribution to the average mixed venous saturation.
- Exceptions to use of SVC saturation for mixed venous saturation measurement:
  - Anomalous pulmonary venous return above the SVC/RA junction.
  - Arteriovenous fistula or malformation above the SVC/RA junction.
  - In general, these pitfalls can be overcome by measurement of venous saturation proximal to (above the level of) the shunts, if possible.
- Shunt calculation is not reliable in patent ductus arteriosus due to the distal site of shunting.

Steps for Evaluation of Constriction Versus Restriction

- Perform standard right heart catheterization as described above.
- Perform standard left heart catheterization, ideally with a pigtail catheter in the left ventricle.
- After zeroing both transducers and documenting simultaneous PCWP/LVEDP, deflate the PA catheter balloon and withdraw it slowly, until it falls into the right ventricle. The balloon can be reinflated in the RV to reduce ectopy.
- Slow the sweep speed and equalize the scales for measurement of simultaneous LV and RV pressures.
- If RA pressure was <15 mmHg, administer 1 L of normal saline (fluid challenge).
- Hemodynamic tracings of LV/RV pressures typically demonstrate a "dip and plateau" or "square root sign" and, in constriction, usually with equalization of diastolic pressures.
- Hemodynamic criteria suggestive of constriction are included in Table 11.2 [6].
- In constriction, during inspiration, there is an increase in the area of the RV pressure curve (compared with expiration), and due to interventricular dependence, there is a simultaneous decrease in the area of the LV pressure curve leading to LV-RV respiratory discordance (Fig. 11.1) [7].
- Systolic area index >1.1 is highly suggestive of constriction [6].

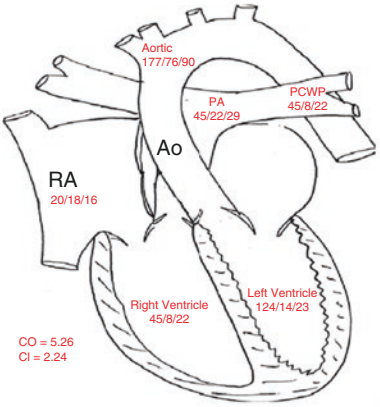
Table 11.2 Hemodynamic criteria suggestive of constriction

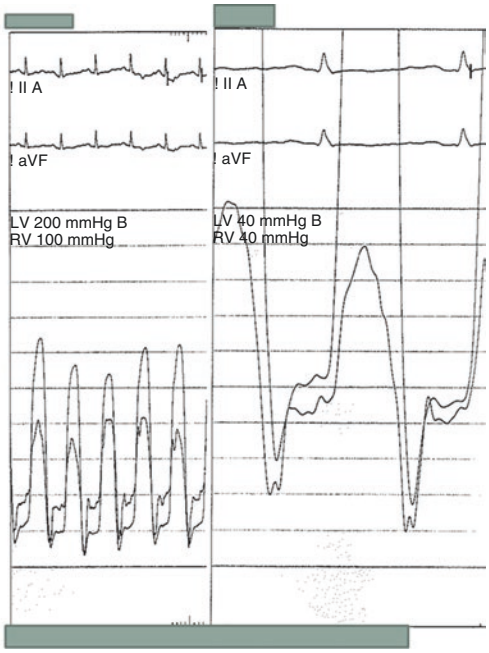
	Constriction	Restriction
LVEDP–RVEDP (mmHg)	≤5	>5
PASP (mmHg)	≤55	>55
RVEDP/RVSP	>1/3	<1/3
Inspiratory fall in RAP (mmHg)	<5	>5
Inspiratory decrease in PCWP > LVDP	Present	Absent
Systolic area index	>1.1	<1.1

1. Diastolic equalization of pressures

LVEDP-RVEDP ≤5 mm Hg

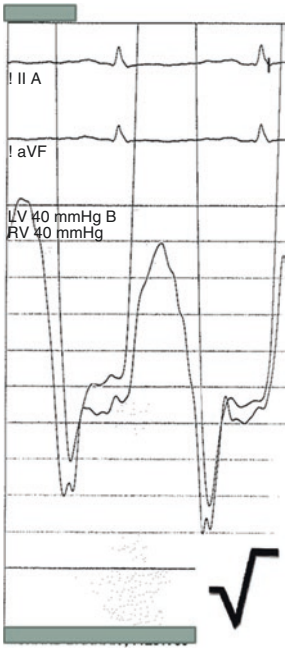
23-22=1





Dip and Plateau diastolic pressure wave (√Sign)

High ventricular Rapid filling wave ≥ 7 mm Hg



Narrow RV Pulse pressure

RVEDP/RVSP > 1/3 **[22/45= 1/2]**

Right Ventricle
45/8/22

Fig. 11.1 Simultaneous RV-LV tracing in constrictive pericarditis showing discordance vs concordance in restrictive cardiomyopathy. (Reprinted from Geske et al. [7] with permission from Elsevier and Journal of the American College of Cardiology)

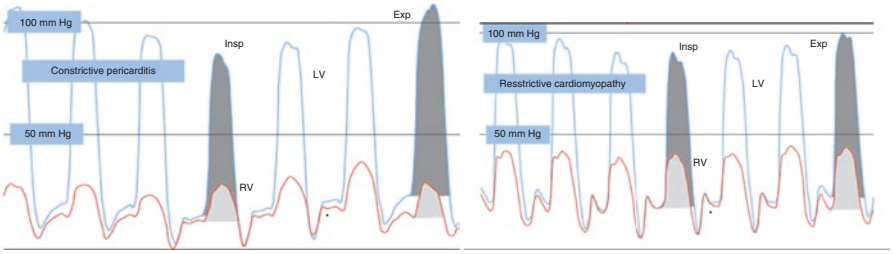


Fig. 11.1 (continued)

Systolic area index = RV area/ LV area in inspiration ÷ RV area/LV area in expiration

Case Depiction of Constrictive Pericarditis Physiology by Cardiac Catheterization

Complications

- While complications are generally rare (<1%), the most common complications are access related (hematoma, pneumothorax).
- Additional adverse events may include usually transient arrhythmia due to catheter stimulation, vagal-induced hypotension, or reactions to vasoreactivity testing.
- Vasoreactivity testing should be generally avoided in patients with significant, decompensated left heart disease or veno-occlusive disease due to the risk of pulmonary edema.

Post-Procedural Care

- Manual compression for hemostasis and routine post-procedural monitoring of vital signs.

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Hemodynamic Assessment of Valvular Stenosis and Regurgitation

12

Reza Masoomi, Gurpreet S. Johal, and Annapoorna Kini

Introduction

Accurate hemodynamic measurements are important for the accurate assessment of valvular lesions, especially when the decision for invasive therapy has to be made. Though guidelines recommend imaging modalities as the standard for valvular assessment, the catheterization laboratory remains important for accurate measurements and the confirmation of diagnoses, especially in patients with suboptimal or equivocal imaging results.

Aortic Stenosis

Indications

- Evaluation of aortic stenosis of uncertain severity and/or in preparation for balloon aortic valvuloplasty, and surgical or transcatheter aortic valve replacement.

Contraindications

- Mechanical aortic valve, active vegetation, and LV thrombus (see Chap. 11).

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Equipment

- 7.5 and 6 Fr sheaths
- Pulmonary artery catheter or wedge catheter
- 5 Fr AR2 catheter
- Optional: dual-lumen pigtail catheter (Langston®)
- Straight-tip Terumo GlidewireTM
- Manifolds (3) and transducers

Access

- 7.5 Fr venous, 6 Fr arterial

Fluoroscopic Views

- Left anterior oblique [LAO] (for aortography, crossing)

Hemodynamics Findings

- There is a significant gradient across the aortic valve.
- There is a delay in aortic pressure upstroke with a prominent anacrotic notch.
- Carabello sign can be seen in severe aortic stenosis (rise in aortic pressure >5 mmHg when the catheter is withdrawn from LV).

Steps

- Perform a right heart catheterization, obtaining right atrial (RA), right ventricular (RV), pulmonary artery (PA), and pulmonary capillary wedge (PCW) pressures, along with PA saturation for calculation of cardiac output (CO), optional measurement of CO by thermodilution method. Leave the PA catheter in the pulmonary artery.
- Connect the femoral artery (FA) sheath to pressure. Advance a 5Fr AR2 diagnostic catheter (over a standard 0.035″ J-wire) to the ascending aorta. Flush the catheter and zero all transducers simultaneously. Record simultaneous pressures from the FA sheath and AR2 catheter. If the gradient is >10 mmHg, consider exchanging for a dual-lumen pigtail catheter (Langston®) to accurately evaluate the pressure gradient across the aortic valve (after crossing into the LV).
- Perform an aortogram in the (LAO) projection (20–25°) to elucidate the valve orifice and degree of aortic regurgitation.
- Administer heparin 2000 units IV/IA.

- In the LAO projection, use the AR2 catheter (AL catheter in a horizontal aorta) to direct a straight-tip Terumo Glidewire™ across the aortic valve; Probing the wire through the stenotic orifice with a slow pull back with clockwise rotation of catheter.
- Upon crossing with the wire, switch to the right anterior oblique [RAO] projection to confirm the position of the wire and then advance the AR2 catheter into the LV over the wire (be careful not to puncture the apex).
- Remove the wire, zero all transducers, and assess the LV-FA gradient. If a significant (>10 mmHg) FA-central aortic gradient was previously noted, exchange the AR2 catheter for a dual-lumen pigtail catheter (Langston®) over an exchange length 0.035″ J-wire.
- Measure peak-to-peak and mean gradient (automated computerized analysis of the area under the curve) (Fig. 12.1).
- Calculate the aortic valve area (AVA) using the Hakki or Gorlin formula (Fig. 12.2) [1, 2].

Hakki formula:

$$\text{Aortic valve area } \left(\text{cm}^2\right) \approx \frac{\text{Cardiac output } \left(\text{l/min}\right)}{\sqrt{\text{Peak to peak gradient } \left(\text{mmHg}\right)}}$$

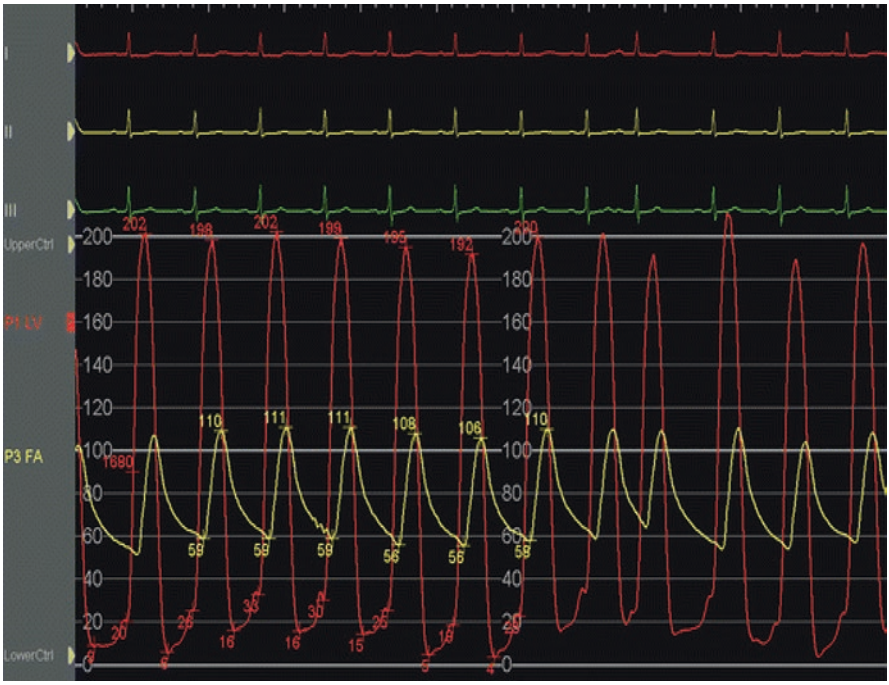

Fig. 12.1 Severe aortic stenosis (LV, aortic pressures)

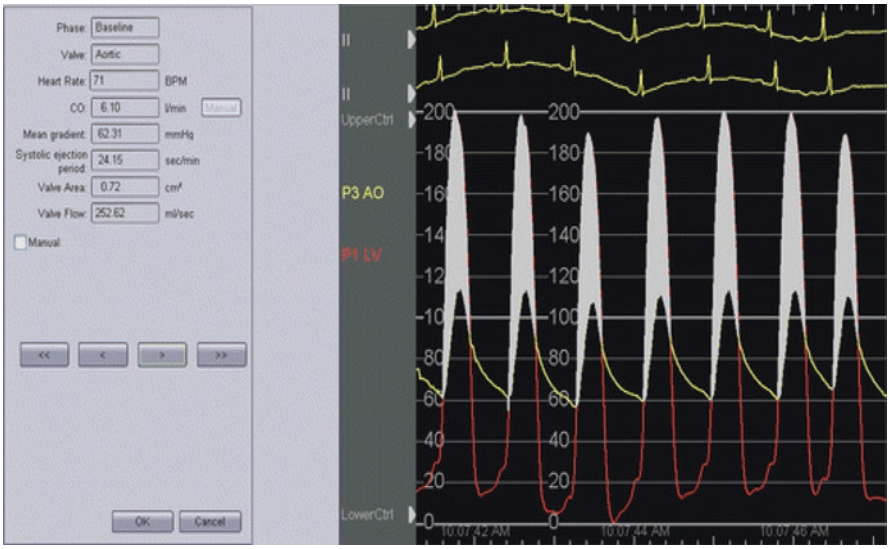

Fig. 12.2 Mean gradient in severe aortic stenosis and AVA (automated computerized calculation base on Gorlin formula)

Gorlin formula:

$$\text{Valve area}\left(\text{cm}^2\right)=\frac{\text{Cardiac output}\left(\text{ml}/\text{min}\right)}{\text{Heart rate}\left(\text{beats}/\text{min}\right)\cdot\text{Systolic ejection period}\left(\text{s}\right)\cdot 44.3\cdot\sqrt{\text{mean gradient}\left(\text{mmHg}\right)}}$$

- If the mean LV-aortic gradient is <40 mmHg with AVA ≤ 1.0 cm^2 (low flow-low gradient AS) with the left ventricular ejection fraction (LVEF) <50%, consider low-dose dobutamine infusion (starting at 5 mcg/kg min, titrating up to 20 mcg/kg min) to differentiate true severe AS vs pseudo-severe AS. Repeat hemodynamics, reassessing pressure gradient and cardiac output, and recalculate the valve area. An increase in the valve area suggests more pseudo-stenosis secondary to LV dysfunction. If the mean gradient becomes ≥40 mmHg in the presence of an AVA ≤ 1.0 cm^2, it confirms severe AS [3]. This test also identifies the presence of contractile reserve (defined as an increase in stroke volume ≥20%), which has prognostic value.
- Record simultaneous LV-PCW pressure tracings. If no valvuloplasty is planned, document a pullback to ensure well-matched aortic pressure tracings (regardless of catheter used).
- Severity of AS:
  - Critical: AVA < 0.7 cm^2
  - Severe: AVA < 1 cm^2
  - Moderate: AVA 1–1.5 cm^2
  - Mild: AVA ≥ 1.5 cm^2

Complications

- Cerebrovascular accident, vascular.

Post-Procedure Care

- Routine post-catheterization care

Mitral Stenosis

Indications

- Evaluation of mitral stenosis severity and/or in preparation for balloon mitral valvuloplasty [4].

Contraindications

- Mechanical aortic valve, active vegetation, and LV thrombus; contraindications for right heart catheterization (see Chap. 12).

Equipment

- 7.5 Fr and 6 Fr sheaths.
- Pulmonary artery catheter or wedge catheter.
- Manifolds (3).

Access

- 7.5 Fr venous, 6 Fr arterial.

Fluoroscopic Views

- Antero-posterior.

Hemodynamic Findings

- PCWP and LV pressure should be closely superimposed but in the presence of stenotic MV, there is a significant gradient between those tracings.

- Chronic MS is associated with markedly increased pulmonary arterial pressure due to pulmonary vasculature remodeling.
- Large A wave is seen due to increased residual volume in the left atrium and a large V wave is seen due to reduced left atrium compliance and increased residual volume in the left atrium.
- Since pulmonary capillary wedge pressure is a substitute for left atrial pressure, some scenarios may lead to falsely elevated PCWP and gradient in the absence of real mitral stenosis.
- Measuring not accurate PCWP due to partial wedging, which mix PA and PCWP pressures> check with wedge saturation.
- Severe MR due to increased flow across the valve and large v wave can overestimate the gradient.
- Pulmonary vein stenosis (usually in the setting of prior procedures like A-fib ablation).
- Pulmonary venoconstriction > administration of nitroglycerin to rule out falsely elevated gradient caused by pulmonary veno-constriction.

Steps by Step Approach

- Perform a right heart catheterization, obtaining right atrial (RA), right ventricular (RV), pulmonary artery (PA), and pulmonary capillary wedge (PCW) pressures, along with PA saturation for calculation of cardiac output (CO), optional measurement of CO by thermodilution method. Leave the PA catheter in the pulmonary artery.
- Advance a 5 Fr pigtail catheter (over a standard 0.035″ J-wire) to the left ventricle. Flush the catheter and zero all transducers simultaneously. Record simultaneous pressures from the LV and PCW positions (Fig. 12.3) to document the transmitral gradient (Fig. 12.4).
- Calculate the mitral valve area (MVA) using the Hakki (above) or modified Gorlin formula.

  Modified Gorlin formula:

$$\text{Mitral valve area} = \frac{\text{Cardiac output}}{\left(\text{Diastolic filling period}\right)\left(\text{Heart rate}\right)\left(37.9\right)\left(\sqrt{\text{Pressure gradient}}\right)}$$

- If mild mitral stenosis is present but there is a discrepancy between other imaging modalities or symptoms, consider exercise with continuous hemodynamic monitoring to evaluate for an increase in the gradient (>15 mm Hg) and/or a significant rise in pulmonary artery pressure (systolic >60 mmHg) and/or a significant rise in pulmonary capillary wedge pressure (≥25) (Figs. 12.5, 12.6, 12.7, and 12.8).

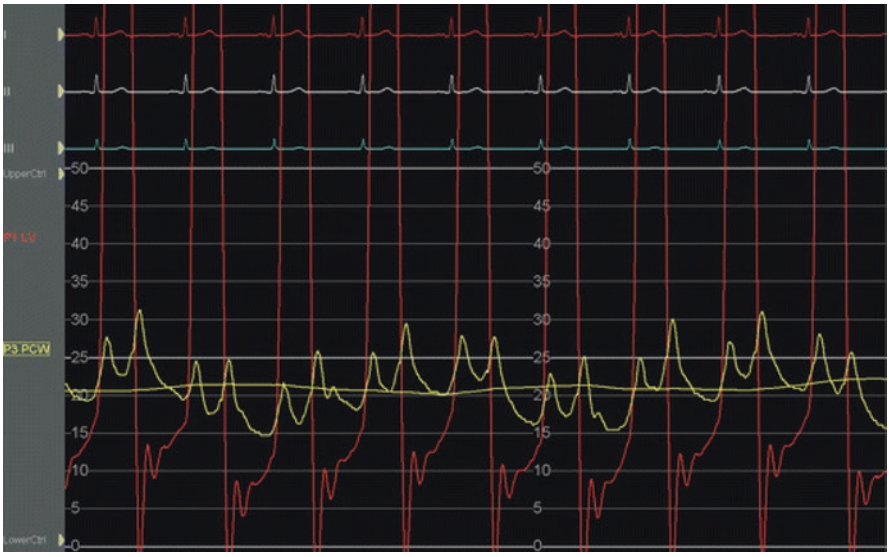

Fig. 12.3 Resting hemodynamics in severe mitral stenosis (LV, PCW pressures)

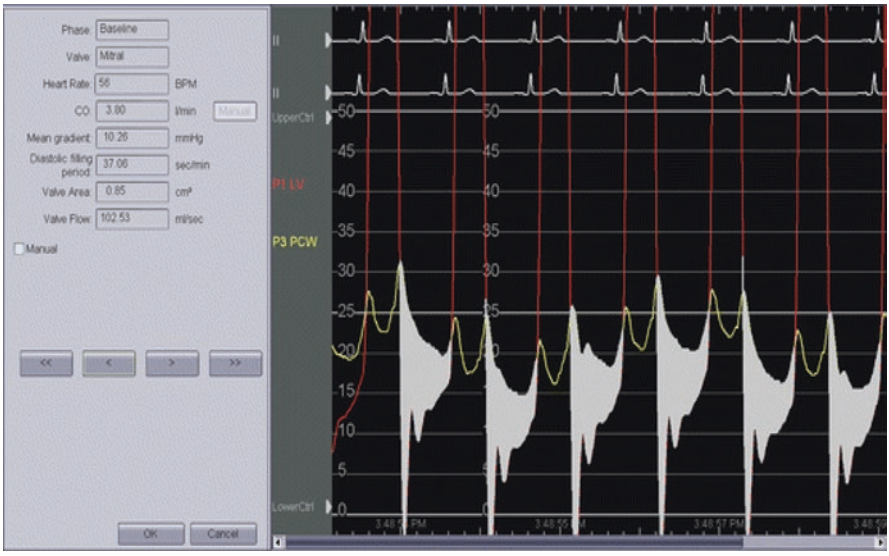

Fig. 12.4 Resting mean gradient and MVA (automated computerized calculation base on Gorlin formula)

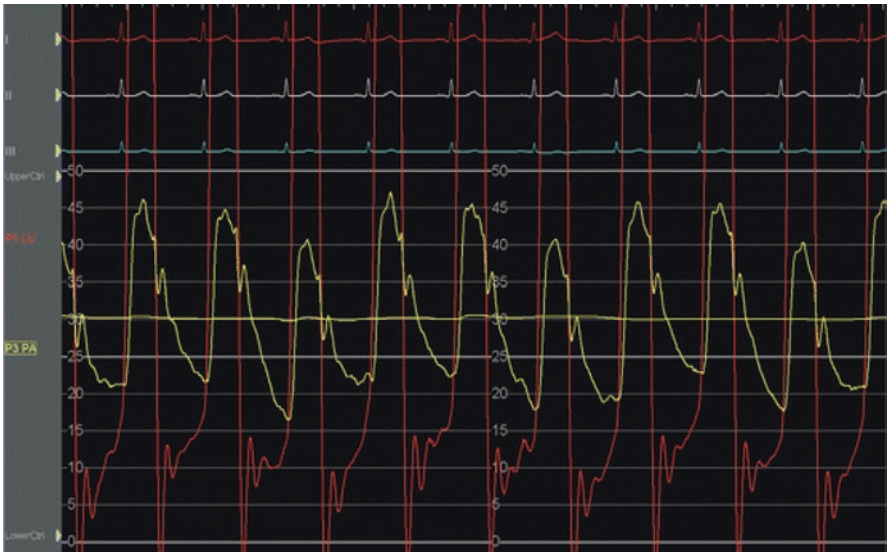

Fig. 12.5 Resting pulmonary artery pressure in severe MS

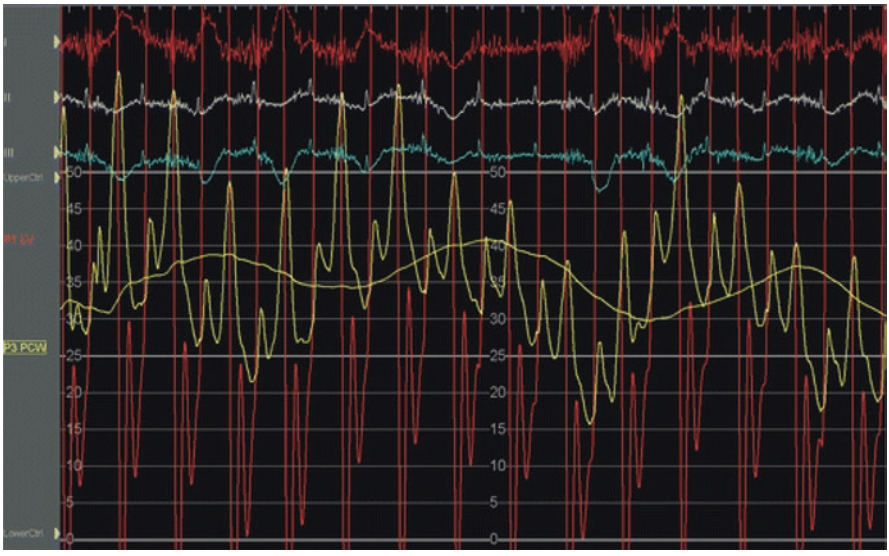

Fig. 12.6 Exercise hemodynamics in severe mitral stenosis (LV, PCWP)

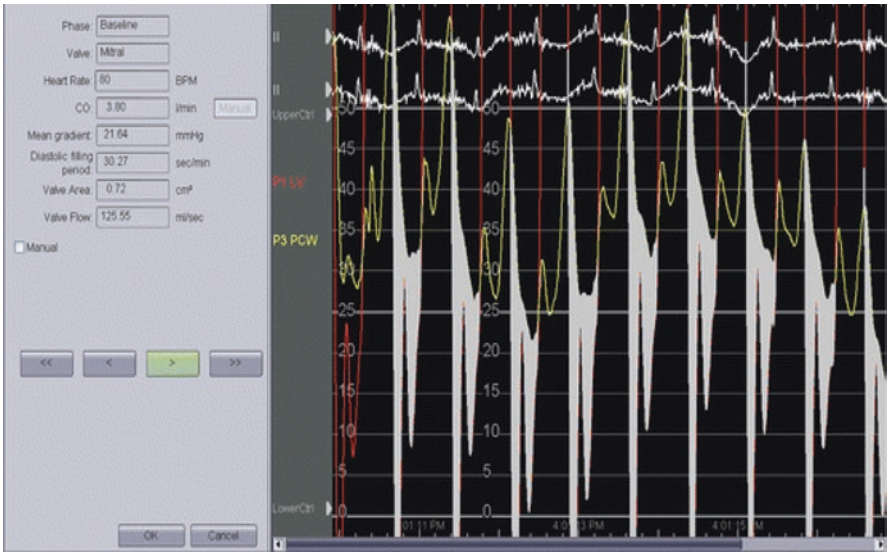

Fig. 12.7 Exercise mean gradient in mitral stenosis

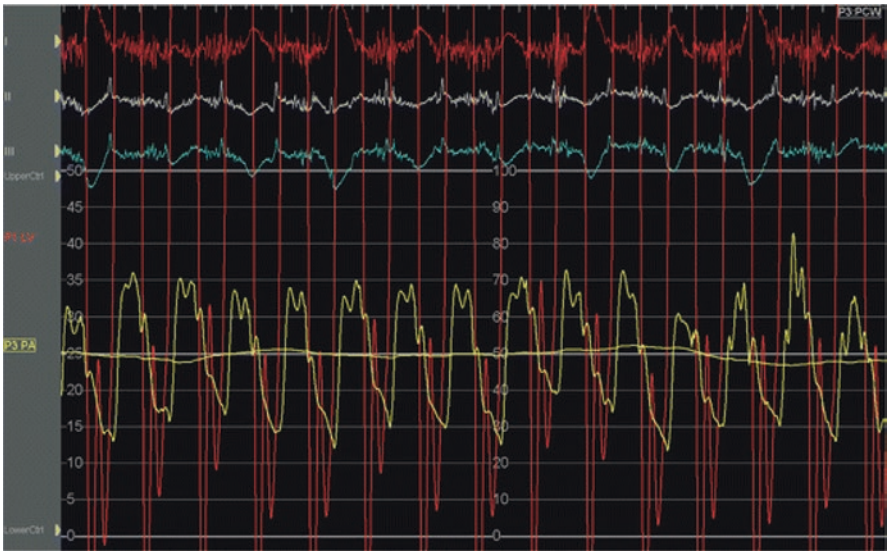

Fig. 12.8 Exercise pulmonary pressures in severe MS

- Perform a left ventriculogram to evaluate for mitral regurgitation (30 cc volume for 20 cc/s @ 450 PSI, RAO 30–45).
- Transseptal catheterization and measurement of left atrial pressure are required if the diagnosis is unclear and mechanical aortic valve or LV thrombus is present.

- Intrapulmonary nitroglycerin (200 mcg) may be injected to rule out a falsely elevated gradient caused by pulmonary venous hypertension.
- Severity of MS:
  - Mild: MVA 2–2.5 cm^2
  - Moderate: MVA 1.6–2.0 cm^2
  - Severe: MVA 1–1.5 cm^2
  - Very severe: MVA < 1.0 cm^2

Complications

- Cerebrovascular accident, vascular.

Post-Procedure Care

- Routine post-catheterization care.

Mitral and Aortic Regurgitation

Indications

- Evaluation of valvular regurgitation of uncertain severity and/or in preparation for possible surgical/transcatheter repair or replacement.
- The semi-quantitative assessment of valvular regurgitation with cardiac catheterization can be done with left ventriculography and aortic angiography.
- The angiographic regurgitation measurement is usually not accurate and practical.
- Right and left heart catheterization have limited diagnostic rules in quantification but those can give some hemodynamics clues like pulmonary hypertension, etc.

Contraindications

- LV ventriculography is contraindicated with very high LVEDP and not feasible with the presence of a mechanical aortic valve, active vegetation, and LV thrombus.

Equipment

- 5 or 6 Fr pigtail catheter.
- Power injector for large-volume contrast injection.

Access

- 5 or 6 Fr arterial.

Hemodynamics Findings

- Aortic regurgitation
  - Wide pulse pressure.
  - Diastasis manifesting as equalization of LVEDP and diastolic aortic pressure.
  - Increased LVEDP depending on the degree of decompensation and acuity of regurgitation.
- Mitral regurgitation
  - Prominent V waves.
  - Resting or exertional elevated PCWP and PA pressure.

Fluoroscopic Views

- Aortogram—LAO 30–45 (may require adjustment to ensure the left ventricle is adequately visualized).
- Left ventriculogram—RAO 30–50 (may require adjustment based on the course of aorta and size of ventricle/atrium in relation to the spine).

Steps for Aortography

- Advance a pigtail catheter over a wire in the usual fashion to the ascending aorta. Position the pigtail just above the sinotubular junction, to avoid contact with the valve.
- Ensure tubing and connections are free of air and are secure.
- The catheter should be held in position to avoid contact with the aortic valve or migration to an inappropriately high position in the ascending aorta.
- Settings for power injection: 40 cc of contrast, 15 cc/s flow, 750 PSI, 0.0 s rise.

Steps for Left Ventriculography

- Advance a pigtail catheter over a wire in usual fashion into the left ventricle, ensuring the wire (and, therefore, catheter) is free in the ventricular cavity and not entangled with the mitral valvular apparatus.
- Ensure tubing and connections are free of air and secure.
- The catheter should be held in position to avoid migration and ectopy or blow-back into the aorta.
- Settings for power injection: 40 cc of contrast, 15 cc/s flow, 650 PSI, 0.2 s rise.

- In patients with marked elevated LVEDP (>20 mmHg), administer nitroglycerin (200mcg) to reduce filling pressure before Left Ventriculography.
- Upon injection, panning the table may be required (usually pushing the table away from the operator–standing on the patient's right) to visualize regurgitation into particularly dilated atria.

Quantification of Regurgitation (MR or AI)

- 1+ dye regurgitation to the contralateral chamber without outlining of the chamber.
- 2+ dye regurgitation to the contralateral chamber with complete outlining of the chamber, but gradual dye clearance.
- 3+ dye regurgitation with progressive opacification of the contralateral chamber in 4–6 beats.
- 4+ dye regurgitation with instantaneous opacification of the contralateral chamber in <3 beats. In the case of mitral regurgitation, the pulmonary veins will be opacified.

Complications

- Cerebrovascular accident, vascular.

Post-Procedure Care

- Routine post-catheterization care

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Vascular Closure Devices and Complications

13

Reza Masoomi and Sahil Khera

Introduction

When compared to manual compression, utilization of vascular closure devices (VCDs) in the appropriate patient population has the potential to reduce time to post-procedure mobility and improve patient comfort [1–3]. However, it has not been clearly established if VCDs reduce overall complication rates when compared to manual compression. When utilizing these devices, one must weigh the potential complications associated with these devices against their potential benefits. This chapter will review VCDs commonly used with respect to their specific indications for use, contraindications, and safe deployment practices.

Perclose™ (Abbott Vascular, Abbott Park, IL): Suture-Based Device

Indications

- The vascular access site should be in the common femoral artery.
- The common femoral artery should be at least 5 mm in diameter.
- 6–8 F arteriotomy can be closed.
- >8 F arteriotomy can be closed with Perclose pre-deployment called Preclose.

Contraindications

- Common femoral artery luminal diameter less than 5 mm in diameter.

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- Significant peripheral vascular disease:
  - Significant luminal encroachment.
  - More than mild fluoroscopically visible calcification.

Deployment Steps

- Place a guidewire (0.038″ or less) through the procedural sheath and remove the sheath while applying pressure (Fig. 13.1a).
- Backload the device over the guidewire until the guidewire comes out of the device guidewire exit port. Use of gauze may help to grip the slippery hydrophilic distal portion of the device (Fig. 13.1b). Rail the device over the guidewire until the guidewire exit port is just above the skin line (Fig. 13.1c).
- Pull the guidewire out and continue to advance the device with gentle side-to-side rotating motion until (usually) brisk pulsatile flow of blood is evident from the marker lumen (Fig. 13.1d). Note that a slow trickle of blood may be seen if the device is incompletely inserted into the vessel. Continue to push the device forward—often a 'give' will be felt as the device is fully inserted.
- Position the device at a 45° angle (angle is usually the same angle used for initial needle entry while obtaining access) and deploy the foot by lifting the lever on the body of the device (Fig. 13.1e).
- Gently pull the device back to position the foot against the arterial wall (Figure Inset). Two things will be evident: (a) There will be firm resistance to further pull back and (b) blood flow from the marker lumen will stop (Fig. 13.1f).
- Stabilize the device with your left hand making sure that firm upward traction is maintained on the device, and with the right hand, deploy needles by pushing on the plunger assembly (Fig. 13.1g).
- While continuing to stabilize the device with the left hand, disengage the needles by pulling the plunger assembly back (Fig. 13.1h), and continue to pull the assembly back until the suture is taut (Fig. 13.1i). Loop the suture around the trimming mechanism located on the body of the device to cut it (Fig. 13.1j).
- Return the foot to its original closed position by pushing the lever down, and pull back on the device with a side-to-side rotating motion until the guidewire port is visible above the skin line. Pull the ends of the rail (long, blue) and lock (shorter, white tip) sutures from the device and secure them with clamps (or wet gauze), and reinsert the guidewire back in through the port (Fig. 13.1k). (PRECLOSE STEPS: The same above steps are used for Preclose. Make sure that the two sutures as secured with adequate slack to the sterile field using a clamp. A second Preclose may be deployed but turn the device clockwise 90° (like 10′ and 2′ o clock) for deployment as compared to the previously deployed Preclose. Then insert the sheath through the guidewire to perform the necessary intervention. At end of the procedure reinsert a long guidewire through the sheath, remove the sheath, and perform the following steps to close the arteriotomy site.).
- Wrap the rail limb (long, blue) of the suture around your left index finger, close to the skin level (Fig. 13.1l).

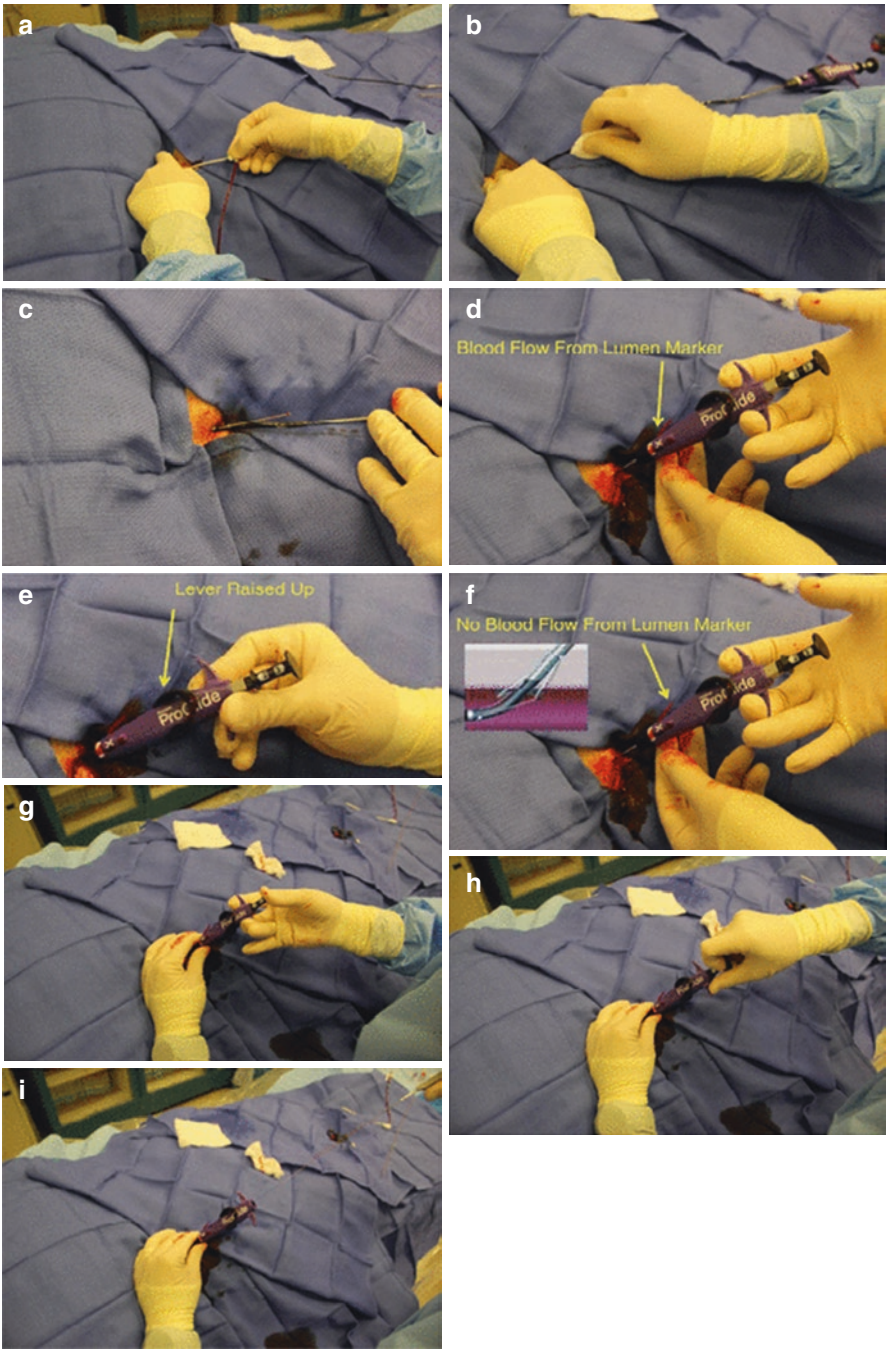

Fig. 13.1 Steps of Perclose closure device deployment

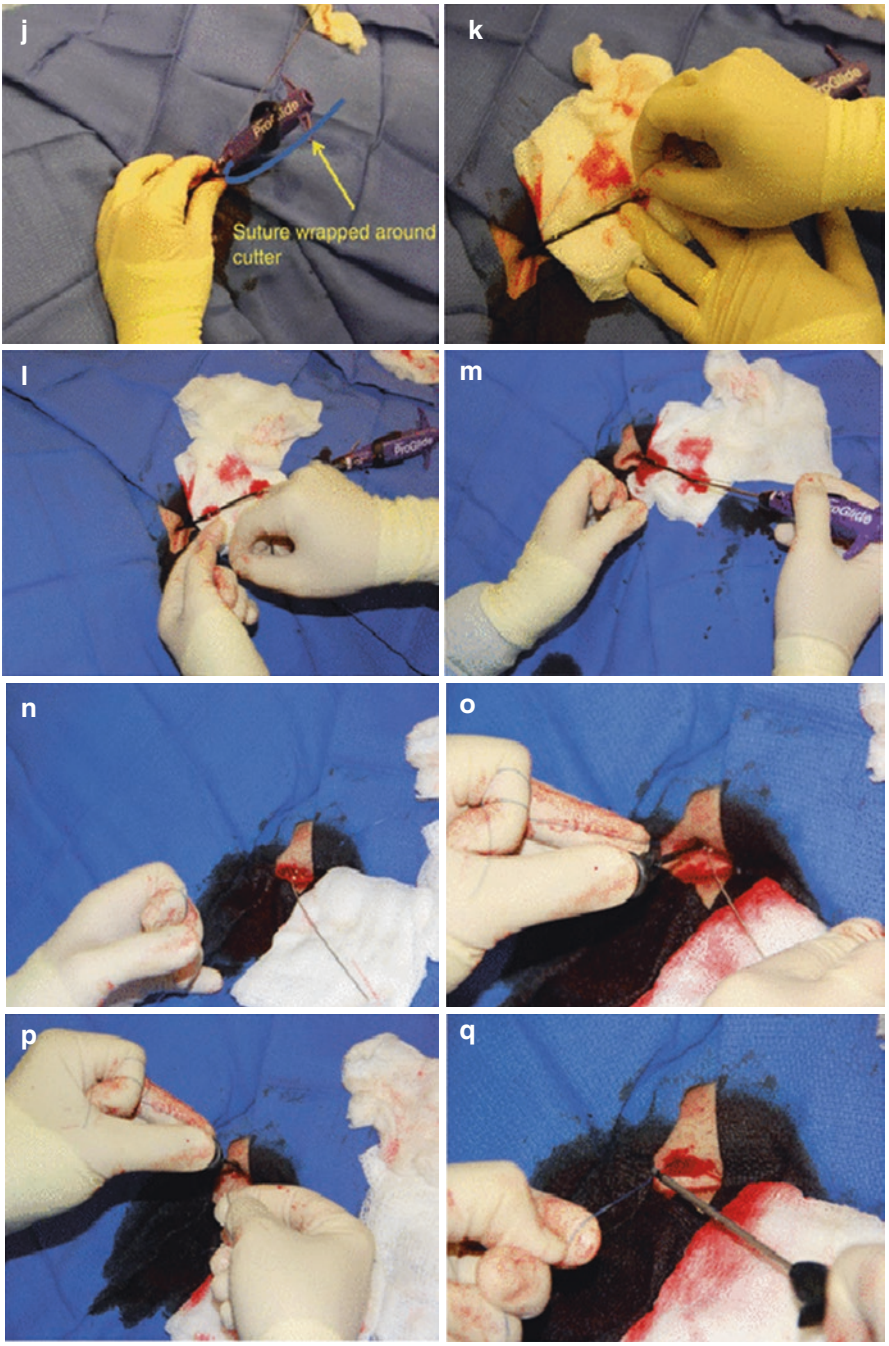

Fig. 13.1 (continued)

- Remove the device with the right hand, while maintaining an adequate length of guidewire inside the artery (Fig. 13.1m).
- While removing the device with the right hand, simultaneously advance the knot to the arteriotomy by applying slow, consistent increasing tension to the rail suture limb, keeping the suture coaxial to the tissue tract (Fig. 13.1n).
- If bleeding is controlled, the operator should then remove the guidewire.
- *If it is evident that hemostasis has not been achieved, the procedure should be aborted at this stage. The suture can be removed by a quick, firm tug on the lock limb of the suture—this will break the knot and allow removal of the entire length of the suture. A size 7.5 F (or greater) sheath should be inserted over the wire and manual compression performed when it is safe to do so.*
- Engage the Knot Pusher on the rail limb and push the slip knot to the arteriotomy (Fig. 13.1o). Once the knot is sufficiently pushed down to achieve hemostasis, lock the knot by pulling the lock suture with the right hand while maintaining tension on the rail limb with the left hand (Fig. 13.1p). Engage both limbs of the suture with the suture cutter (Fig. 13.1q), advance the cutter below the skin until there is resistance, and then cut the sutures.

StarClose SE™ (Abbott Vascular, Abbott Park, IL): Nitinol Clip

Indications

- The vascular access site should be in the common femoral artery.
- The common femoral artery should be at least 5 mm in diameter.
- 5–6 F arteriotomy can be closed.

Contraindications

- Common femoral artery luminal diameter less than 5 mm in diameter.
- Significant peripheral vascular disease.
- Significant luminal encroachment.
- More than moderate fluoroscopically visible calcification.

Deployment Steps

- Create a 5–7 mm skin incision at the sheath site to accommodate the insertion of the clip delivery tube into the tissue tract.
- Insert the guidewire and exchange the procedural introducer sheath for the StarClose SE exchange sheath (Fig. 13.2a).
- With the heel of the left hand on the patient for support, hold the bottom of the sheath hub and insert the distal tip of the flex-guide into the hub using the right hand (Fig. 13.2b).

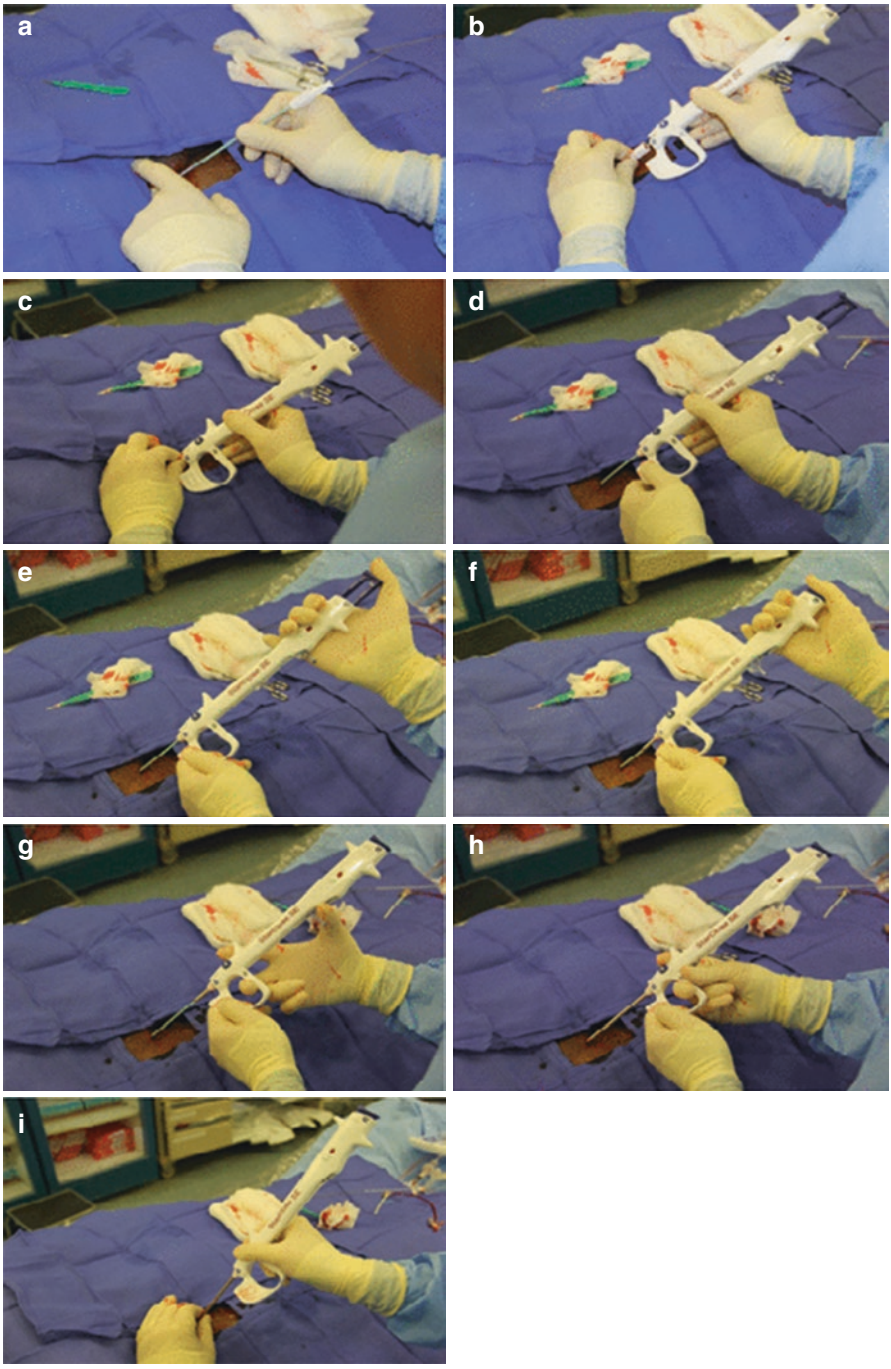

Fig. 13.2 Steps of StarClose closure device deployment

- Slowly advance the flex-guide down through the exchange sheath. If there is resistance, the exchange sheath may be kinked.
- Connect the clip applier to the sheath hub by pushing the device onto the sheath until it CLICKS into place (Fig. 13.2c).
- With the heel of the left hand on the patient for support, grasp the stabilizer to secure the device at the angle of the tissue tract. Pull back the device 2–3 cm (Fig. 13.2d).
- While continuing to stabilize the device with the left hand as described above, assume a 'syringe grip' on the proximal end of the device with the right hand by placing the index finger and the middle finger on the proximal posts and the thumb on the plunger (Fig. 13.2e).
- Firmly depress the plunger with the right thumb until it CLICKS into place. The number '2' appears in the number window at this point. This step will deploy the locator wings inside the blood vessel and initiate splitting of the exchange sheath (Fig. 13.2f).
- Check to make sure the number '2' is completely visible in the number window.
- While continuing to stabilize the device with the left hand as described above, switch the position of the right hand to a 'pistol grip', and gently retract the device with the right hand until slight resistance is felt as the locator wing makes contact with the vessel wall (Fig. 13.2g).
- Fully depress the thumb advancer using the pad of the right thumb until it CLICKS into place. The number '3' appears in the number window at this point (Fig. 13.2h). The clip is resting on the surface of the vessel and is ready to be deployed.
- Raise the body of the device to a 60–75° angle.
- While continuing to stabilize the device with the left hand as described above, switch the position of the right hand to a 'palm grip.' The thumb should rest on the deployment button. Gently push the device down on top of the artery with the right hand to seat the clip delivery tube on top of the access site (Fig. 13.2i) and deploy the device by depressing the deployment button with the right thumb. A final audible click should be heard with device deployment.
- Maintain the downward pressure for 2–3 s. Place the left hand on the puncture site in the palm-down position with the clip delivery tube extending up between the index and middle finger.
- Provide counter-traction with the left hand on the patient's body, and remove the device with the right hand.

Angio-Seal™ (St. Jude Medical): Collagen Plug Device

Indications

- The vascular access site should be in the common femoral artery.
- The common femoral artery should be at least 5 mm in diameter.
- 5–6 F arteriotomy can be closed with 6 F Angio-Seal.
- 7–8 F arteriotomy can be closed with 8 F Angio-Seal.

Contraindications

- Common femoral artery luminal diameter less than 5 mm in diameter.
- Significant peripheral vascular disease.
- Significant luminal encroachment.
- More than mild fluoroscopically visible calcification.

Deployment Steps

- Insert the Angio-Seal guidewire into the procedure sheath.
- Remove the procedure sheath, leaving the guidewire in place to maintain vascular access (Fig. 13.3a).
- Insert the arteriotomy locator into the insertion sheath. If done correctly, the reference indicators (arrows) on the two pieces should match up, and there should subtle click as the two components lock together (Fig. 13.3b). (This step should be performed prior to removal of the procedure sheath.)
- Thread the arteriotomy locator/insertion sheath assembly over the guidewire into the puncture tract. The reference indicators (arrows) on the arteriotomy locator and insertion sheath assembly should be facing up (Fig. 13.3c).
- Advance the assembly until blood begins to flow from the drip hole located near the distal end of the locator (Fig. 13.3d). The blood flow should be brisk, and pulsatile. Once satisfactory backflow of blood is confirmed, pull the assembly back until blood flow stops. Then push the assembly forward again (typically around 1 cm) until brisk blood flow is achieved again. This step ensures that the assembly is not placed too deeply into the artery.
- With the heel of the left hand on the patient for support, hold the insertion sheath with the thumb and forefinger and remove the arteriotomy locator and guidewire (Fig. 13.3e).
- Grasp the TIP of the Angio-Seal device and insert the tip into the delivery sheath. At this point, there should be some backflow of blood from around the tip of the device (Fig. 13.3f). (If there is NO flow, then the insertion sheath may have been pulled out of the artery. (Abort the procedure and apply manual pressure for hemostasis.)
- Continue to advance the Angio-Seal device until completely inserted into the insertion sheath and snapped together. There should be an audible click. At this point, the reference indicators on the Angio-Seal device and the insertion sheath should match up and should face up (Fig. 13.3g).
- Pull back on the handle of the Angio-Seal device until there is an audible click. The handle of the Angio-Seal device may have to be rocked left and right to ensure that the device is locked in the back position (Fig. 13.3h).
- Pull back on the device handle at the angle of the puncture tract, maintaining a steady uninterrupted motion until there is resistance (Fig. 13.3i). As soon as the delivery suture and a green tamper on the suture becomes visible, advance the

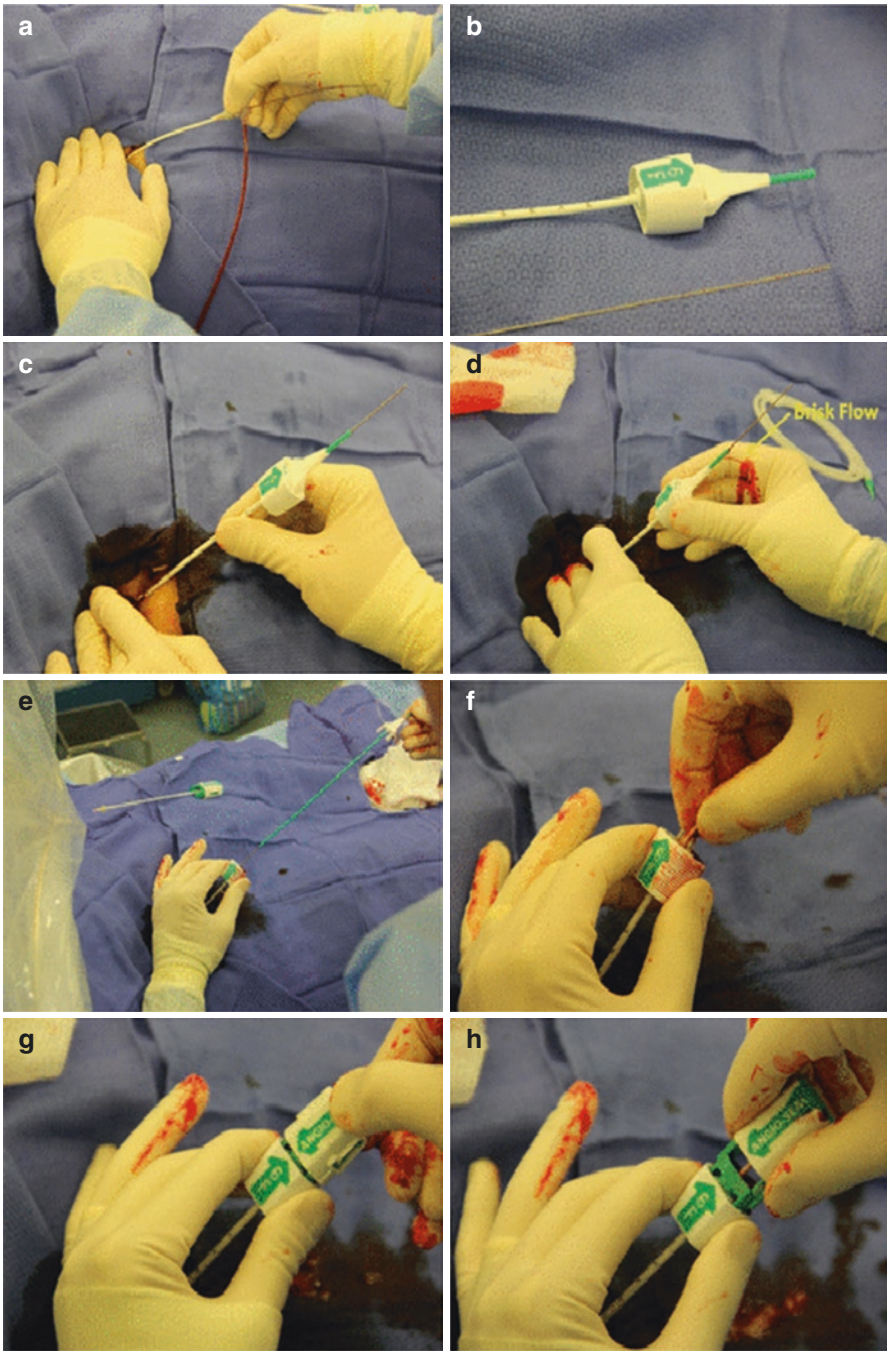

Fig. 13.3 Steps of Angio-Seal closure device deployment

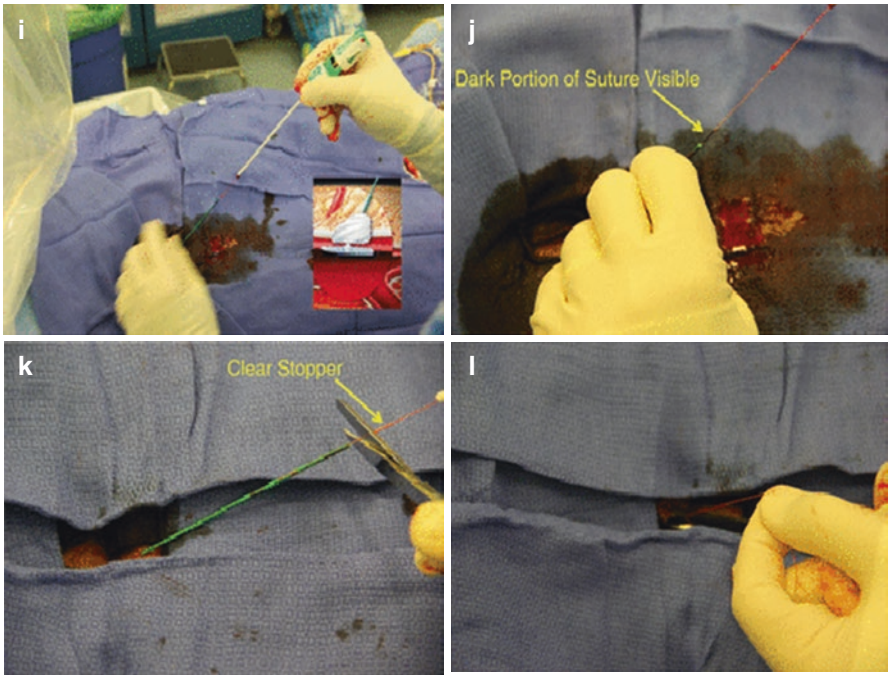

Fig. 13.3 (continued)

tamper while maintaining tension on the suture until resistance against the vessel wall is felt and in most cases, a black compaction marker is revealed (Fig. 13.3j).

- Cut the delivery thread below the clear stopper (Fig. 13.3k). Then push down on the skin using a blunt-tip sterile scissor and cut the suture below the skin level (Fig. 13.3l).
- Apply gentle external pressure over the site for 1 min to prevent tract ooze.

MANTA™ Vascular Closure Device (Teleflex): Collagen-based

Indications

- The vascular access site should be in the common femoral artery.
- Designed specifically for large bore femoral arterial access site closure.
- 10–14 F sheaths can be closed with 14 F Manta.
- 15–20 F sheaths can be closed with 18 F Manta.

Contraindications

- Common femoral artery luminal diameter less than 5 mm in diameter for the 14 F or < 6 mm in diameter for the 18 F MANTA.
- Femoral artery puncture in the same vessel within the prior 30 days, and/or recent (<30 days) vascular closure device placement in the same femoral artery.
- Marked tortuosity of the femoral or iliac artery.
- Allergies to bovine products, collagen and/or collagen products, polyglycolic or polylactic acid polymers, stainless steel, or nickel.
- Significant peripheral vascular disease.
- Significant luminal encroachment.
- More than mild fluoroscopically visible calcification.

Deployment Steps

- Determine the depth of the access site before the large bore procedure.
- Insert depth locator over 0.035″ wire then retract it till flow stops > Deployment depth = puncture depth + 1 cm (Fig. 13.4a).
- Exchange the sheath with Manta sheath after completion of the procedure.
- Insert the Manta sheath and remove the introducer (keep the wire in) (Fig. 13.4b).
- Advance manta closure device over the guidewire, till the click is heard (Fig. 13.4c).
- Retract device and sheath (as a whole system) at 45 degrees till you get to the previously determined deployment depth.
- Rotate deployment lever to release anchor (Fig. 13.4d).
- Withdraw the device till you feel tension and the indicator shows yellow-green (Fig. 13.4e).
- Then advance the blue lock advancement tube and retract the device. You will hear a click and the indicator turns green (this seals the arteriotomy site) (Fig. 13.4f).
- Bring back the lock advancement tube to confirm hemostasis then remove the guidewire (Fig. 13.4g).
- Cut the sutures below the skin level (Fig. 13.4h).

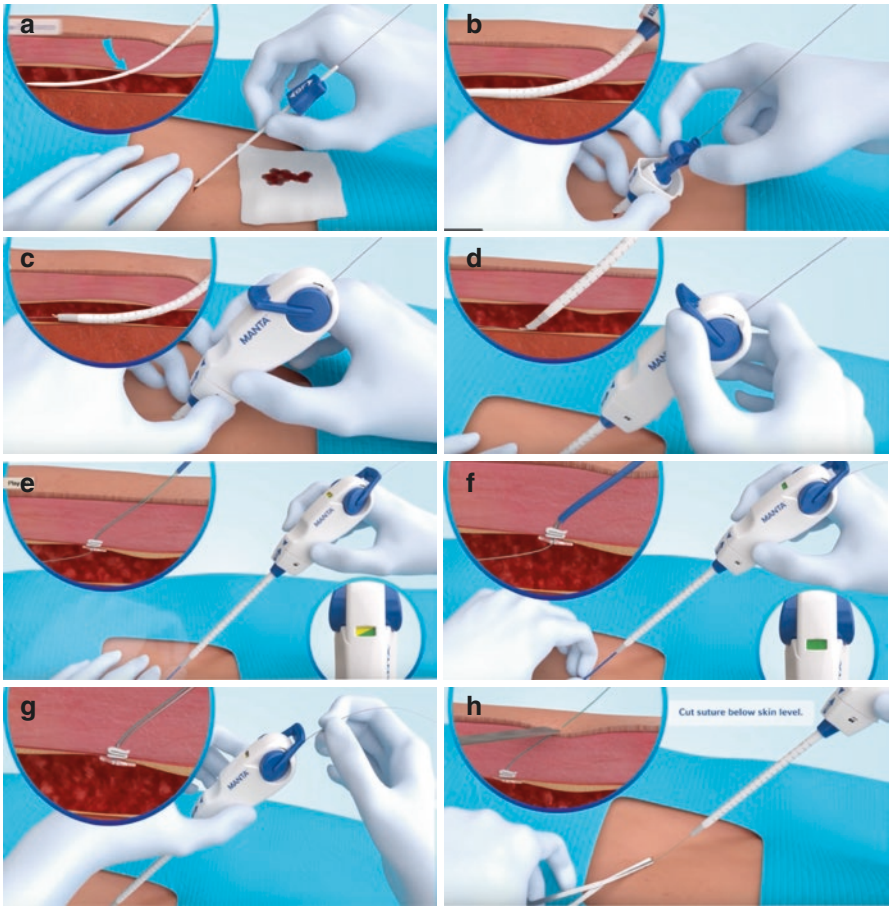

Fig. 13.4 Steps of Manta closure device deployment

VASCADE® (CARDIVA Medical Inc.): Resorbable Extra-Vascular Collagen Patch

- VASCADE® MVP Venous Vascular Closure System (6 F to 12 F venous sheaths).
- VASCADE® 5 F and 6/7 F Vascular Closure System (5 F to 7 F venous or arterial sheaths).

Indications

- Common femoral arterial or venous punctures.

Contraindications

- Significant peripheral vascular disease.
- Prior deployment of same access site within 3 months.
- Be aware and avoid any intravascular deployment.

Deployment Steps

- Dip the device tip in saline then insert it through the procedural sheath until the lock is midway at the hub (lock is silver color in 5 F and white color in the rest) (Fig. 13.5a).
- Then deploy Vascade disc by pulling the black actuator until you feel a detent and green segment is exposed (Fig. 13.5b).

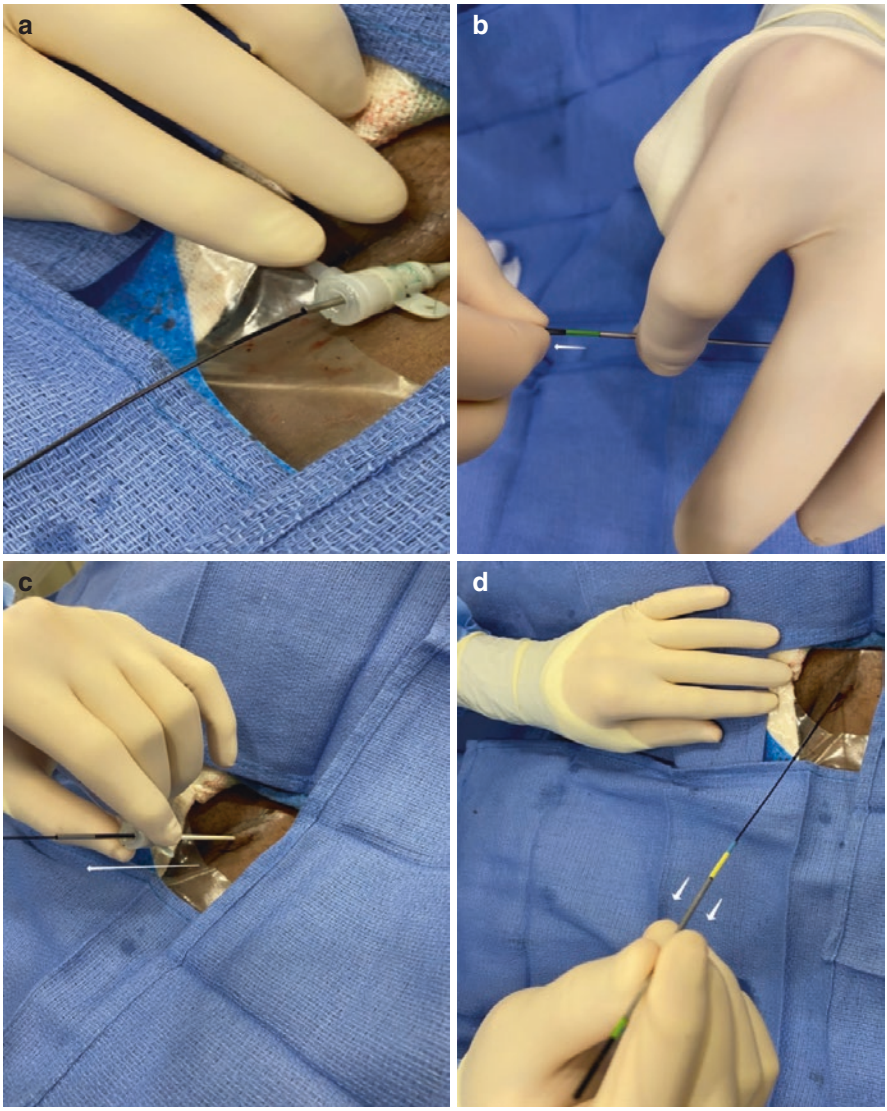

Fig. 13.5 Steps of Vascade closure device deployment

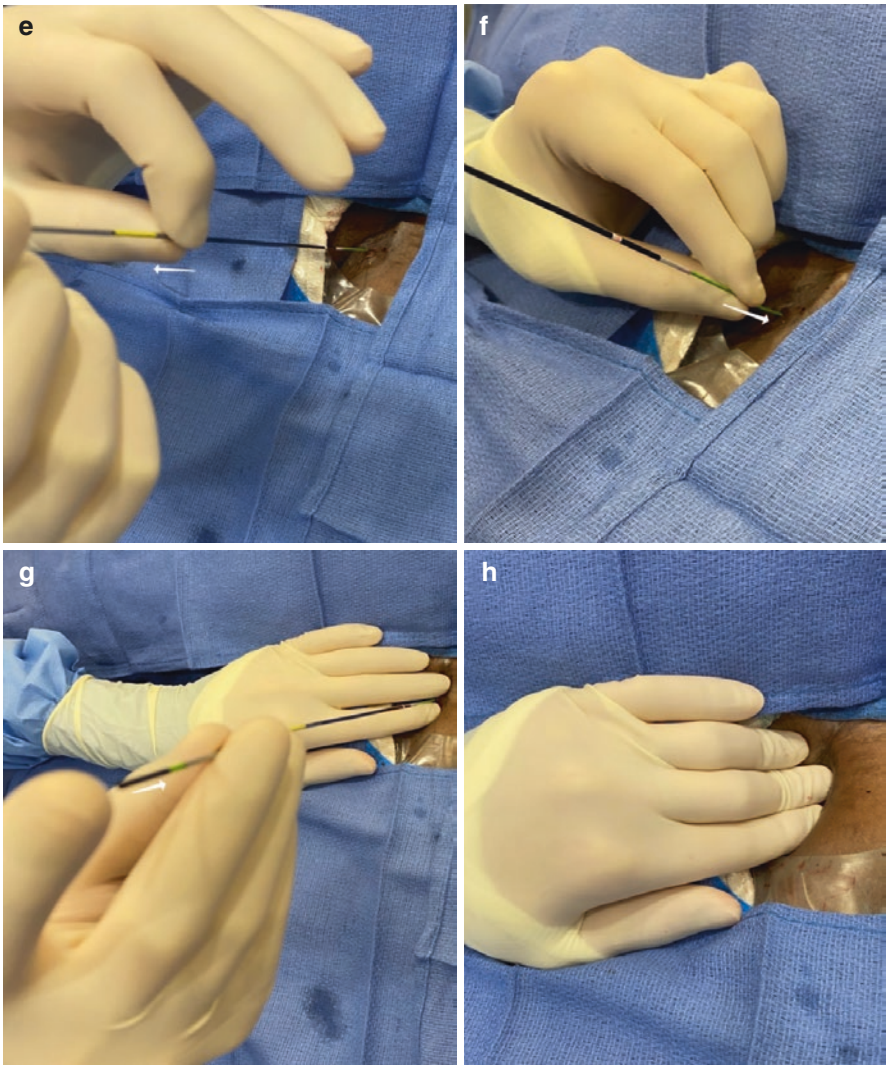

Fig. 13.5 (continued)

- Remove the introducer sheath and position the Vascade disc against the intimal aspect of the arteriotomy or venotomy, providing both temporary hemostasis and protection from intravascular placement of the Collagen Patch (The VASCADE Clip can be applied at skin level to maintain the position of the disc.) (Figs. 13.5c and 13.5d).
- After confirming the position of the collagen patch fluoroscopically, slide yellow-blue part into the lock that makes the black sleeve unlocked. Then retract the black sleeve to expose the collagen patch to the tissue tract. The system is left

in place for a brief period to allow the patch to swell (15 s for artery and 30 s for vein) (Fig. 13.5e).
- Advance the green push rod 2–3 times while maintaining back tension on the device (Fig. 13.5f).
- Relax tension and press down the black actuator to collapse the disc (Fig. 13.5g).
- Then while maintaining proximal manual compression, remove the device, and continue to apply pressure for 3–5 min (Fig. 13.5h).

MynxGrip™ (AccessClosure, Santa Clara, CA): Polyethylene Glycol Plug Device

Indications

- Common femoral artery punctures.
- Superficial femoral artery punctures.
- Mild peripheral vascular disease.
- 5–7 F arteriotomy can be closed.

Contraindications

- Significant peripheral vascular disease.
- Severe calcification proximal to or at the arterial puncture site.

Deployment Steps

- Insert MynxGrip into the procedural sheath up to the white shaft marker (Fig. 13.6a).
- Inflate the anchor balloon with about 2 cc of saline with supplied locking syringe until the black marker is fully visible on the inflation indicator and close the device stopcock so that the balloon stays inflated in the vessel (Fig. 13.6b). Leave the syringe on the device stopcock.
- Grasp handle and withdraw catheter until the balloon abuts the distal tip of the procedural sheath. The operator will feel resistance at this point.
- Continue to withdraw the balloon catheter until the balloon abuts the arteriotomy site (Figure Inset). The operator should feel a second point of resistance (Fig. 13.6c). Do NOT continue to pull back. Open the procedural sheath stopcock—there should be no backflow of blood.
- At this point, slight relaxation of tension will cause backflow of blood from the procedural sheath stopcock. Reapplication of tension will stop the backflow (Fig. 13.6d). This maneuver confirms that the balloon is in the correct position and is abutting the anterior surface of the vessel at the arteriotomy site.

- While lightly holding the device handle to maintain adequate tension on the balloon catheter, detach the shuttle and advance it until resistance is felt (Fig. 13.6e).
- Grasp the procedural sheath and withdraw it from the tissue tract. Continue retracting until the shuttle locks on the handle (Fig. 13.6f).
- Immediately grasp the advancer tube at the skin and gently advance until the green marker is visible. This step must be performed immediately because the PEG plug must be delivered to the vessel surface before it starts to expand (Fig. 13.6g).
- Lay the device down hold this position for 30 s (Fig. 13.6h). This delay will ensure full expansion of the PEG on the vessel surface.
- Lock the syringe on the device stopcock to max negative, and apply light fingertip compression proximal to the insertion site. Open stopcock to deflate the balloon. To ensure complete balloon deflation, wait until air bubbles and fluid have stopped moving through the inflation tubing (Fig. 13.6i).

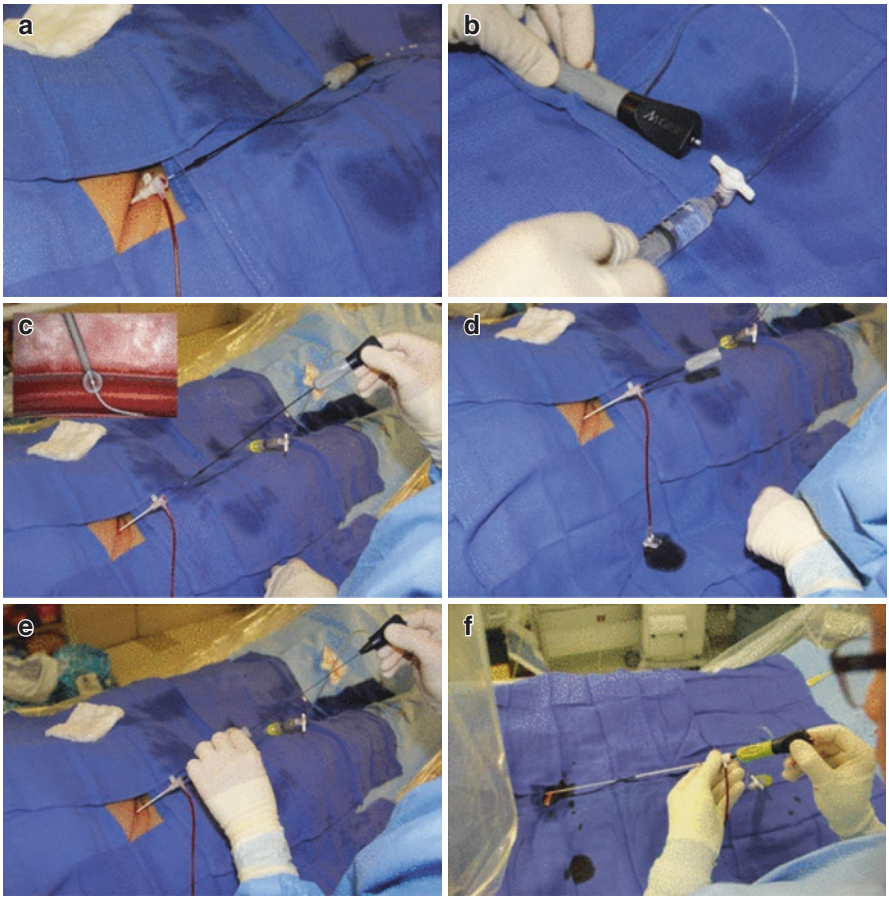

Fig. 13.6 Steps of Mynx closure device deployment

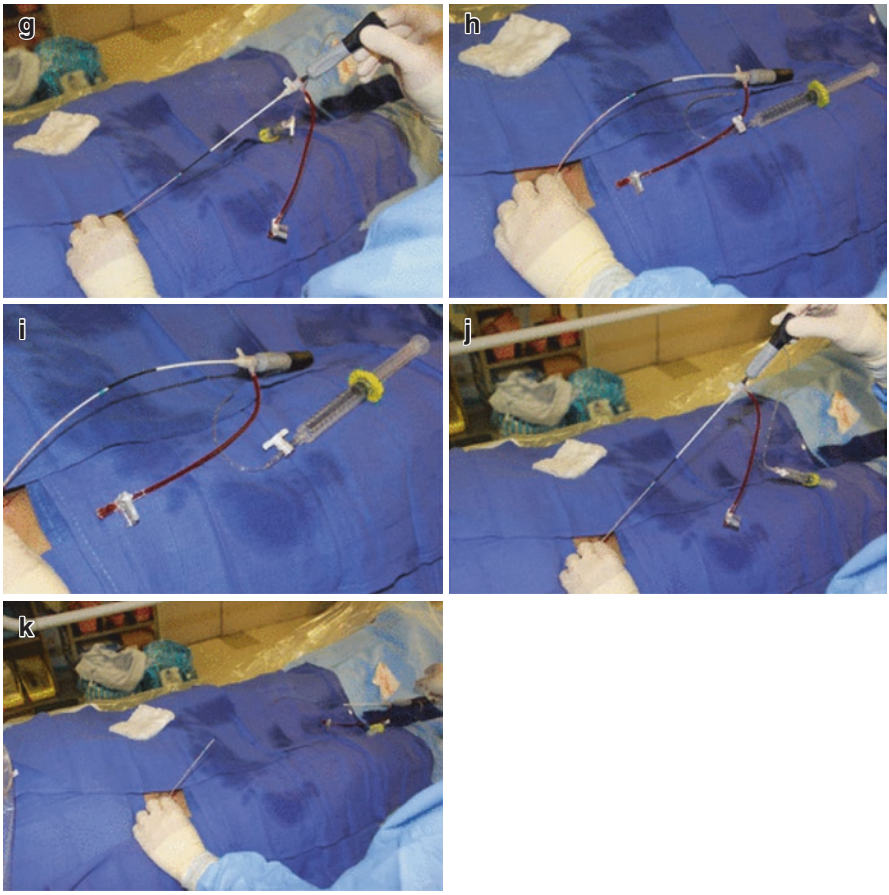

Fig. 13.6 (continued)

- Lightly grasp the advancer tube at the skin with thumb and forefinger and realign with the tissue tract (Fig. 13.6j).
- Slowly withdraw balloon catheter through the advancer tube lumen while holding the advancer tube (Fig. 13.6k).
- Remove advancer tube from the tissue tract. Fingertip compression can be applied for up to 2 min as needed.

Transradial Band™ (Terumo Interventional Systems, Somerset, NJ): Non-occlusive Radial External Hemostasis Device

Indications

- Radial artery punctures.
- 5–7 F arteriotomy can be closed.

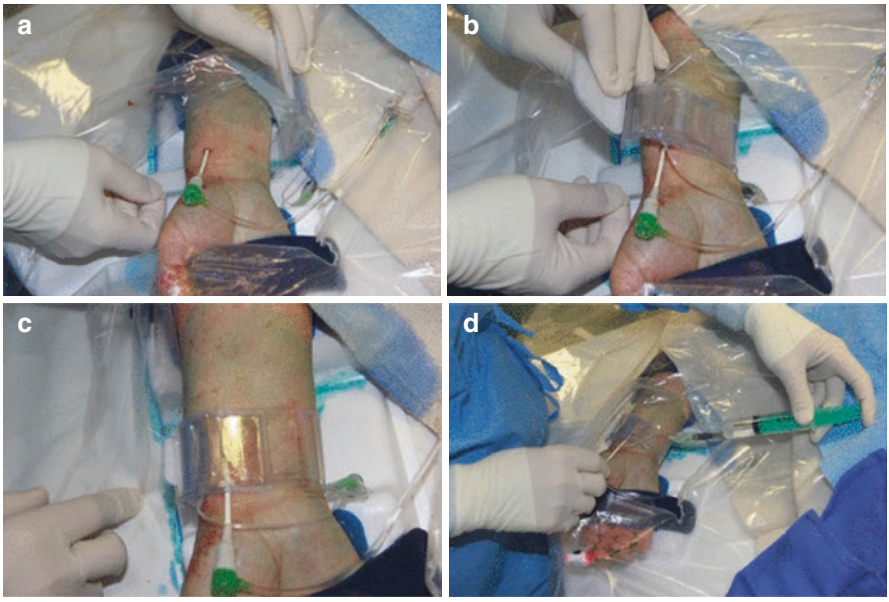

Fig. 13.7 Steps of TR band deployment

Deployment Steps

- Choose appropriate TR Band size (regular or large).
- Pull the access sheath midway back through the arteriotomy site (Fig. 13.7a).
- Wrap TR Band around the wrist and secure in place via the Velcro band (Fig. 13.7b).
- For proper positioning, the green marker on the translucent band should be placed proximal to the percutaneous access site (Fig. 13.7c).
- Instill 15 cc of air (Fig. 13.7d). Completely remove the sheath then remove air 1 cc at a time until bleeding occurs at which time 1–2 cc of air is reinjected into the balloon. The idea is to achieve patent hemostasis without occluding the radial artery, which can be assessed by the reverse Barbeau's test.
- Save the syringe for future deflation.
- Approximately 1 h after the sheath is pulled (2 h for PCI) air removal can begin.
- Withdraw 1 cc of air at a time utilizing the provided syringe and observe for any bleeding.
- If bleeding occurs, reinject air that was removed until hemostasis is achieved, and reattempt deflation in 30 min.
- If no bleeding is observed, the operator should remove all air, deflate the balloon completely, and observe for bleeding. If there is no bleeding, the operator can remove the TR Band and place a protective covering (e.g., Tegaderm) over the radial percutaneous site.
- Vital signs and pulse check should be performed every 15 min for the first hour while TR Band is in place, every 30 min during the second hour, and on an

hourly basis thereafter until post-sheath removal. Assess for presence of feeling in the fingers and capillary refill in distal fingers and nail beds until TR Band is removed.

FemoStop™ Mechanical External Compression Device (St. Jude Medical, St. Paul, MN)

Indications

- Secondary hemostatic aid for femoral artery access site after delivery of a vascular closure device.
- Secondary hemostatic aid for femoral artery access site after manual compression.
- Hemostatic aid for femoral artery access site oozing or minor bleeding.

Contraindications

- Significant peripheral vascular disease.
- Poorly controlled hypertension.
- Uncooperative patient.
- This device should NOT be used as a primary hemostatic device; e.g., this device should not be placed as the only hemostatic measure after pulling a sheath.

Deployment Steps

- Place the band on the bed prior to transitioning the patient over on top of it (Fig. 13.8a).
- Place the dome of the FemoStop to cover the arteriotomy site (Fig. 13.8b).
- Make sure FemoStop is positioned in a manner to apply direct vertical pressure down over the arteriotomy site (Fig. 13.8c).
- Increase pressure in the dome to approximately 20 mmHg above patient's systolic blood pressure and maintain for 3 min (Fig. 13.8d).
- Next, reduce pressure to mean arterial pressure over the next 5 min, and document distal pulses have not become compromised by this pressure level (Fig. 13.8e).
- Gradually reduce dome pressure over the next 1–1.5 h until the device can be safely removed.
- We recommend this device should not be placed overnight. If a FemoStop is applied for more than 4 h, it should be released for 30 min every 4–5 h to avoid DVT. Prolonged use of this device has been associated with arterial leg ischemia as well as formation of deep venous thrombus.

Fig. 13.8 (**a**) Place the band on the bed. (**b**) Place the dome of the FemoStop over the arteriotomy site. (**c**) Pull on the side until the device is taut. (**d**) Inflate to 20 mm Hg above patients SBP. (**e**) Reduce the pressure to MAP and ensure distal pulses are felt

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Part II

Coronary Intervention

Basics of Intervention

14

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Introduction

Preprocedural evaluation (focused history, physical examination, stress test, and risk grading, evaluating the appropriateness for coronary intervention) and adequate postprocedural care are critical for procedural and clinical success.

Preprocedural Components

History

- Current symptoms, presentation, and angina classification.
- Any symptoms of heart failure.
- History of any cardiac procedures—in patients with known pre-existing coronary artery disease (CAD)), complete and comprehensive review of all prior catheterizations, percutaneous interventions, and cardiac surgeries is vital. A Review of prior angiograms to identify the location of prior bypass graft origins and the type of catheters/devices used is important.
- Peripheral vascular disease (PVD)—information regarding prior peripheral vascular interventions and surgeries is also vital in planning access.
- Chronic kidney disease (CKD))—identify patients at risk for contrast-induced nephropathy.
- Smoking, alcohol and substance abuse history, and the possibility of withdrawal.
- History of obstructive sleep apnea and lung disorders, in regard to the effects of sedation.

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- Ability to tolerate dual antiplatelet therapy and any planned future surgeries/procedures within the next year to decide on the type of stent that can be inserted.
- Any history of prior allergic reactions.

Focused Physical Examination

- Height, weight, body mass index.
- Blood pressure (BP), heart rate (HR), and pulse oximetry.
- Signs of HF (rales, JVD, edema).
- Focused neurological exam.
- Peripheral bilateral lower extremity pulses must be documented.
- Allen and Barbeau's test for radial access.

Prior Studies

- Old labs and EKG if available as a baseline for future comparisons.
- Prior PCI, CT surgery reports, or CABG reports.
- Prior echocardiogram if done.
- Stress test and risk grading of stress test.
- Prior CT coronary angiography if done.

Informed Consent

- Informed consent, risks, benefits, and alternatives of the procedure are explained in non-technical language to the patient.
- A documentation of the following should be in the patient's chart prior to the procedure. The risks, benefits, and alternatives of cardiac catheterizations/interventions including but not limited to neurovascular trauma, MI, CABG, or death were explained to the patient and/or designated power of attorney [1].
- They entirely understand the procedure and have signed the informed consent (Table 14.1).

Contraindications

- There are no absolute contradictions to cardiac catheterization/PCI in acute STEMI/cardiogenic shock other than patient refusal or inadequate equipment or cardiac catheterization facility. As most procedures are elective, the patient can be rescheduled if they have the following:
  - Acute flares of any systemic disorders.
  - Active infection/septic shock.

Table 14.1 Major complications of PCI [1]

Complications from NCDR CathPCI Registry	NCDR CathPCI Registry	Mount Sinai Heart
2010 through July 2012	2010–July 2012 (n = 787,980)	2010–2012
	n = 787,980	n = 14,584
Any adverse event	1.53%	1.0%
Cardiogenic shock	0.47%	0.08%
Pericardial tamponade	0.07%	0.08%
CVA/stroke	0.17%	0.05%
New requirement for dialysis	0.19%	0.01%
Emergent CABG performed during admission	0.17%	0.02%
Any vascular complication requiring treatment	0.44%	0.09%

  - Acute gastrointestinal bleeding or severe anemia which has not been worked up.
  - Electrolyte imbalance.
  - Pregnancy.
  - Recent cerebrovascular accident (<1 month).
  - Rising creatinine but patient not on dialysis.
  - Acute exacerbation of congestive heart failure.
  - INR > 3.5 for radial or INR > 2.5 for femoral.
  - Alcohol intoxication or acute substance abuse.

Preprocedural Preparation

- Patient and family teaching (procedure, results, complications).
- Labs:
  - Type and cross match.
  - Complete blood cell and platelet counts.
  - Prothrombin time (PT), partial thromboplastin time (PTT).
  - Electrolytes, blood urea nitrogen (BUN), creatinine.
- 12-lead ECG.
- One or two peripheral IV lines.
- Skin-shave and prepare both inguinal areas and the wrist for radial artery.
- Dietary status, NPO for at least 4 h:
  - Patients scheduled for morning to noon procedures should be NPO since midnight except for scheduled medications.
  - Patients scheduled for afternoon procedures should have a full breakfast and then be made NPO except for scheduled medications.
  - Patients scheduled for evening procedures should have a full breakfast and lunch and then placed NPO except for scheduled medications.
- Allergic reactions:

  - Aspirin allergy and aspirin desensitization: Allergic reactions to aspirin include aspirin-exacerbated respiratory disease and angioedema. As dual antiplatelet therapy is required in all patients receiving stents for at least 1 month up to 1 year or longer, aspirin desensitization in aspirin-allergic patients should be performed. This can be completed in a day in conjunction with an allergist/immunologist in CCU/general cardiology ward or over a few days to a week as an outpatient.
  - Contrast allergy: Patients with a history of significant contrast allergy (such as angioedema, shock, anaphylaxis, and bronchospasm) should get Prednisone 50 mg PO 13 hours before, 7 hours before, and 1 hour before the procedure. IV Solu-Cortef 100 mg and IV Benadryl 50 mg should be administered in the cath lab prior to the procedure.

    Patients with mild contrast allergy (skin rash, itching, hives) or allergy to shellfish should receive IV Solu-Cortef 100 mg and IV Benadryl 50 mg in the cath lab prior to the procedure.

    Urgent IV premedication for medically urgent patients: IV Solu-Medrol 200 mg 5 hours and 1 hour prior to the procedure and IV Benadryl 50 mg 1 hour prior to the procedure.
- Medications on procedure day:
  - Aspirin 162 mg orally.
  - Continue patient's regular antihypertensive medications, statins, and immunosuppressive medications.
  - Hold oral hypoglycemic medications.
  - Usual dose of basal insulin or half to one-third dose of NPH insulin can be given on the morning of the procedure. Hold short-acting insulin.
- Hydration (Tables 14.2 and 14.3) [2, 3].

Table 14.2 Preprocedural hydration

	Start hydration with normal saline (0.9NS) 3 h prior to procedure if outpatient and 12 h prior if inpatient
LVEF %	For all patients but especially if eGFR <60
>50	1–2 mL/kg/h
31–50	0.5 mL/kg/h
<30	0.3 mL/kg/h for 3 h only for all patients

Table 14.3 Intraprocedural and postprocedural hydration

	Continue hydration with normal saline (0.9 NS) during procedure and for 6–8 h post-procedure if outpatient and 12 h if inpatient
LVEF %	For all patients but especially if eGFR <60. If eGFR >60, intravenous hydration can be given for a shorter duration with instructions for liberal discharge oral hydration
>50	1–2 mL/kg/h
<50	Measure LVEDP in the catheterization lab. Give a bolus of NS based on LVEDP
	<12: 500 ml bolus 12–18: 250 ml bolus
	>18: No bolus
31–50	Subsequent infusion of 0.5 mL/kg/h
<30	Subsequent infusion of 0.3 mL/kg/h

Periprocedural Components

Vitals

- BP, HR, and pulse oximetry are documented at the start of the procedure, every 5 min. Continuously during the procedure, and every 15 min post-procedure for the first 2 h and hourly thereafter.

Medications

- Conscious sedation.
  - Versed (midazolam): start with 0.5 mg IV in the elderly and patients with systemic diseases and 1–2 mg in other patients and then as needed to achieve sedation.
  - Morphine sulfate for analgesia: 2–4 mg IV to start and then as needed.
  - Meperidine for patients with morphine allergy: 50–150 mg given 30–90 min before the beginning of anesthesia either subcutaneously or intramuscularly.
  - Acetaminophen: 1 g IV to reduce the need for narcotic analgesia.
  - Metoclopramide: 5 mg IV to prevent opiate-induced nausea.
- Antidotes for conscious sedation:
  - Flumazenil: 0.2 mg over 15 s and repeat doses at 1 min intervals if required to a maximum of 1 mg. In the event of re-sedation, 0.2 mg at 20 min intervals as needed to a maximum of 3 mg/h.
  - Naloxone: 0.4–2 mg and repeat doses at 2 min intervals if required to a maximum of 10 mg. If no response after 10 mg total, consider other causes of respiratory depression.
- Anticoagulation for diagnostic procedures.
  - Heparin: 1500–2000 U in patients with prior CABG prior to the engagement of grafts or in patients with severe aortic stenosis prior to crossing the valve.
  - Heparin 50 U/kg in radial access.
- If heparin needs to be reversed use:
  - 1 mg of protamine for every 100 U of heparin (maximum dose is 50 mg).
  - 0.5 mg protamine/100 U of heparin if heparin was discontinued 30 min to 1 h prior to the procedure.
  - 0.25 mg protamine/100 U of heparin if heparin was stopped over 2 h prior to the procedure.
- Anticoagulation and antithrombotic regimens during PCI:
  - Bivalirudin: bolus (0.75 mg/kg) and continuous infusion (1.75 mg/kg/h). If creatinine clearance is <30 ml/min, bolus dose remains the same, but infusion dose is reduced to 1 mg/kg/h or 0.25 mg/kg/h if on dialysis. If ACT < 250 3 min after bolus, administer additional 1/2 bolus of bivalirudin or administer additional 1/3 bolus if ACT is 251–299.
- If PCI is planned, load the patient with:
  - Clopidogrel – 600 mg if clopidogrel naïve (<5 days on clopidogrel) or 300 mg if on daily maintenance of clopidogrel.

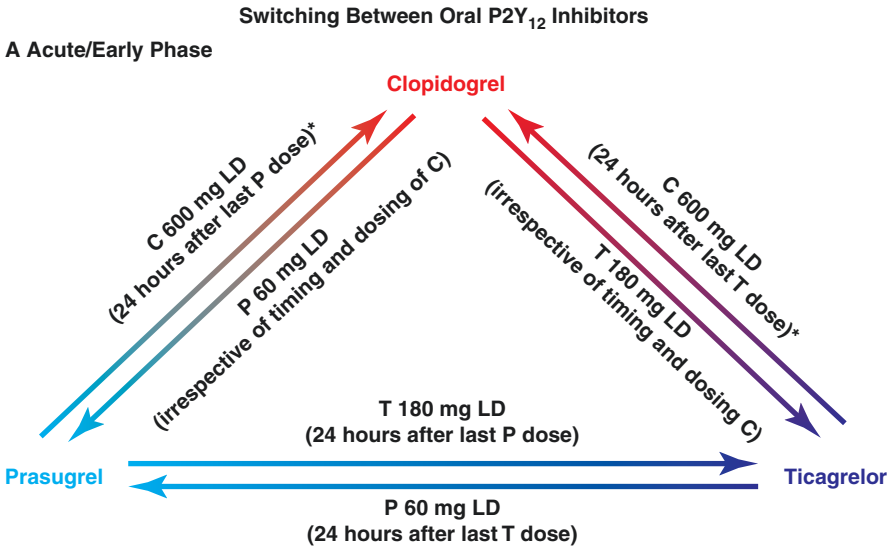

Fig. 14.1 Switching between oral P2Y12 inhibitors. (Adapted from Angiolillo et al. [1])

  - Prasugrel—60 mg if prasugrel naïve or 30 mg if on daily maintenance of prasugrel or clopidogrel. Contraindications are active pathological bleeding, prior TIA or stroke, hypersensitivity to prasugrel, and age > 75.
  - Ticagrelor—180 mg if ticagrelor naïve or 90 mg if on daily maintenance of ticagrelor. Contraindications are hypersensitivity to ticagrelor, active pathological bleeding, history of intracranial hemorrhage, and severe hepatic impairment. If switching from Clopidogrel to Ticagrelor give 180 mg of Ticagrelor (Fig. 14.1).
  - Cangrelor is approved as an adjunct to percutaneous coronary intervention (PCI) to reduce the risk of periprocedural myocardial infarction (MI), repeat coronary revascularization, and stent thrombosis in patients who have not been treated with a P2Y12 platelet inhibitor and are not being given a glycoprotein IIb/IIIa inhibitor. Dose: 30 mcg/kg iv bolus infused over 1 minute before PCI, then immediately follow bolus injection with 4 mcg/kg/min iv infusion, continue for at least 2 hours or duration of PCI, whichever is longer.
- IC nitroglycerin for more accurate stenosis severity visualization: 100–200 mcg boluses. If hypotension occurs, administer IV fluid boluses if no volume overload or congestive heart failure is present. Avoid nitroglycerin in patients with severe AS. It is contraindicated in right ventricular infarction or after recent use of sildenafil/vardenafil (<24 hrs) or tadalafil (<48 hrs).
  - Mix: 25 mg in 250 D5W solution = 100 mcg/ml.
  - Use proper medication bottle/tubing.

- IC verapamil: for SVG graft stenosis/slow flow, 250–500 mcg boluses. Monitor for bradycardia.
  - Mix: 1 ml = 2.5 mg, dilute in 10 mL NS syringe = 250 mcg/ml.
- IC nitroprusside for slow flow: up to 50–100 mcg or as needed.
  - Mix: 12.5 mg in 250 mL D5W solution = 50 mcg/mL.
  - Cover the medication bottle with a dark plastic bag to retain medication potency.
- IC Neosynephrine: 100–200 mcg boluses. Give 100 mcg IV (1 mL) initially followed by NS flush. Monitor for bradycardia. Higher dosage may cause nausea and may require intravenous metoclopramide or ondansetron.
  - Mix: 1 mg in 10 mL NS solution; 100 mcg = 1 mL.

Postprocedural Management and Care

Bed Rest

- Coronary angiogram (no AC) with successful VCD deployment: 2 h.
- Coronary angiogram (no AC) with manual hold hemostasis: (sheath size-2) hours.
- PCI with successful VCD deployment: 2 h post discontinuation of anticoagulation.
- PCI with manual hold hemostasis: (sheath size-1) hours after sheath removal.

Fluids

- Continue as per Tables 14.2 and 14.3.

Labs

- CBC, BMP, and troponin I at 3–6 h in all patients and subsequently if there are any lab abnormalities.
- CBC, BMP, and troponin I at 12–18 h if monitored overnight and subsequently if there are any laboratory abnormalities.

Imaging

- EKG: 3–6 h after the procedure and subsequently as per symptoms.
- Echo: after NSTEMI/STEMI to evaluate LV function if a ventriculogram is not performed.

Disposition After the Procedure

- Diagnostic procedure—same-day discharge:
  - 2 h discharge if closure device was used successfully.
  - 4 h discharge if manual compression hemostasis was achieved (5–6 Fr sheaths).
- Simple PCI—same-day discharge:
  - 4–6 h if closure device was used successfully.
  - 6–8 h if manual compression hemostasis was achieved.
- Inpatient admission—any patient who needs inpatient monitoring:
  - Presentation as STEMI or non-STEMI or hemodynamic instability (cardiogenic shock, CHF, significant bradyarrhythmia or tachyarrhythmia).
  - Major vascular complications.
  - Age > 85 years.
  - Significant comorbid conditions:
    Ventricular arrhythmia or rapid atrial arrhythmia.
    Decompensated systolic heart failure or LVEF <30%.
    Chronic renal insufficiency (SCr ≥ 1.8 mg/dl).
    Prior organ transplant or immunosuppressive state.
- Complex or high-risk coronary intervention such as:
  - Unprotected LM or LM equivalent.
  - Vein or arterial graft intervention.
  - Bifurcation lesion requiring both branch interventions.
  - Use of atherectomy devices.
  - Use of thrombectomy devices.
  - CTO requiring bilateral injection.
  - Prolonged procedure with fluoro time > 60 min.
- Procedural complications such as:
  - No flow/slow flow.
  - Side branch closure.
  - Perforation.
  - Residual dissection (Type C or more).
  - Thromboembolism.
  - Postprocedural chest pain or EKG changes.
  - Hemodynamic instability requiring treatment.
  - Elevated cardiac enzymes: CK-MB >3 × baseline.
- Inability to ambulate after the procedure due to:
  - Poor coordination.
  - Vasomotor instability.
  - Dizziness or suspected neurological issues/events.
- High-risk patients requiring therapeutic long-term anticoagulation.
- No social support or inability to get access to medical care in case of an emergency.

Post-PTCA Pharmacology

- Hold metformin for 48 h post-procedure.
- Dual antiplatelet therapy for at least 4 weeks after stenting with a bare-metal stent and 12 months with a drug-eluting stent.
- Start high dose statin therapy.
- Evaluate the need for cardiac rehabilitation.
- Smoking cessation counseling in indicated patients.
- Dietary counseling and risk factor/lifestyle modification.
- Anti-anginal therapy and maximal medical management as indicated by current guidelines.
- Cardiac rehabilitation for post-ACS patients.

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Difficult Stent Delivery

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Introduction

Stent delivery remains challenging in complex coronary anatomy in around 5% of PCIs, despite colossal technical advances in the field of interventional cardiology. Unsuccessful stent placement is associated with inferior short and long-term outcomes. Failure of stent deployment may lead to stent embolization during withdrawal maneuvers. It is imperative to identify these lesions and plan stent delivery meticulously.

Characteristics Associated with Stent Delivery Failure

The following characteristics are associated with stent delivery failure:

- Vessel tortuosity—iliac, femoral, and coronary
- Lesion severity
- Lesion length
- Coronary calcifications (before and at the lesion)
- Stent length
- Poor guiding catheter support
- Previously deployed stents

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Steps to Help Prevent Difficulties in Stent Delivery

The following approaches/techniques are used to help prevent difficulty with stent delivery:

- Long sheath
- Appropriate guide selection
- Lesion preparation/plaque modification
  - Low-profile balloon
  - High-pressure NC balloon
  - Cutting balloon
  - Atherectomy devices

Long Sheath

- Anticipate the need for a long sheath. Rotations and manipulations at the proximal end may not transmit to the distal end of devices and guide in excessively tortuous iliac and femoral arteries. A long sheath, preferably 45 cm will improve guide support. Initially place the sheath tip in the descending aorta and advance further if extra backup is required.

Appropriate Guide Selection

- Coaxial engagement is the key for stable guide position and it is imperative to choose the correct guide to provide passive support. (Fig. 15.1) Active support is provided by deep seating the guide (see Chap. 7 Guiding Catheter Selection). A larger guide catheter diameter provides better support.

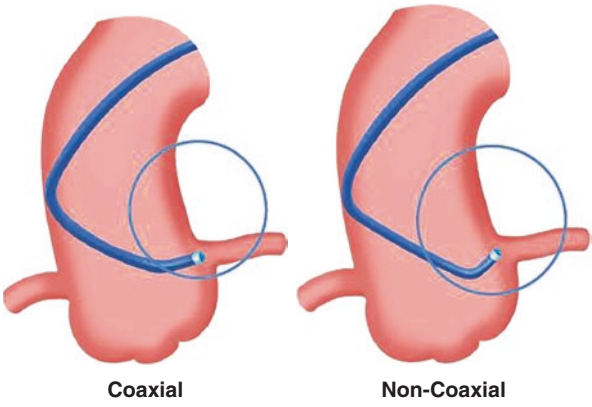

Fig. 15.1 Coaxial guide engagement

Table 15.1 Comparison between Guidezilla and Guideliner V3

Features	Guidezilla II	Guideliner V3
Proximal shaft	Stainless steel hypotube	Stainless steel ribbon
Coating	Silicon wipe	Z-glide
Collar	Platinum iridium	All polymer
Collar transition	17 cm Half-pipe	6 mm Hypotube transition
Marker band	Distal marker band Radiopaque collar	Distal marker Proximal marker
Size (ID in mm)	6 F(1.45), 7 F(1.60), 8 F(1.83)	6 F(1.42), 7 F(1.57), 8 F(1.80)

Adequate Lesion Preparation

- This is the most important step. Adequate predilation with high-pressure balloons or cutting balloons must be performed prior to stent insertion. If you have trouble passing even a small (1.2/8 mm) compliant balloon or a microcatheter, then consider plaque modification techniques like rotational or orbital atherectomy/Excimer laser coronary atherectomy (ELCA)/Intravascular Lithotripsy.

Stepwise Approach to Facilitate Stent Delivery

The following approaches/techniques are used to facilitate stent delivery:

- Deep seating the guide
- Deep inspiration
- Buddy wire or changing the wire
- Guide support extension catheters
- Change stent length
- Buddy Balloon
- Buddy-in-jail technique
- Miscellaneous
- PTCA alone

Deep Seating Guide [1, 2]

- Deep intubation of the guide into the coronary artery improves backup support and pushability of devices regardless of the anatomy of the coronary vessel and the morphology of the lesions. Use this technique with caution in diffusely diseased or small proximal vessels, as it is prone to cause dissection. The guide should be gently maneuvered into the coronaries over the shaft of a PTCA balloon or stent delivery catheter to minimize the endothelial trauma, which may occasionally result in ostial stenosis.

Deep Inspiration [3, 4]

- The easiest maneuver to try is deep inspiration. Deep inspiration displaces the diaphragm and the heart into a vertical position and straightens the coronary tree slightly, which facilitates balloon and stent delivery. It may not be feasible in a deeply sedated patient.

Changing Wire

- Changing wires remains one of the simplest and less expensive strategies for difficult stent delivery. A long stent loaded on a floppy wire may not have enough support for its passage, in which case changing to a firmer wire might be useful. However, in certain cases stents loaded on extra support wire may push against the wall of a heavily calcified and angulated lesion, making the stent passage difficult. Changing to a less firm wire might be helpful in this situation. A microcatheter is useful here for changing wires across a difficult-to-cross lesion (see Chap. 8).
- Runthrough™, BMW™, and Luge Prowater™ are examples of firm, nonhydrophilic wires.
- Fielder™, Whisper™, and Choice PT™ are examples of floppy, hydrophilic wires.
- Mailman™ and Grandslam™ are examples of extra support wires.
- Hi-Torque Wiggle Wire™ (Fig. 15.2) is a specialty wire with its unique feature of 60 mm of sinusoidal shaping, which starts 60 mm from the distal tip, which alleviates the friction by altering the angle of contact between the balloon and the body of the stent [5].

Buddy Wire

- A second wire, *buddy wire technique*, may help to straighten the vessel and improve the guiding catheter coaxiality. A variant of the buddy wire technique is the *gliding wire technique*, where a second hydrophilic wire is inserted to allow the stent to glide over the hydrophilic coating of the second wire. Another variant is the *anchor wire technique*, where a second wire is inserted in a non-target

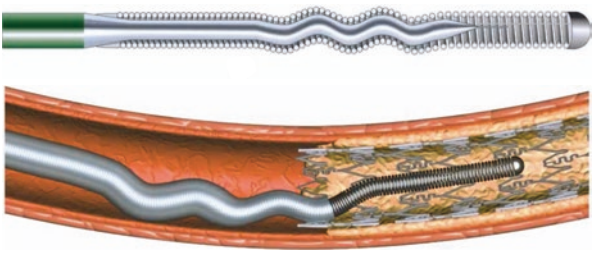

Fig. 15.2 Hi-Torque Wiggle Wire™

vessel of the left system or a branch of the vessel for the right coronary system. The extra guide wire will anchor and stabilize the guide catheter.

Guide Support Extension Catheters

- Guideliner V3™
  - As a "mother and child" system (catheter inside a catheter), the Guideliner V3™ is a 25 cm guide extension connected to a pushrod with a 17 cm "half-pipe" collar, which helps in deep seating for added backup guiding catheter support in challenging cases to facilitate device delivery (Fig. 15.3) [6]. It also allows coaxial alignment when a difficult coronary ostium takeoff prevents guiding catheter placement. Sometimes, the Guideliner is advanced deep into the coronary artery with the help of an anchor balloon to facilitate stent delivery, especially long stents (balloon anchoring technique). Guideliner Balloon Assisted Tracking (GBAT) can be utilized when the anchoring technique fails. It involves tracking of the Guideliner over a nominally inflated noncompliant balloon partially protruding from its tip.
  - It is contraindicated in vessels with <2.5 mm diameter. It should be considered either to increase backup support or enable stent delivery when problems are encountered using conventional techniques or upfront in the setting of very complex disease.
- Guidezilla™ II
  - Support extension catheter provides additional backup support and facilitates easy delivery of ancillary devices. Balloon-assisted tracking *(also known as the Inch-worming technique)* involves advancing the Guidezilla II over a recently deflated balloon for further advancement down the artery. Never advance the Guidezilla II Catheter more than 15 cm beyond the tip of the

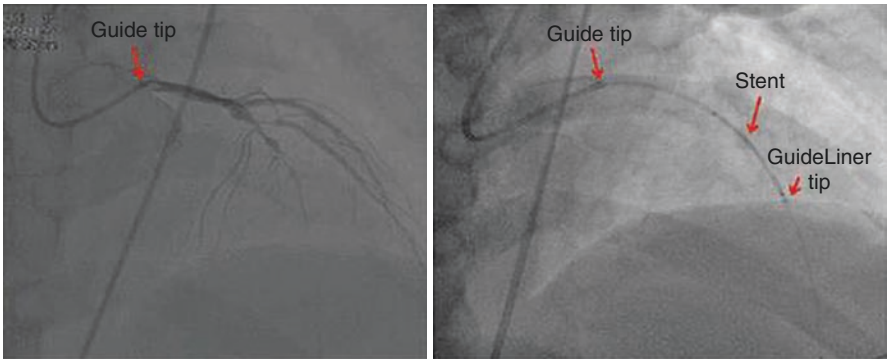

Fig. 15.3 Guideliner: deep seating for stent delivery

guide catheter. Complications include pressure dampening, balloon kinking, distal marker tip dislodgement, coronary stent damage/stripping, coronary artery ischemia or dissection, and/or occlusion may occur [7].

Change Stent Length

- A less attractive and expensive option is changing to one or two shorter stents or a different stent type with better flexibility.

Buddy Balloon [8]

- Non-inflated/Inflated: *Anchor balloon technique* (Fig. 15.4). It was initiated in PCI of chronic total occlusions and is achieved by inflating a balloon in a non-target vessel to obtain enough support to cross a lesion. There is a risk of

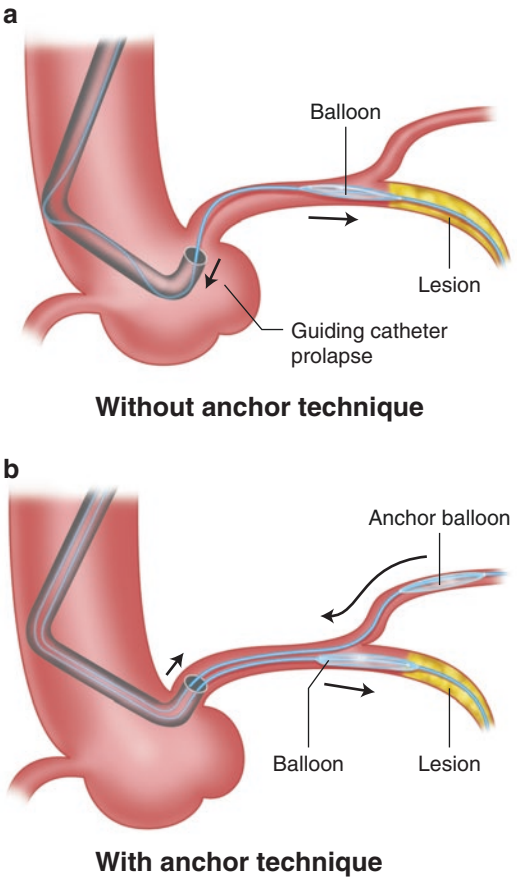

Fig. 15.4 Anchor balloon technique for stent delivery

injury to the ostium of the coronary artery by the guiding catheter and the non-target vessel by an anchor balloon. It may not be feasible in all cases due to poor support by non-axial traction and failure of an appropriate non-target vessel.

Buddy-in-Jail Wire Technique [9]

- The buddy-in-jail technique is rarely used because of the availability of guide support extension catheters (Fig. 15.5). It is useful when all the standard techniques fail to deliver the stent to a distal stenosis and there is additional proximal stenosis that the interventionalist intends to stent. A second non-hydrophilic coated buddy wire of at least medium support is wired to the distal vessel. The proximal vessel is then stented and the buddy wire is jailed. The jailed in buddy wire provides the support to deliver the distal stent through the proximally placed stent by the following mechanisms:
- The guiding catheter will be anchored more securely within the ostium of the vessel.
- The jailed buddy wire may straighten the proximal portion of the vessel and minimize the wire bias.
- The jailed wire will provide a stiff rail for stent tracking.
  - A non-coated buddy wire should be used and the radiopaque portion of the wire should not be jailed. The proximal stent should be deployed using only modest pressure (12 atm or less) to minimize the risk of wire entrapment or fracture. Before deployment of the distal stent, the jailed buddy wire should be removed to avoid "double jailing." The proximal stent must be postdilated at the end.

Miscellaneous

- Rotaglide facilitated stent delivery—Application of Rotaglide solution over the stent surface reduces the catheter friction, thereby facilitating stent delivery in challenging coronary anatomy [10].
- Partial Balloon Inflation technique—Constant and steady pressure applied at the Tuohy-Borst hemostatic valve while gently inflating the balloon at lower pressures (2–3) atm, helps in achieving the successful stent delivery to target lesion [11].

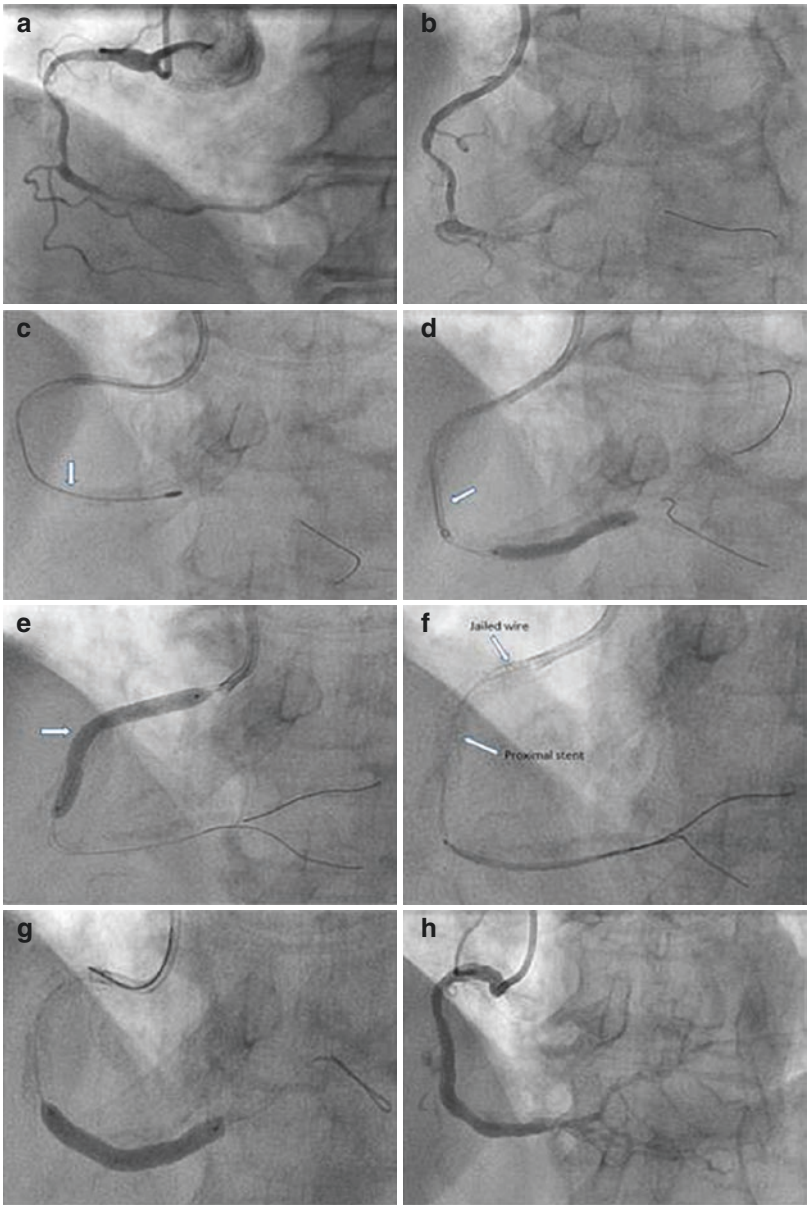

Fig. 15.5 Buddy-in-jail wire technique. (**a**) Selective RCA angiogram showing severe calcific stenosis of proximal and distal RCA. (**b**) Wire being exchanged with help of a microcatheter. (**c**) Rotablation performed using 1.25 mm burr (arrow). (**d**) Predilation performed after tracking the balloon distally with help of a Guidezilla guide support catheter. (**e**) Stent (arrow) being deployed in proximal RCA while jailing one of the wire. (**f**) Distal stent being tracked distally with the help of the jailed wire. (**g**) Deployment of distal stent after removing the previously jailed wire. (**h**) Final angiographic result

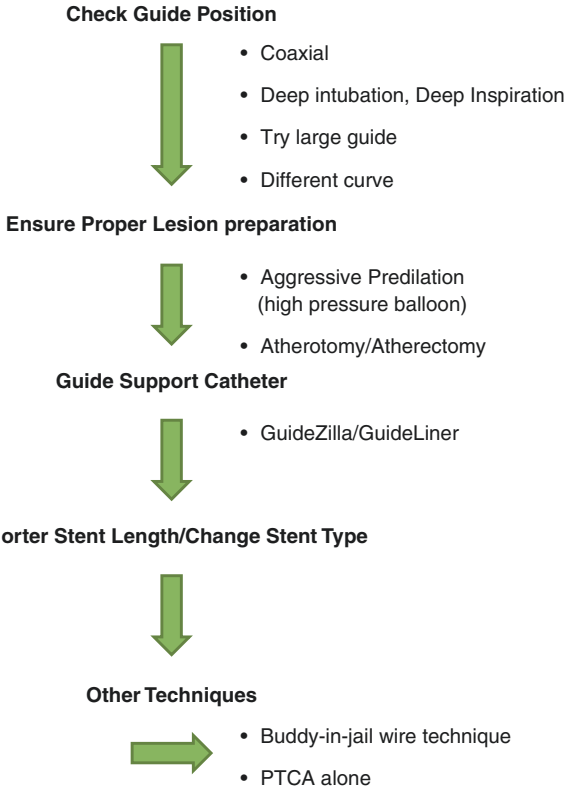

Fig. 15.6 Our institutional stepwise approach for difficult stent delivery

Settling for PTCA Without Stent

- Rarely, when all measure fails, provisional PTCA with optimal results confirmed by intravascular ultrasound or fractional flow reserve may be the best solution.
- See below, Fig. 15.6 for our Institutional stepwise approach for facilitating stent delivery.

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Bifurcation Lesions

16

Sunny Goel, Gurpreet S. Johal, and Annapoorna Kini

Introduction

Coronary bifurcations are involved in 15–20% of all percutaneous coronary interventions (PCI). It remains one of the most challenging lesions in interventional cardiology in terms of procedural success rate as well as long-term cardiac events. The optimal management of bifurcation lesions is a subject of considerable debate.

Definition

Bifurcation lesion is defined as a lesion with stenosis >50% involving a bifurcation with a significant side branch vessel (SBV). A significant SBV is a branch, whose loss is of consequence to a particular patient (symptoms, location of ischemia, viability of the supplied myocardium, collateralizing vessel, left ventricular function, etc.) [1, 2].

Medina Classification

Medina classification is the most commonly used classification which indicates the location of significant stenosis (>50%) in the bifurcation tree (Fig. 16.1). This classification divides lesions into seven categories using a three-component binary key based on visual assessment of lesion severity. Stenosis >50% is assigned 1 for each of three arterial segments of bifurcation in the following order, proximal main vessel (PMV), distal main vessel (DMV), and side SBV [1, 2].

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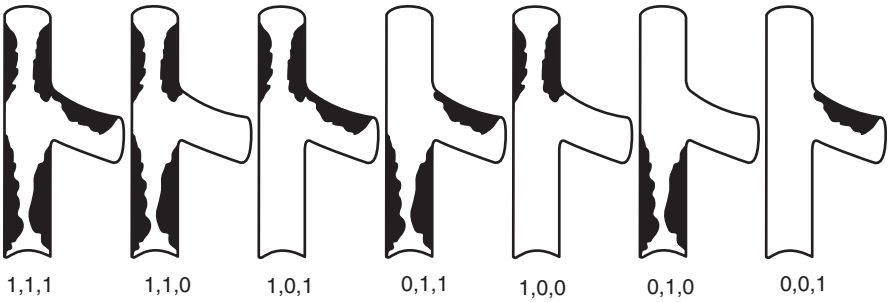


Fig. 16.1 Medina classification for bifurcation lesions. (Used with permission from Bifurcaid Application [3])

Table 16.1 Guide catheter selection

LCA bifurcation lesions	Provisional: 6 Fr VL/EBU guide
	Dedicated 2 stent: 7 Fr VL/EBU guide
RCA bifurcation lesions	Provisional: 6 Fr IM or AL 0.75
	Dedicated 2 stent: 7 Fr IM or AL 0.75

Approach to Bifurcation Intervention

Access

- Femoral or radial:
  - 6 Fr for provisional stenting approach or DK crush.
  - 7 Fr Guide is required for most of the dedicated 2 stent strategies, left main PCI and if SB > 2.25 mm.
  - If opting for femoral access, a 45 cm long sheath is preferred as it provides extra support.

Guiding Catheter Selection (Table 16.1)

Optimal Angiographic Views

- Distal left main: *LAO Caudal (30–60°, 25–30°) or AP caudal (0, 25°–40°).*
- LAD/diagonal bifurcation: *RAO cranial (10°, 40°) or AP cranial (0, 25°–40°).*
- Diagonal ostium visualization: *LAO cranial (40–45°, 25–30°).*
- For early diagonals: *LAO caudal (45–55°, 25–30°).*
- LCX/marginal: *AP caudal (0, 25°–40°), and LAO caudal (45–55°, 25–30°).*
- Distal RCA/ RPDA: *AP/LAO Cranial (0–55°, 30°).*

Coronary Wiring

- Polymer coated wires which are easy to recross and can be jailed (e.g. Fielder™, Whisper) are preferred for SBV wiring, and workhorse wires (e.g. Runthrough™) are preferred for MV wiring.

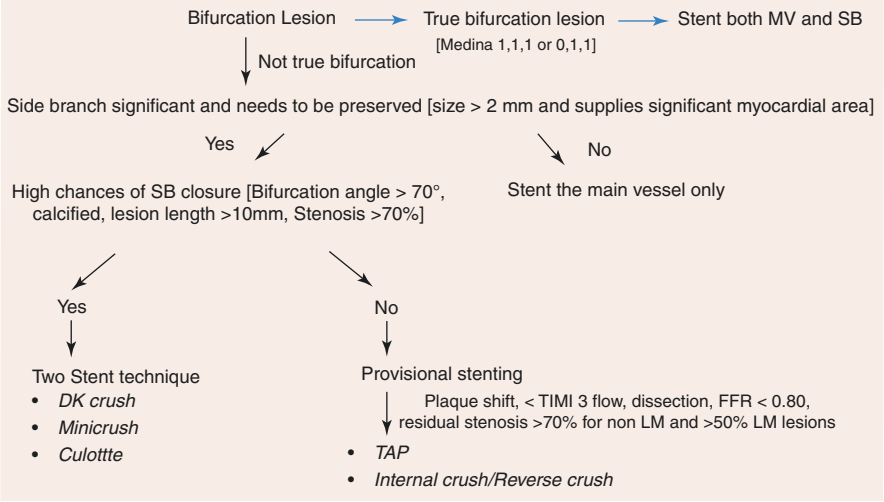


Fig. 16.2 Interventional algorithm for bifurcation coronary lesions

- The more complex lesion/branch should be wired first.
- When wiring the second vessel, avoid excessive torqueing and 360° turns of the wire. Use small side-to-side movements to prevent intertwining.
- While shaping the wire tip, try to best adapt the length and angulation of the tip curve to the given anatomy.

Lesion Preparation

- Adequate lesion preparation is essential for bifurcation lesions. Use of cutting balloons and atherectomy devices may be required for severely calcified lesions.

Bifurcation Algorithm (Fig. 16.2)

Provisional Stenting Approach

These are the common steps for provisional stenting:

- Wire only the MV and perform MV pre-dilation.
- Assess for plaque shift into SBV. If there is plaque shift and TIMI 3 flow is compromised, wire the SBV and perform PTCA (1:1 sizing) or Cutting balloon (CB) PTCA (1/4 size smaller than reference vessel) of the SBV.
- Reassess side branch. If TIMI 3 flow is restored, without dissection or residual stenosis proceed with provisional stenting of the MBV (nominal pressure) keeping the wire in the SBV.

- Remove the jailed SBV wire. If high-pressure balloon post-dilation of the MBV stent is required, rewire the SBV and perform post-dilation.
- Reassess the side branch again. If the patient is chest-pain-free, there is TIMI III flow and no dissection in SBV, take final angiogram; otherwise perform kissing balloon inflation (KBI)* or bailout stenting of SB using the TAP or inverted crush strategies (Fig. 16.3).

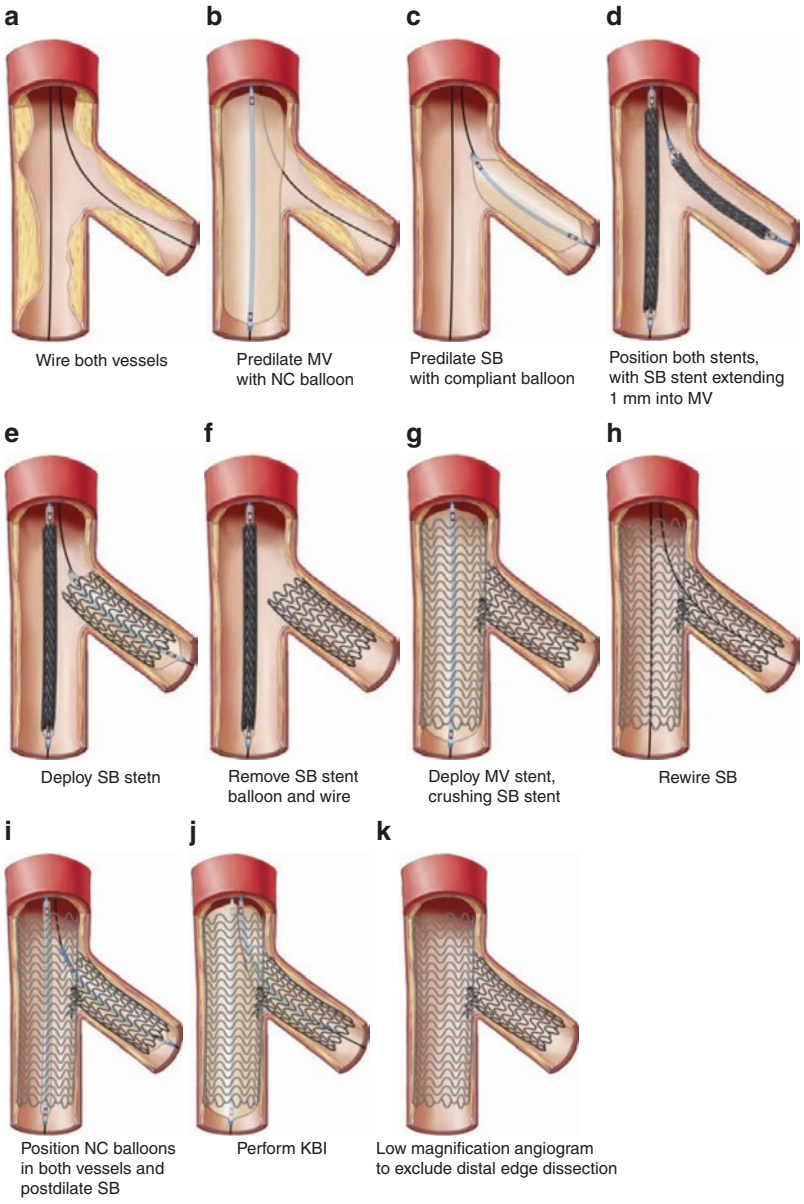


Fig. 16.3 T-stenting and protrusion (TAP)) beginning after main vessel stenting. (Used with permission from Bifurcaid Application)

- *KBI: Rewire the SBV through the strut of MV stent. Initially perform PTCA of jailed side branch using a smaller complaint balloon at high pressures (18–20 atm) to open the stent struts. Perform final KBI with NC balloons in the MBV and SBV (1:1 sizing). The proximal optimizing technique (POT) is recommended following KBI to ensure optimal stent expansion in the proximal MV.

Two-Stent Approach

These are the common steps for two-stent techniques:

- Wiring of MBV and SBV and lesion preparation using PTCA/CBA/Atherectomy.
- PTCA of more severe stenosis is preferably performed first.
- Stenting strategy will depend on the anatomical characteristics as described below.
- Mini crush (Fig. 16.4), DK crush (Fig. 16.5), and Culotte (Fig. 16.6) are the preferred two-stent techniques (Table 16.2).

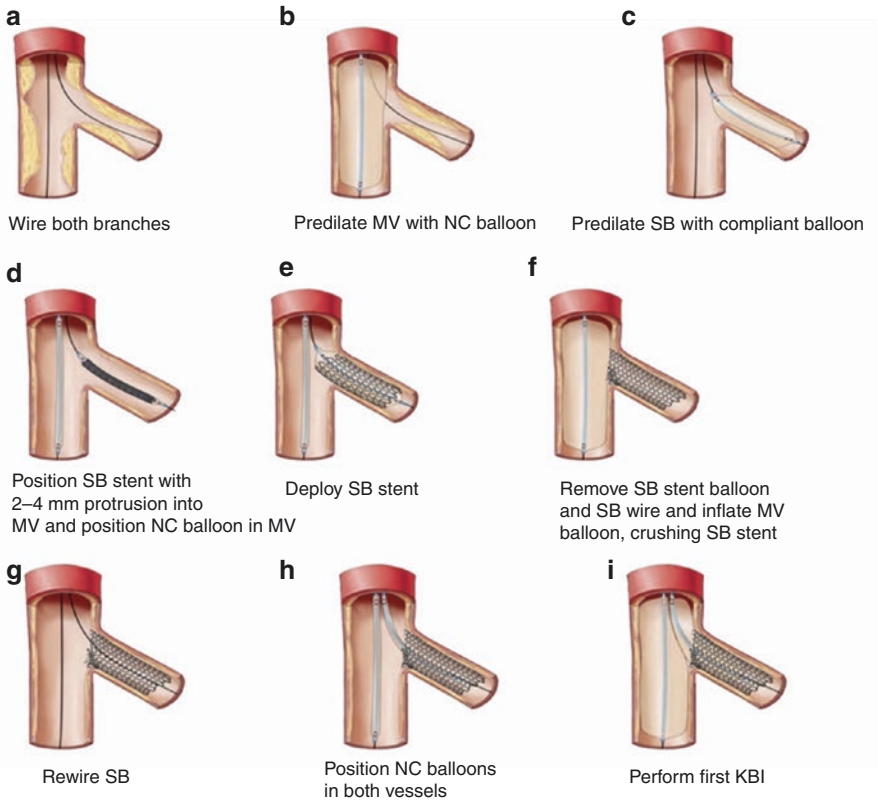


Fig. 16.4 Mini-crush stenting technique. (Used with permission from Bifurcaid Application)

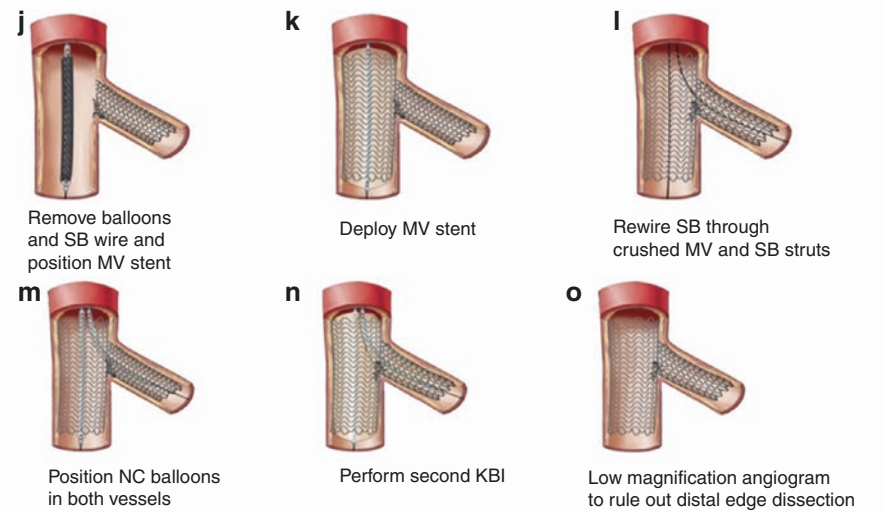


Fig. 16.4 (continued)

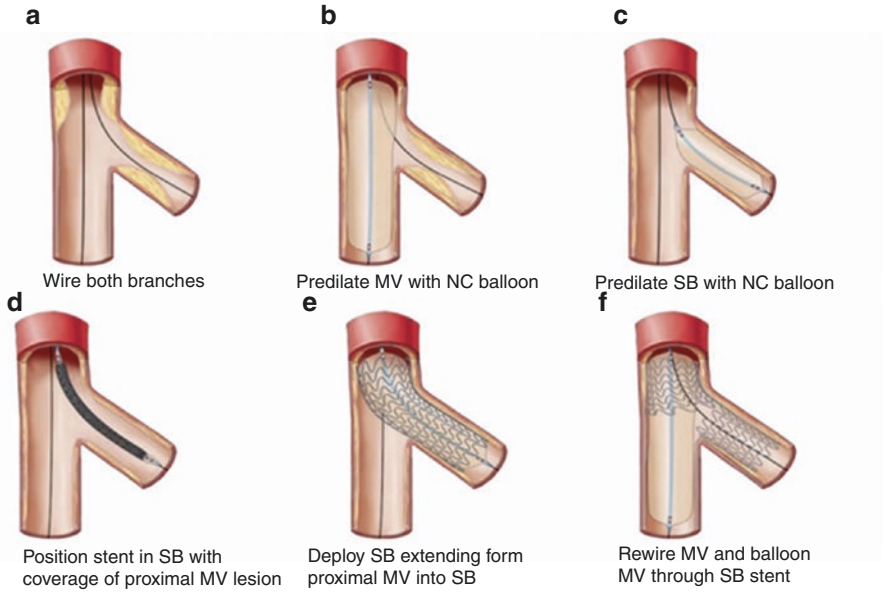
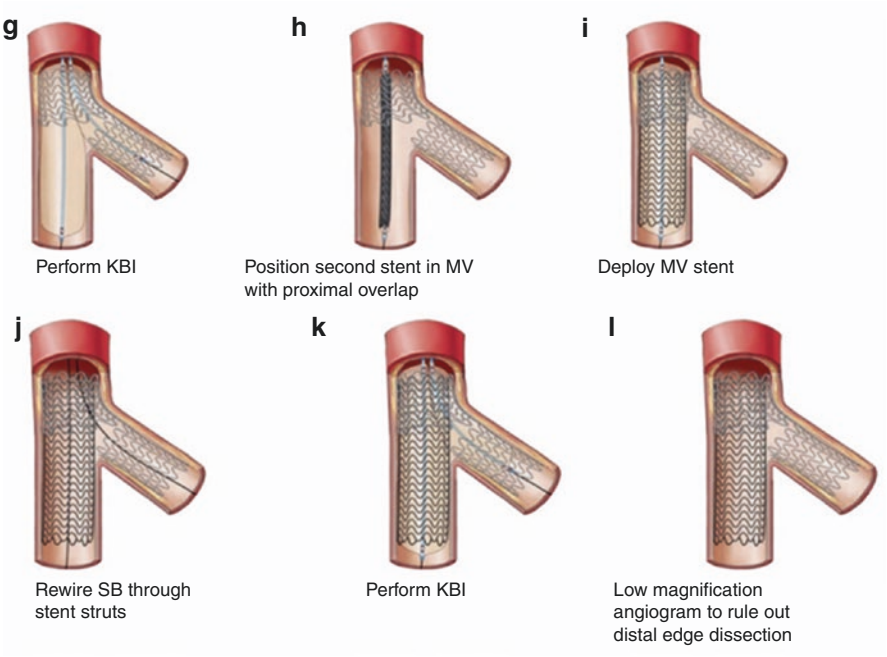


Fig. 16.5 Double kissing (DK) crush technique. (Used with permission from Bifurcaid Application)



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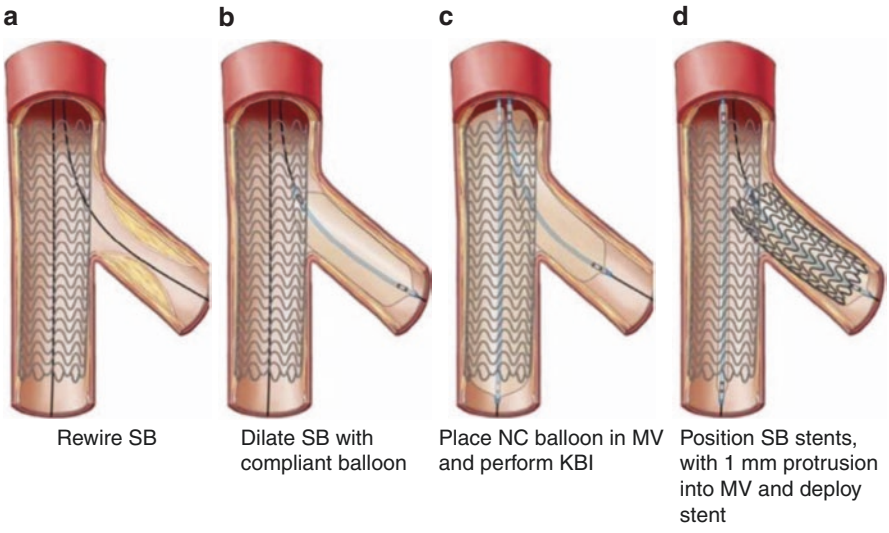

Fig. 16.6 Culotte technique. (Used with permission from Bifurcaid Application)

Fig. 16.6 (continued)

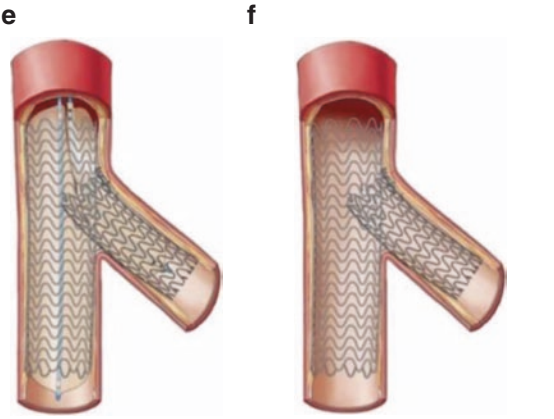

Table 16.2 Important studies comparing provisional versus dedicated two-stent strategy

Study	Year	Total number (provisional/2 stent strategy)	2-stent technique used	Clinical follow up (months)	Primary end-point (provisional vs 2-stent strategy)
Colombo et al. [4]	2004	85 (22/63)	T-stent, V stent, and Y stent	6	Angiographic restenosis of either branch (18.7% vs. 28%; p = NS)
Pan et al. [5]	2004	91 (47/44)	T-stent	11	Angiographic restenosis of either branch (7% vs. 25%; p = NS)
NORDIC [6]	2006	413 (207/206)	Crush, Culotte, other unspecified	6	MACE [cardiac death, MI, ST or TVR] (2.9% vs. 3.4%; p = NS)
BBK [7]	2008	202 (101/101)	T-stent	12	Angiographic restenosis of SB at 9 months (23% vs. 27.7%; p = NS)
CACTUS [8]	2009	350 (173/177)	Crush	6	MACE [Cardiac death, MI, or TVR] (15% vs. 15.8%; p = 0.95)
BBC ONE [9]	2010	500 (250/250)	Crush, Culotte	9	MACE [Cardiac death, MI, or TVF] (8% vs. 15.2%; p < 0.05)
Lin et al. [10]	2010	108 (54/54)	DK crush Culotte T-stenting	8	MACE [Cardiac death, MI, ST, or TVR] (21% vs. 6%; p < 0.01)
DK CRUSH II [11]	2011	370 (185/185)	DK crush	12	MACE [Cardiac death, MI, or TVR] (17.3% vs. 10.3%; p = 0.07)
NORDIC Baltic IV [12]	2013	450 (221/229)	Culotte, T stent, other unspecified	6	MACE [Cardiac death, MI, TLR], or ST (5.5% vs. 2.2%; p = 0.07)
Kim et al. [13]	2015	419 (206/213)	Crush	12	MACE [Death, MI or TVR] (18.5% vs 17.8%; p = NS)
EBC TWO [14]	2016	200 (103/97)	Culotte	12	MACE [Death, MI or TVR] (7.7% vs 10.3%; p = NS)
DK CRUSH V [15]	2019	484 (242/242)	DK crush	36	TLF (16.9% vs 8.3%, P = 0.0050

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Ostial Lesion Interventions

17

Radha Mehta and Annapoorna Kini

Introduction

Ostial lesions pose distinctive technical challenges and are associated with geographic miss rates of up to 54 percent and a three-fold increase in target lesion revascularization [1]. This chapter provides an overview of equipment and interventional techniques for ostial lesions.

Ostial Lesion

Definition

- Ostial lesions are defined as lesions >50% within 3 mm of the origin of the vessel. It could be at the aorto-ostial or branch-ostial junction.
- Always try to exclude coronary spasm by giving IC nitroglycerin and injection after disengaging the guide catheter.

Etiology

- Atherosclerosis: most common.
- Calcific atherosclerotic lesions of the aorta encroaching on the coronary ostia.
- Congenital coronary anomalies.

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Anatomic Challenges

- Inability to engage the guide and maintain position/dampening of pressures.
- High degree of elastic recoil.
- Different takeoff angles from aorta.
- Difficulty in precise placement.
- Respiratory and cardiac motions especially with ostial RCA lesions in transradial approach.
- Inability of tubular stent design to adequately cover the funnel-shaped ostium.

Equipment

- Sheath: 6 Fr.
- Guide selection:
  - Choose a less aggressive guide that will provide coaxial alignment without the tendency of deep engagement; this will also facilitate disengagement of the guide during stent placement.
  - Guide catheter with side holes may be preferred in cases of subtotal aorto-ostial lesion.
  - Guide selection will depend on the vessel takeoff and various other factors.
  - For RCA, IM or FR 4 guide with side holes should be used.
  - For LM, FL guides (due to their short tip) with side holes should be used.
  - For RCA bypass grafts, multipurpose or AR2 with side holes should be used.
  - For OM or diagonal grafts, AR2 or AL1 with side holes should be used.
- Position:
  - Guide should not be fully engaged or deep seated.
  - For severe ostial disease, pre-load the wire in the guide before vessel intubation. This will facilitate rapid wiring and catheter disengagement after wiring.
  - A buddy wire may also be used to provide additional stability or as a marker in the ascending aorta or side branch to assist in positioning the stent.
  - "Floating wire technique" can be used to identify the true ostium. One guidewire is placed in the target vessel and the second guidewire in the aorta. The second wire will help demarcate the true ostium and prevent the guide catheter from deeply intubating the vessel.
  - Once the device (balloon, stent, etc.) is positioned, the guide is gently withdrawn into the aorta. Prior to complete removal of the device from the artery, use the device to "rail in" the guide tip (prevents damage to the deployed stent).
- Wires:
  - Standard workhorse 0.014″ wire is appropriate for most cases.
- Balloons:
  - Cutting/scoring balloon—Flextome™ or AngioSculpt™.

Access

- Femoral access is preferred as it provides better guide stability.

Fluoroscopic Views

- LAO-caudal for ostial RCA.
- AP/LAO-cranial for ostial LM.

Step-by-Step Approach

- Lesion preparation:
  - Ostial lesions tend to have higher calcium and fibrous tissue content with increased elastic recoil. Therefore, we prefer to use cutting /scoring balloons prior to deployment of the stent.
  - Rotational atherectomy should be considered for heavily calcified lesions.
  - Excimer laser atherectomy can be used for aorto-ostial lesions particularly restenosis lesions.
- Stent positioning:
  - Since the guide is disengaged during stent positioning, it may be difficult to visualize the ostia. If possible, use the presence of ostial calcium to assist with stent positioning (Figs. 17.1 and 17.2).
  - Modified Szabo technique in aorto-ostial lesions: a second wire is placed and looped in the aorta to outline the sinus of Valsalva. It defines the junction of the coronary artery and aorta to assist with stent positioning and serves to provide further guide stability [2, 3].

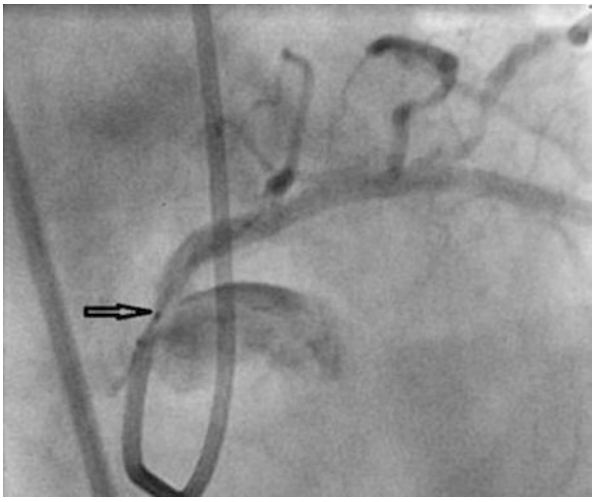

Fig. 17.1 Placement of an ostial left main stent in RAO-cranial view. *Arrow* marks the proximal edge of the stent at the ostium

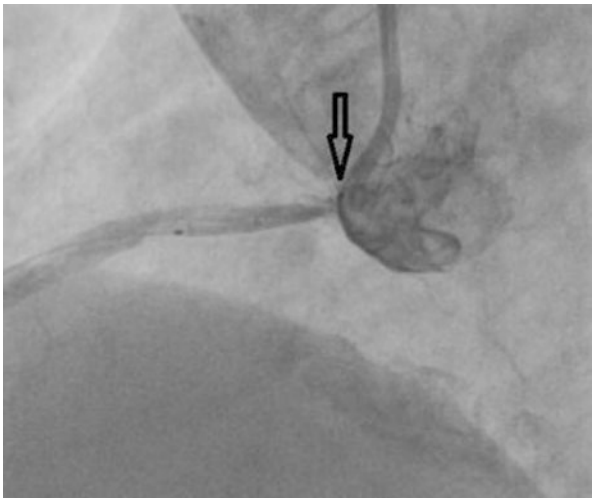

Fig. 17.2 Placement of an ostial RCA stent in LAO-caudal view. *Arrow* marks the proximal edge of the stent at the ostium

  - Stent pull-back technique in branch-ostial lesions: inflate a balloon at low pressure in the parent vessel (size balloon 1:1), then pull back the stent to the ostium of the SB to create a dent in the balloon that is positioned in the parent vessel [4].
  - It is important to know where the stent edge is relative to the radiopaque markers. Most currently available drug-eluting stent platforms have the stent just inside of the radiopaque markers, except for the XienceTM platform (Abbott Vascular), which crimps the stent at the center of the radiopaque marker. Use of collimation along with a high frame rate cine or stent boost can enhance visualization.
- Stent deployment:
- The stent should be positioned:
  - The stent should be positioned protruding into the aorta by 1–2 mm to prevent recoil of the lesion at the stent edge.
  - In aorto-ostial lesions with flush engagement of the guide, to ensure lesion coverage, we deploy the stent at low pressure 1–2 mm inside the guide, ensuring the lesion is covered and then unsheathe the remaining stent from the guide followed by full deployment at high pressure.
  - Avoid using very short (<12 mm) stents to ensure adequate anchoring of the stent and to provide adequate lesion coverage distally.
  - Size stent 1:1 ratio and deploy appropriately at high pressures (≥12 atm) to ensure optimal apposition.
  - In LM lesions inflation and deflation should be quick (repeat two to three times as needed).
  - After stent deployment, we often perform light "flaring" of the ostium of the stent with high pressure using 0.5 mm higher balloon size than the stent size.

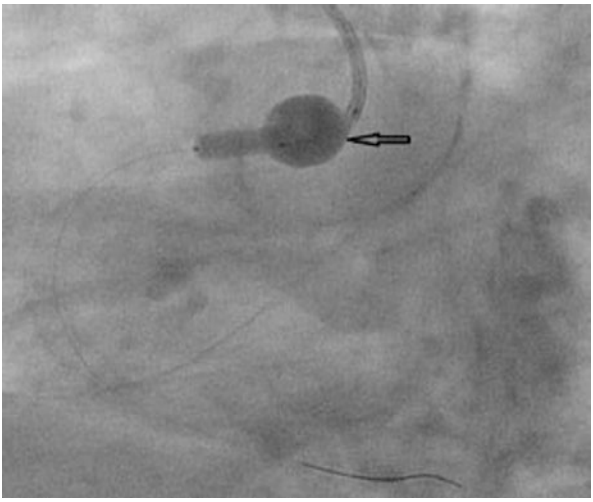

Fig. 17.3 Use of a Flash Ostial Balloon™ for post-dilatation of an ostial RCA stent. *Arrow* marks the outer compliant anchoring (proximal) portion of the balloon

- Flash Ostial Balloon™ may be used for flaring of ostial stent (Fig. 17.3).
- Use of post-intervention IVUS can help ensure adequate coverage of the lesion and stent expansion for optimal results and has been associated with a lower risk of major adverse cardiac events [5].

Accuracy of Stent Placement

- Accurate aorto-ostial stent placement requires the entire circumference of the proximal edge of the stent to be placed in the aorto-ostial zone (within 1 mm of the aorto-ostial plane).
- Geographic miss: diagnosed when at least a segment of the circumference of the proximal edge is located proximal or distal to the aorto-ostial landing zone (Fig. 17.4).
- Imaging assistance during PCI using OCT and IVUS has shown to help reduce the incidence of geographic miss [6, 7].

Complications [9]

- Guide catheter-induced dissection.
- Misplacement of the stent.
- Inadequate stent expansion.
- High restenosis rate.
- Side branch closure.
- Stent dislodgment.

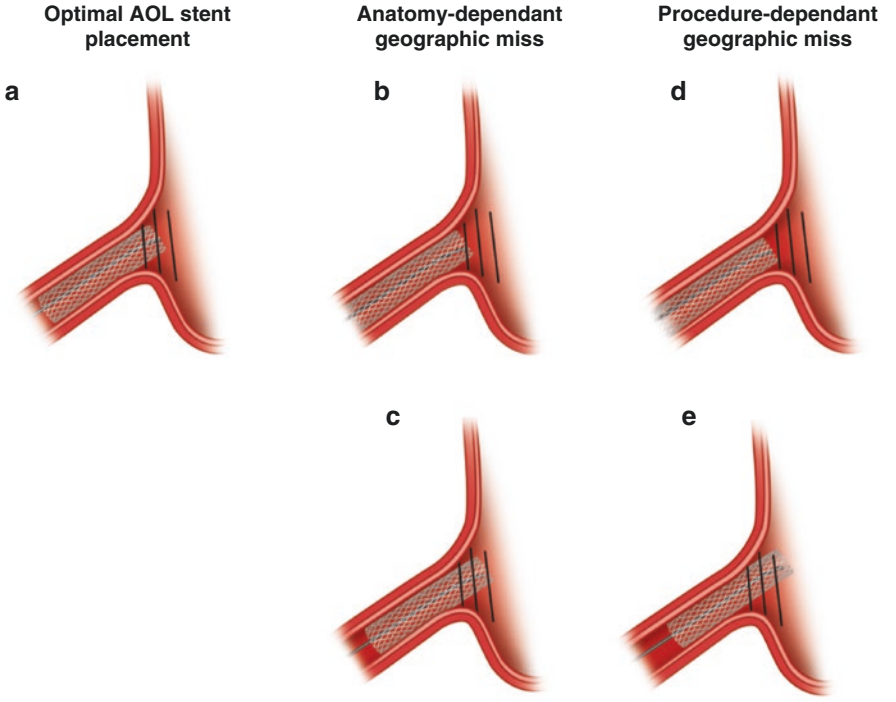

Fig. 17.4 Classification of geographic miss according to presumed underlying mechanism. Optimal stent positioning (**a**). When at least a segment of the stent edge is located within the aorto-ostial landing zone (AOLZ) (**b**, **c**), or when the stent edge extends both proximal and distal to the AOLZ, geographic miss may be attributed to the angulated coronary artery takeoff from the aorta preventing precise localization of the cylindrical stent structure within the constraints of the AOLZ and is therefore defined as anatomy-dependent. Geographic miss in which all aspects of the stent edge extend beyond the AOLZ either distally (**d**) or proximally (**e**) may be attributed to incorrect stent implantation technique and defined as procedure-dependent. (Adapted from Jaffe et al. [8])

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Left Main Coronary Interventions

18

Radha Mehta and Samin K. Sharma

Introduction

Interventions of the left main coronary artery (protected and unprotected) require special and careful consideration of other lesions assessment, lesion preparation, and proper device selection and placement.

Left Main Stenosis

Location

- Majority of left main lesions are distal bifurcation with 20% involving the isolated ostium and 10% involving isolated mid-body (Fig. 18.1).

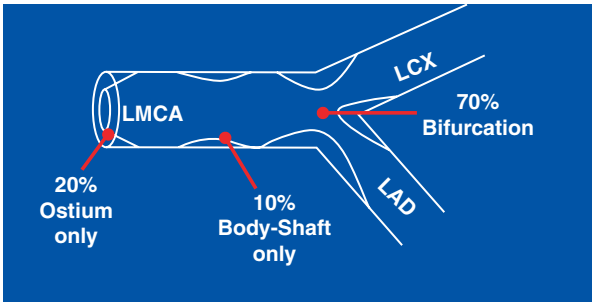

Fig. 18.1 Percentage of distribution of the location of involvement in left main stenosis

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Diagnosis of Left Main (LM) Disease

- Angiographic diagnosis of LM disease may not be accurate especially in intermediate lesions, especially in short LM lesions, and in diffuse disease process. Diffuse LM disease may conceal stenosis and cause the vessel to appear disease free.
- The identification of diffuse LM disease should be suspected when the reference diameter of the LM is similar to the reference diameter of the left anterior (Murray's law) descending coronary artery (LAD) [1].
- Additional imaging or physiological assessment is recommended if warranted.
- Role of intravascular ultrasound (IVUS):
  - The cut-off for intervention of LM is minimal luminal area (MLA) <6 mm^2 [2]. This has been shown to correlate with a fractional flow reserve (FFR)) <0.8 (Fig. 18.2).
- Role of FFR:
  - FFR values (>0.80) in left main lesions are associated with excellent long-term outcomes [3].
  - Presence of downstream disease may affect the FFR value of LM disease.
  - Myocardial bed supplied by the LM artery may be larger if it supplies collaterals to an occluded RCA.
  - It is postulated that the presence of a significant lesion in either the LAD or the LCX makes the myocardial bed smaller for the LM leading to a falsely higher FFR. The left main FFR alone cannot be accurately measured when there are significant downstream serial lesions. If the LAD and LCX are hemodynamically insignificant, the left main FFR will be accurate.

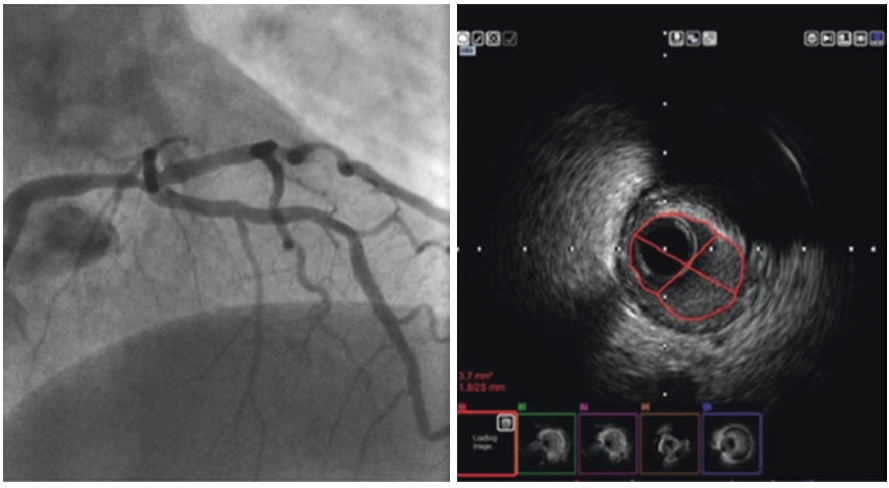

Fig. 18.2 Moderate LM disease by angiography and IVUS showed MLA of 3.7 mm^2

Supporting Evidence for Percutaneous Intervention for Left Main Stenosis

The EXCEL, NOBLE, SYNTAX, and PRECOMBAT trials had a direct comparison of PCI vs CABG for left main intervention.

EXCEL Trial [4]

- 1905 patients
- Primary endpoint: a composite of death from any cause, stroke, or MI.
- At three years, the primary endpoint occurred at a similar rate in both groups (15.4 versus 14.7 percent p = 0.018 for noninferiority, p = 0.98 for superiority).
- At 5 years: no difference in the primary outcome in both groups (22.0 versus 19.2 percent, odds ratio 1.19, 95% CI 0.95–1.50, p = 0.13).
- Death from any cause, a secondary outcome, occurred more frequently in the PCI group (13.0 versus 9.9 percent).
- The rate of ischemia-driven revascularization was higher with PCI (16.9 versus 10.0 percent).
- Thrombosis (stent/ graft) was higher in CABG vs PCI (6.5% vs.1.6%).

NOBLE Trial [5]

- 1201 patients.
- Primary endpoint: composite of all-cause mortality, non-procedural MI, any repeat coronary revascularization, and stroke.
- At 5-year, Kaplan–Meier estimates of the primary endpoint were 28 and 19 percent, respectively (HR 1.58, 95% CI 1.24–2.01)]. There was no difference in all-cause mortality (9 percent in both groups).
- Revascularization occurred significantly more often after PCI as did non-procedural MI (17 versus 10 and 9 versus 3 percent, respectively).

PRECOMBAT Trial [6]

- 600 patients.
- Primary endpoint: death from any cause, MI, stroke, or ischemia-driven target vessel revascularization.
- Follow-up at 1 year showed noninferiority of PCI over CABG. The event rates were 8.7 and 6.7 percent for PCI and CABG (absolute risk difference 2, 95% CI-1.6 to 5.6, respectively) mainly driven by the higher rate of ischemia-driven target lesion revascularization in the PCI group (6.1 versus 3.4 percent).
- At five years, the event rates for the primary outcome were not statistically significant with 17.5 and 14.3 percent in the PCI and CABG groups.

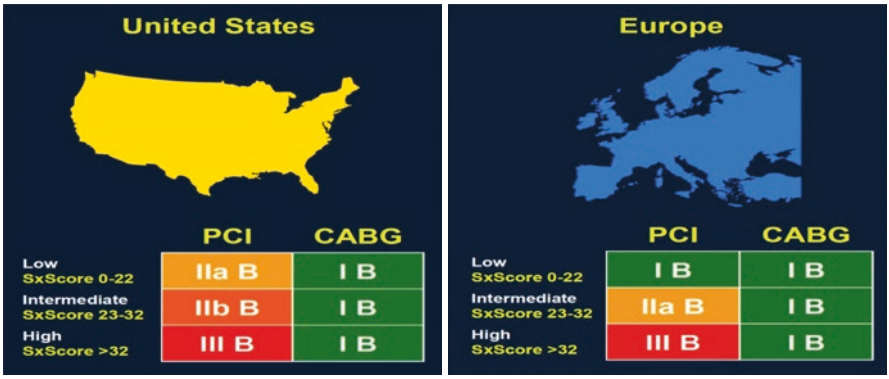

Fig. 18.3 Current Guidelines for CABG vs PCI for Left main and multivessel disease. Adapted from Windecker et al. and Fign et al. [7, 8]

Ischemia-driven target lesion revascularization remained the principal reason for the higher rate in the PCI group.

PCI may thus be considered an acceptable revascularization modality for selected patients with left main CAD, a decision which should be made after heart team discussion, taking into account each patient's individual risk factors and preferences.

Current Guidelines [Fig. 18.3]

Protected Versus Unprotected LM Intervention

- PCI is the preferred approach in protected LM because of the increased risk associated with repeat CABG. Recent and emerging data have shown that PCI is safe in unprotected LM.

Need for Hemodynamic Support

- Hemodynamic support devices are rarely required if LV EF > 50%. We recommend the use of IABP for EF 35–50% and Impella for EF < 30%. In rare cases of complex distal left main with occluded RCA (or non-dominant), we recommend the use of an IABP or Impella to avoid hemodynamic deterioration even when the LV function is normal (Fig. 18.4).

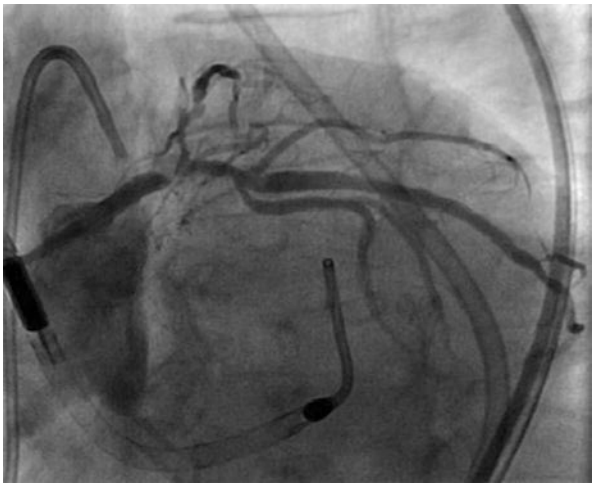

Fig. 18.4 Patient with significant LM disease and LVEF 15%; PCI of LM was performed with Impella support

Planning for Intervention: Simple Versus Complex Lesion (DEFINITION Study)

Simple vs. Complex Lesions

- A simple LMB lesion has SB diameter stenosis <70% and lesion length < 10 mm. This is seen in 75% of cases and can be treated with a single-stent provisional approach.
- A complex LMB lesion has SB diameter stenosis >70% and lesion length > 10 mm. A simple lesion can change to a complex lesion with the presence of 2 of the following 6 minor criteria:
  - 1) Moderate to severe calcification.
  - 2) Multiple lesions.
  - 3) LAD-LCx bifurcation angle >70.
  - 4) Main vessel reference vessel diameter < 2.5 mm.
  - 5) Thrombus-containing lesion.
  - 6) Main vessel lesion length > 25 mm.
- Complex lesions generally require a 2-stent strategy.

Femoral Versus Radial Approach

- Both approaches are suitable, but the femoral approach is preferred if there is a need for a larger size guide (>6Fr), rotational/orbital atherectomy, or hemodynamic support.

Optimal Angiographic Views

- AP caudal, AP cranial, LAO cranial, and LAO caudal for visualization of bifurcation.

Approach

Ostial LM

- Sheath size: 6F or 7F,
- Guide selection: FL or VL (with side holes). Avoid aggressive guides and deep engagement and ventricularization.
- Wires: Runthrough, Fielder. Buddy wire may be needed to provide additional support.
- Lesion preparation/debulking: Predilation with compliant or semi-compliant balloons. May need Flextome/AngioSculpt for adequate vessel preparation. Rotational or orbital atherectomy may be required for heavily calcified lesions (if rotational atherectomy of LM is planned, place a temporary pacing wire). If using rotational atherectomy, disengage the guide slightly and start rota from inside the guide so that the ostium is also adequately prepared.
- Stent selection: DES is preferred.
- Stenting strategies.
  - Disengage the guide slightly but keep it close enough to enable adequate visualization of the vessel.
    While holding wires taut, pull back the guide slowly to disengage it from the ostium but while maintaining wire and guide control.
    Place stent to cover the lesion with a slight overhang into the aorta.
    Deploy stent with inflation of stent balloon to high pressures at the ostium.
    Postdilate stent and "flare" ostium with high-pressure inflation of the noncompliant balloon with a portion of the balloon protruding into the aorta.
    Flash ostial balloon may be used for flaring of the ostium of the stent.
  - Modified Szabo technique.
    Wire lesion.
    Place a second, steerable wire in the aorta to stabilize the guide.
    Place stent over the initial wire and position with 1–2 mm ostial overhang into the aorta.
- Postdilate with final "flaring" of ostium with a noncompliant balloon with high pressure using 0.5 mm higher balloon size than the stent size.

Mid-LM

- Sheath size: 6F.
- Guide selection: VL vs EBU vs FL.

- Wires: Runthrough, Fielder.
- Lesion Preparation/Debulking: Predilation with compliant or semi-compliant balloons. May need Flextome/AngioSculpt for moderately calcified vessels and rotational atherectomy may be required for moderate to severely calcified lesions (temporary pacing wire may be needed if rotational atherectomy is planned).
- Stent selection: DES is preferred.
- Stenting strategies: true-mid LM lesions have landing zones proximally and distally, and hence a focal stent placement after adequate preparation of the lesion is feasible. Postdilate to high pressures using a noncompliant balloon.

Distal LM

- Sheath size: 7F or 8F.
- Guide Selection: VL vs EBU.
- Wires: Runthrough, Fielder. Wire both the LAD and LCx and wire the more significant or tighter lesion first. If rotational atherectomy is considered, wire only the vessel that is heavily calcified either directly with a RotaWire/Viperwire or exchange with a Fielder wire/ Finecross microcatheter.
- Lesion preparation/debulking: Predilation with compliant or semi-compliant balloons. May need Flextome/AngioSculpt for moderately calcified vessels. Rotational or orbital atherectomy may be required for moderate to severely calcified lesions (Fig. 18.5). Temporary pacing wire may be needed if rotational atherectomy is planned.
- Stent selection: DES is preferred.
- Stenting strategies: follow approach to treatment of LM bifurcation lesions as detailed in Fig. 18.6.

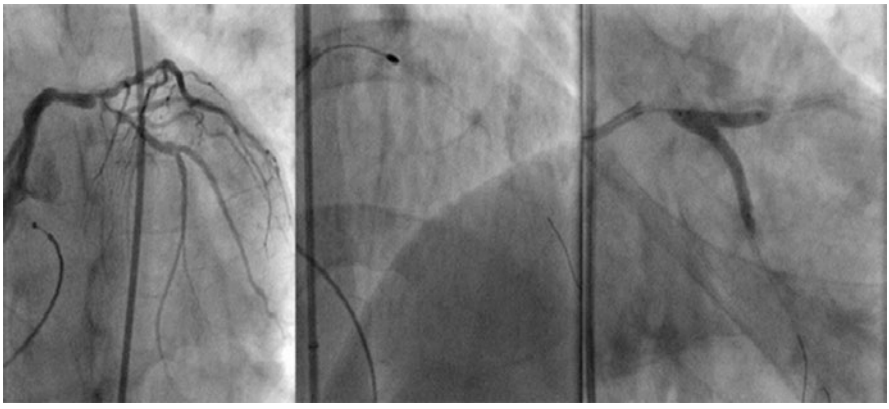

Fig. 18.5 Distal LM disease being debulked by rotational atherectomy

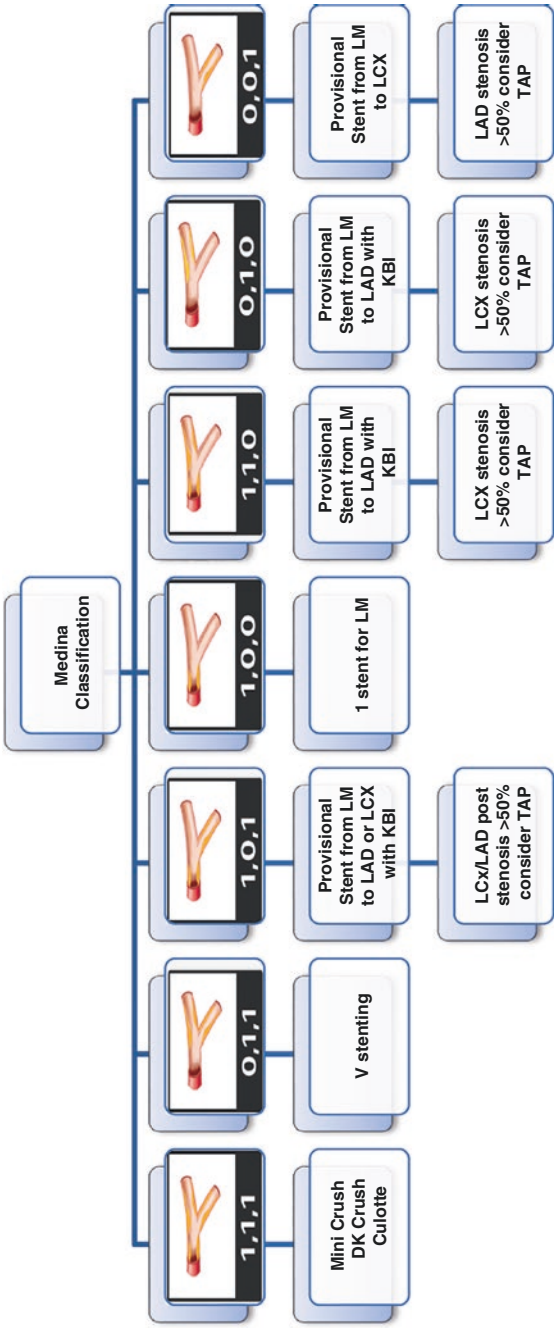

Fig. 18.6 Strategies for left main intervention based on the Medina classification, modified from Bifurcaid app with permission

Provisional Stenting

- If no significant disease in side branch(Medina 1,1,0 or 0,0,1 or 0,1,0).
- Wire the MBV with Runthrough and the SBV with Fielder.
- Adequate lesion preparation and debulking should be performed.
- Confirm the position of the stent in MBV and deploy.
- Withdraw SBV wire and rewire SBV through MBV stent strut.
- Final kissing balloon inflation (KBI).

Two-Stent Strategy

- Bifurcation angle can also help guide the intervention strategy. For wider bifurcation angles >70 or when the LCx is smaller than the LAD (but larger than 2 mm), DK crush is the ideal 2-stent technique. If the bifurcation angle is <70 and the LCx diameter is within 0.5 mm of the LAD diameter, either the Culotte or the DK crush technique can be performed.
- SKS or V stenting has advantages of relative ease of the procedure and good angiographic results obtained with little hemodynamic disruption also, the need to rewire the side branch is eliminated. If the bifurcation lesion is Medina [0,1,1], then the best strategy would be V stenting strategy. For SKS, PMV must be large enough and at least 2/3 the size of (DMV + SBV).
- Below is a brief description of different two-stent strategies. For a detailed description of two-stent strategies, *see Bifurcation Interventions Chapter 16.*

SKS/V Stent

- Rarely used except in emergent cases.
- SKS: >2 mm overlap in LM, V stent: ≤2 mm overlap in LM.
- Wire the MBV with the Runthrough wire and wire SBV with the Fielder wire.
- Lesion preparation and debulking should be performed as necessary.
- Both stents positioned with proximal overlap with both markers exactly aligned.
- Simultaneous stent deployment in MBV and SBV (equal pressures) with careful deployment to avoid risk of proximal vessel dissection.
- Final kissing balloon inflation (KBI) (Fig. 18.7).

Mini Crush

- Wire the MBV with the Runthrough wire and wire SBV with the Fielder wire.
- Lesion preparation as needed.
- Place SBV stent with ≤2 mm protrusion into the MBV.
- Deploy SBV stent, and then remove wire and stent balloon.
- Deploy MBV stent (completely cover and crush protruding SBV stent).

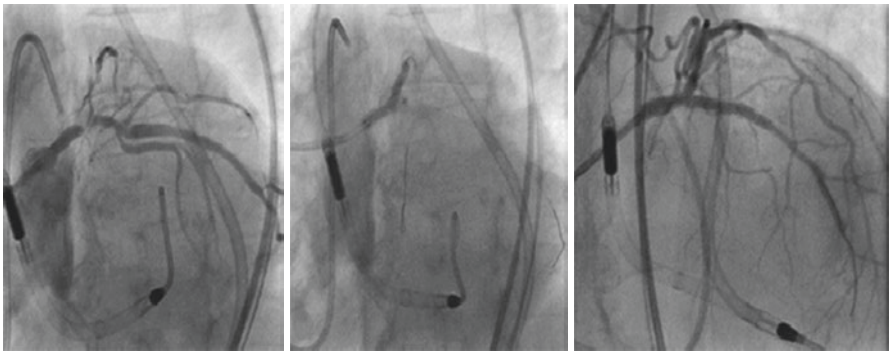

Fig. 18.7 SKS stenting of LM disease with Impella support

- Rewire SBV stent through struts of MBV stent and advance a noncompliant balloon (if difficulty is encountered, use a compliant balloon first) to dilate the ostium adequately.
- Final KBI.

Double Kissing CRUSH (DK-CRUSH)

- Wire the MBV with the Runthrough wire and wire SBV with the Fielder wire.
- Adequately prepare and stent the SBV first.
- Balloon the MBV with a noncompliant balloon (First Crush).
- Perform first KBI.
- Take the SBV wire out.
- Stent the MBV (Second Crush).
- Rewire the SB through MV stent struts.
- Perform second KBI.

Culotte Stenting

- Optimal when significant diffuse side branch and main vessel disease.
- Wire the MBV with the Runthrough wire and wire SBV with the Fielder wire.
- Lesion preparation and debulking should be performed as necessary.
- Place and deploy SBV stent with the proximal end extending into the PMV for 3–5 mm.
- Remove the jailed wire and rewire the DMV through the SBV stent struts.
- Advance balloon over the wire and dilate to open the stent cell in preparation for stenting the MBV.
- Remove the SBV wire.
- Advance the MBV stent and position it to overlap the proximal portion of the SBV stent.

- Deploy the stent and rewire the SBV.
- Final KBI.

T-Stenting and Small Protrusion (TAP)

- Used when prior patent MV stent or as a bailout after provisional stenting.
- Wire the MBV with the Runthrough wire and wire SBV with the Fielder wire.
- Lesion preparation as needed.
- Place MBV stent.
- Remove the jailed wire from SBV.
- Rewire the SBV through the MV stent struts.
- Place SBV stent with 1–2 mm protrusion into the MBV.
- Advance balloon in the MBV.
- Deploy the stent in SBV.
- Pull the stent balloon system back, align it with the MV balloon, and inflate both the balloons simultaneously (Fig. 18.8).

Trifurcation of Distal LM

- If the distal LM trifurcates into LAD, LCX, and ramus, the treatment of trifurcation lesion could be treated as bifurcation lesion with LM and two larger branches of the three, and leave the smaller of the three branches alone.

Imaging after Stent Placement

- IVUS or OCT can be used to assess adequate stent expansion, complete stent strut apposition, and rule out any dissections. At 3 years, in patients receiving DES for LM lesions, a mortality benefit was seen with IVUS guidance compared with angiographic guidance (4.7% vs.16.0%). Randomized trials, however, have failed to show a clinical benefit of IVUS use.

Antiplatelet Selection

- DAPT is recommended at least for 1 year.

Surveillance Angiography

- There is no evidence for routine use of surveillance angiography, and hence, we recommend repeat angiography only if clinically indicated.

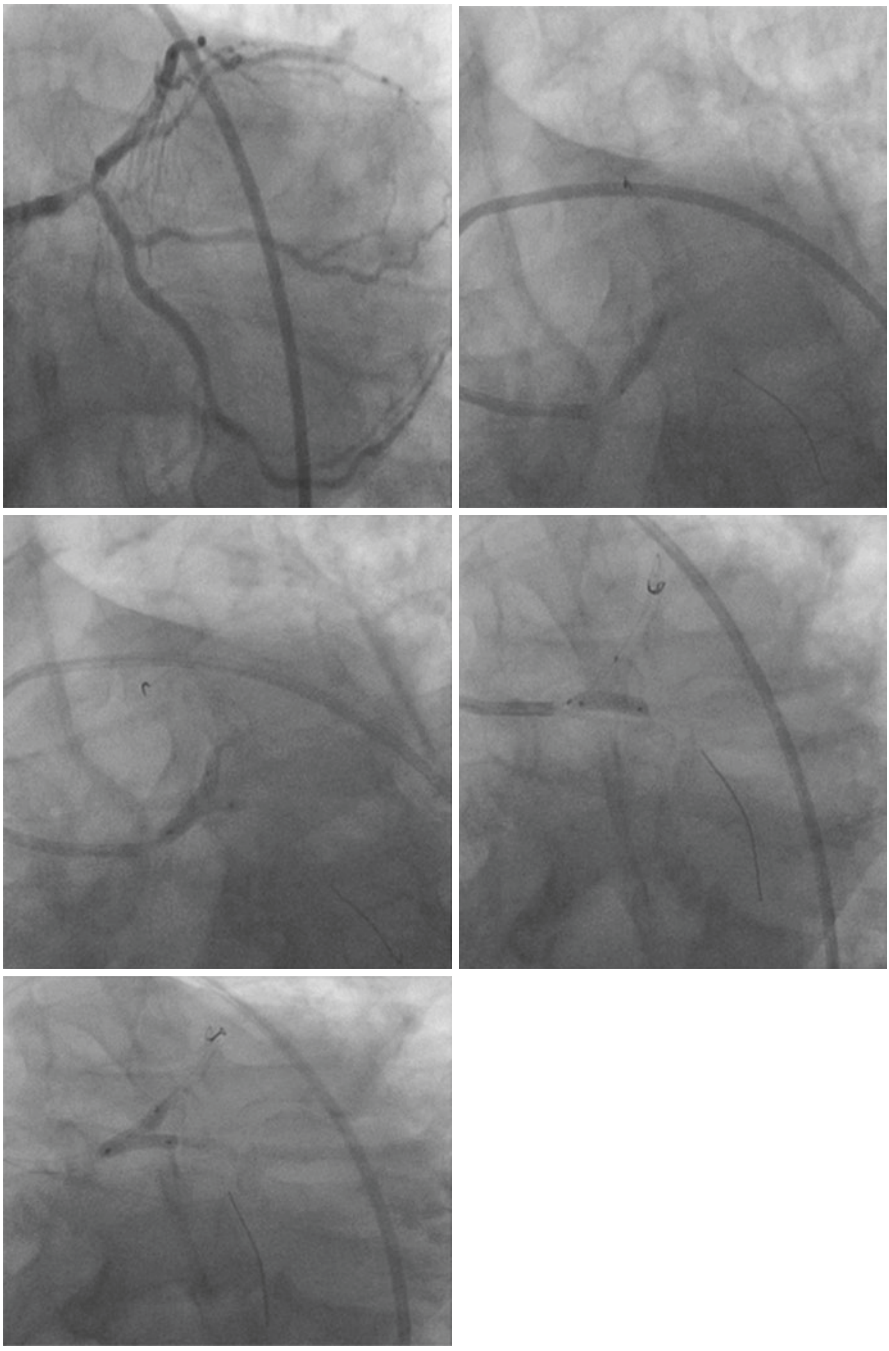

Fig. 18.8 Tap technique for distal LM disease

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Chronic Total Occlusions

19

Lorenzo Azzalini, Gurpreet S. Johal, and Annapoorna Kini

Introduction

Chronic total occlusion (CTO) is defined as a 100% stenosis with Thrombolysis In Myocardial Infarction (TIMI) 0 flow for more than 3 months. These lesions are found in approximately one fifth of coronary angiograms, but they have traditionally been undertreated due to a lack of awareness of pertinent data demonstrating benefit of CTO PCI, combined with traditionally low success rates and high complication rates. However, recent randomized trials have consistently demonstrated improved quality of life and physical function following successful recanalization of CTOs, compared to optimal medical therapy. The present chapter will deal with the technical aspects of CTO PCI.

CTO Principles for Best Practice

There are 7 shared principles that are widely accepted as best practices for CTO PCI [1]:

1. The primary indication for CTO PCI should improvement of ischemic symptoms.
2. Dual coronary angiography, as well as a meticulous and methodical review of the angiogram, is key for planning and safely performing CTO PCI.
3. Use of a microcatheter is fundamental for optimal guidewire manipulation and exchanges.
4. Antegrade wiring, antegrade dissection and re-entry, and the retrograde approach are complementary and necessary crossing strategies. While antegrade wiring

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(also known as antegrade wire escalation) is the most common initial technique, the retrograde approach and antegrade dissection and re-entry are often required for CTOs that are more complex.
5. If the initial crossing strategy fails, early change to a different technique increases the likelihood of successful crossing, and maximizes procedural efficiency.
6. Dedicated CTO PCI training and expertise, as well as sufficient case volume and the availability of specialized equipment, will increase the likelihood of successful recanalization while facilitating prevention and management of complications.
7. Following successful occlusion crossing, great attention to lesion preparation and stenting technique (often using intracoronary imaging) is required to ensure optimal stent expansion, thus maximizing long-term patency rates.

Angiogram Analysis

CTOs have certain favorable and unfavorable characteristics that should be looked for in detail during angiography (Table 19.1).

- There are several scores available to grade the procedural difficulty of a CTO, the most widely used being the Japanese-CTO (J-CTO) score [2]. This score comprises several of the aforementioned unfavorable characteristics, as shown in Fig. 19.1. The J-CTO score has been shown to correlate with success rates using a variety of crossing algorithms [2, 3].

Table 19.1 Favorable and unfavorable characteristic of CTOs

Favorable characteristics	Unfavorable characteristics
Short segment (<20 mm)	Long segment (>20 mm)
Tapered stump	Blunt stump
No side branches or bridging collaterals at proximal cap	Side branches at the proximal/distal cap or within the occlusion
Straight segment	Tortuosity within the occlusion
Functional CTO (distal vessel visualized through faint intraocclusional channel)	Lack of interventional collaterals
	Ostial CTO
	Circumflex CTO
	CTO in post-CABG patients
	Calcification

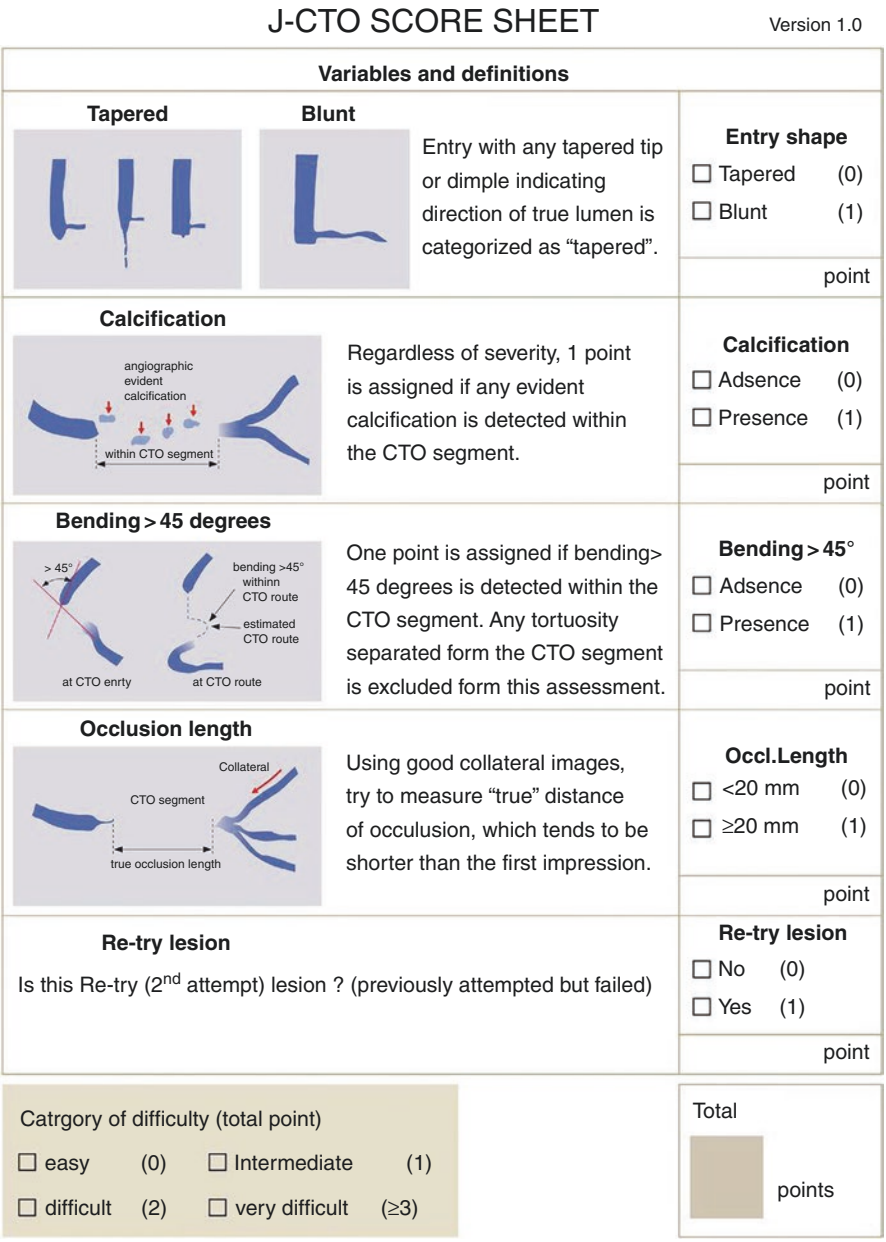

Fig. 19.1 J-CTO score to evaluate CTO complexity

Good CTO PCI Clinical Practice

The following strategies should be used on a day-to-day basis to maintain good CTO PCI clinical practice:

- "CTO days" in the cath lab schedule: this will allow the operators to exclusively focus on few cases, maximizing procedural safety and cath lab workflow
- Avoid ad hoc CTO PCI: this will allow careful preprocedural planning, as well as a thorough discussion of the pros/cons, risks, expected benefits, and alternatives of the procedure.
- Evaluate the patient's risk of developing contrast-induced acute kidney injury preprocedural with validated scores.
- Adequately hydrate the patient pre- and post-procedure.
- Predetermine maximum contrast volume that can be used (contrast volume/eGFR ratio <3).
- Minimize unnecessary contrast media injections (e.g., "tests").
- Maintain ACT >250 s if an antegrade approach is used, 300–350 s if the retrograde approach is employed.
- Check ACT every 30 min.
- Use low-dose radiation settings (e.g., 7.5 fps, low-dose fluoroscopy modality), avoid unnecessary cines, avoid steep projections, change projections frequently, minimize the distance between the x-ray tube and the intensifier.

Crossing Strategies

A systematic approach to CTO PCI maximizes success rates as well as procedural efficiency and safety.

- Traditionally, there have been two different "philosophic" approaches to CTO PCI, the North American and the Japanese, whose main differences are based on the tenet of maximizing procedural efficiency (thus allowing ample use of dissection and re-entry techniques) for the former, and striving to maintain an intraplaque track through the occlusion for the latter (thus frequently adopting parallel wiring and intravascular ultrasound-guided wiring).
- Such approaches have led to the development of the Hybrid Algorithm [4] (Fig. 19.2) and the Asia-Pacific Algorithm [5].

CTO Procedural Components

Vascular Access

- Different access route combinations.
  - Single femoral access or radial access if no collaterals are seen from the contralateral vessel.
  - Dual femoral access.

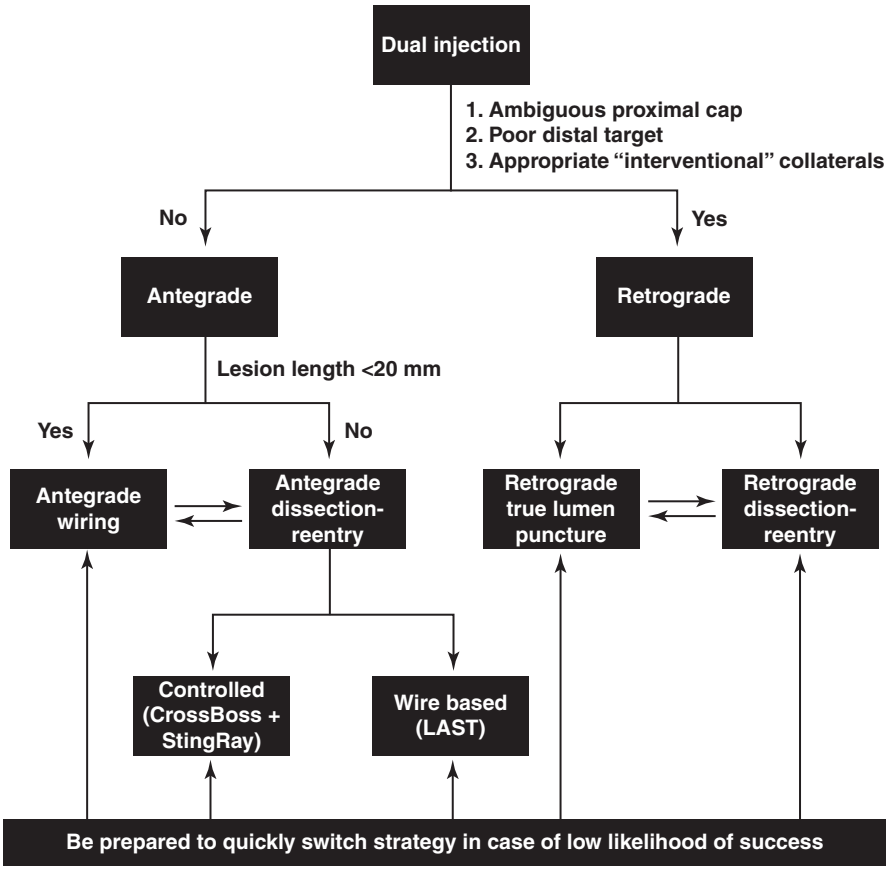

Fig. 19.2 Hybrid Algorithm. (Abbreviations: LAST, limited antegrade subintimal tracking)

  - Femoral and radial access.
  - Dual radial access.
- CTO PCI requires solid guide catheter support, which is achieved using aggressive curves (e.g., AL catheters for the right coronary artery), guide catheter extensions, and especially large-bore catheters (7 Fr and 8 Fr). Therefore it is not surprising that CTO PCI makes ample use of the femoral route, as three-quarters of procedures involve the use of at least one femoral access [6, 7]. However, when judged feasible by the operator, the radial approach should be considered, as it has been associated with decreased vascular complications and bleeding also in CTO PCI [6, 7].
- For most cases, we usually recommend a left transradial approach for contralateral injection (and possibly the retrograde approach) and a right femoral approach as the main access to recanalize the CTO.

Sheath

- Femoral: long (e.g., 45 cm) 6–7 Fr sheaths.
- Radial: 6 Fr sheaths (e.g., Terumo Glidesheath Slender allows using 7 Fr guide catheters in a sheath that has the outer diameter of a 6 Fr sheath).

Guides

- Selection of guide support is an important step in CTO interventions. It depends on vessel origin, tortuosity, size of the aortic root, site of CTO in vessel segment, and experience of the operator (see Table 19.2).
- Guide support depends on coaxial alignment and stability of the guide. It can be further maximized with the following techniques:
  - *Mother–and–child technique*: Use of guide catheter extension. This is advanced into the coronary using balloon-assisted tracking or distal balloon anchoring.
  - *Balloon anchoring:* Inflate a small semi-compliant balloon in a side branch which is proximal to the occlusion and not a source of collaterals.

Guidewires

- Guidewire selection is a critical step in CTO recanalization (Table 19.3). Each wire has specific properties that make it suitable for a specific task. These are:
  - Tip load: Guidewire tip load can be classified in low (<2 g), intermediate (2–6 g), and high (>6 g). Low tip-load guidewires are utilized to get a first impression of the resistance of the proximal cap, and in soft parts of the occlusion. Intermediate tip-load guidewires are used in more resistant parts of the occlusion, particularly when steering towards a different plane is sought. High tip-load guidewires are utilized to puncture-resistant caps and to deal with highly calcified areas.

Table 19.2 Guide selection for CTO

CTO vessel	Guide
Left main CTO—ostial	FL
LAD and long left main	VL/EBU/FCL
LCX	VL/EBU
RCA ostial	FR (with side holes)
RCA	AL 0.75/IM
RCA upsloping	AL 0.75/SCR

Table 19.3 Techniques for guidewire manipulation in CTO PCI

Technique	Purpose	Examples
Puncturing	Penetrating the proximal (or distal) cap using a high tip-load tapered-tip guidewire.	Confianza Pro family, Hornet family
Drilling	Navigating through fibrocalcific parts of the occlusion by means of a quickly rotating (90 degrees to one side, then 90 degrees to the other) and pushing an intermediate-tip load guidewire.	Gaia Family, Miracle family
Sliding	Using a polymer-jacketed tapered-tip low tip-load guidewire in a gentle and controlled spinning fashion to navigate through soft parts of the occlusion (e.g., "microchannels").	Fielder family, Fighter
Push-deflect-torque	Advance the guidewire until it is deflected by fibrocalcific tissue, retract 1 mm, steer a few degrees away, then push again.	Gaia family
Knuckling	Advancement of a polymer-jacketed guidewire into the subintimal space until it acquires a knuckle-like shape: this will be advanced along the path of least resistance to safely navigate long coronary segments.	Pilot 200, Fielder XT family
Retrograde approach	Use of a very low tip-load guidewire (<1 g) with high-torque response to navigate through a retrograde channel (septal, epicardial, bypass graft).	Sion, Sion Black, Fielder XT-R, Samurai RC

  - Presence/absence of a tip taper: Tapered-tip guidewires are designed to enhance lesion crossability and to increase penetration power in fibrocalcific segments.
  - Presence/absence of a polymer jacket: The polymer jacket increases lubricity, enhancing movement through tortuosity or areas of tissue resistance (particularly when knuckled), and may confer resistance to vessel exit. Non-jacketed guidewires provide better engagement at points of resistance and are less easily deflected.
  - There are several ways in which guidewires can be manipulated, as shown in Table 19.3.
- Wire selection is guided by the following characteristics of CTO:
  - Presence of micro-channels.
  - Calcification.
  - Dissection.
  - Type of proximal cap: blunt or tapered.

CTO with Micro-Channels

- Low-tipload polymer-jacketed wires should be the first choice:
  - Fielder (1st-generation).
  - Fielder XT (2nd-generation): tapered tip (0.009").
  - Fielder XT-A/XT-R (3rd-generation): tapered tip (0.009") and dual-core (1:1 torque response).

CTO with Calcification

- Wires with more penetration power and tip load are used in such cases.
- Wires can be escalated depending on the resistance to wire advancement offered by the plaque.
- The following escalation sequence is recommended:
  - Gaia First/Second.
  - Gaia Third/MiracleBROS.
  - Confianza Pro 12/Hornet 14/Progress 200T.
  - Astato 20.

Tips and Tricks for CTO Wiring

- Single 30–45° bend within 1–2 mm from the tip (Fig. 19.3).
- Tactile feedback from the tip should be continuously assessed by the operator. This is better with modern dual-core wire (e.g., Fielder XT-A/XT-R, Gaia Family).
- Observe and monitor the movement of the tip. Free tip movement can represent distal true lumen wiring, but also wire exit into the pericardial space (especially if the movement is excessive and/or the wire track does not follow the expected vessel course as observed in multiple angiographic projections).
- Contralateral injection is fundamental to check guidewire tip position with regard to the distal vessel.
- Different angiographic views to confirm the position of wire with respect to true lumen.

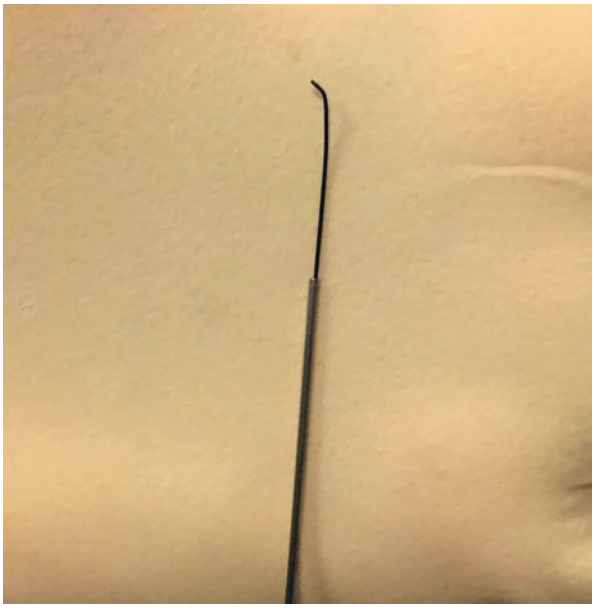

Fig. 19.3 Typical CTO guidewire tip

Microcatheters

- Use of a microcatheter is fundamental both for the antegrade and retrograde approach as it greatly enhances guidewire support and maneuverability, facilitates guidewire exchange, and protects collaterals (with the retrograde approach).
- Microcatheters can be classified as low-torque or high-torque.
  - Low-torque devices have a simple braided structure to resist kinking, yet minimize crossing profile. Examples include Terumo Finecross (blunt tip) and Asahi Caravel (tapered tip).
  - High-torque devices have an addition of a coil. This increases torque transfer from hub to tip, enabling the device to be spun, thus reducing static friction (e.g., Teleflex Turnpike family, Asahi Corsair, OrbusNeich Teleport). This function is useful when traversing long collaterals or occlusions.
- The intended function of a microcatheter determines which design type is selected. Specialty microcatheters, such as angled-tip (Teleflex Supercross), deflectable-tip (Teleflex Venture) or dual-lumen (Teleflex TwinPass Torque) microcatheters exist.

Traversing the CTO Length

- Once the wire crosses the proximal cap, advance the microcatheter through it.
- Consider whether to escalate/de-escalate depending on the characteristics of the plaque at the guidewire tip site.
- If the wire buckles, it should be retracted, reoriented, and rotated rather than forced through a lesion.
- Use landmarks such as a prior stent or calcification as a guide to wiring.
- Monitor guidewire advancement in multiple fluoroscopic projections.
- Use contralateral injection to monitor guidewire advancement.

Parallel Wire Technique

- In the parallel wire technique, if the first wire enters the subintimal space, it is left there. This will have three functions:
  1. Mark the channel to subsequently avoid it.
  2. Occlude the channel to prevent hematoma development.
  3. Straighten the vessel in the occlusion segment.
- Subsequently, a second wire (usually of the same or higher tip load) is used to attempt true lumen wiring.
- This technique can also be used with two microcatheters, allowing for easy exchange of the two wires (seesaw technique).

After Crossing

- Once the CTO is crossed anterogradely, confirm the position of the wire in the true lumen by contralateral injection. Injection via the microcatheter tip is not advised because it may extend the dissection, in case of an actual subintimal situation.
- Advance the microcatheter until the distal vessel is reached, and then exchange the guidewire used for crossing with a workhorse wire.
- The microcatheter is then removed. This can be accomplished with four techniques:
  - Trapping: the preferred method, as it ensures minimal guidewire displacement compared with the use of guidewire extensions or hydraulic exchange. With trapping, a balloon (2.0/2.5 mm for 6-Fr guiding catheters, and 2.5/2.75 mm for 7-Fr; dedicated trapping balloons now also exist) is advanced freely (i.e., not onto a wire) into the guiding catheter, distal to the position where the tip of the microcatheter to be extracted is located (the latter has to be previously pulled inside the guide catheter as proximal as possible). Balloon inflation "pins" the guidewire against the catheter wall, thus preventing its movement. The microcatheter is then safely pulled out.
  - Guidewire extension: each guidewire manufacturer has its own specific extension, which has to be kept in mind when choosing the right guidewire extension. Slack in the guidewire/extension system might cause loss of distal guidewire position, so great care should be used if this technique is employed.
  - Hydraulic extraction (Nanto technique): the indeflator is connected to the guidewire lumen of the microcatheter after pulling back the latter until the proximal end of the guidewire is at the hub level. The microcatheter is further pulled back while applying constant inflation pressure with the indeflator, leaving the guidewire in the coronary artery. The nano technique is easier to perform than trapping, but also less reliable in maintaining distal guidewire position.
  - 300-cm guidewire.

Microcatheter Not Crossing (Uncrossable Lesion)

- Several techniques can be used if the microcatheter is not crossing following antegrade wiring of the lesion:
  - Exchange for a different microcatheter. Usually, braided tapered-tip microcatheters (Turnpike family, Corsair) perform best in uncrossable occlusions. This is accomplished by quick spinning advancement through the lesion.
  - Dilatation with very small profile CTO-dedicated balloons (1.0 mm; e.g., Sapphire II Pro) and crossing reattempt with the same microcatheter.

- Guide catheter extension and 1.0/1.2 mm balloon.
- Laser (0.9" catheter). This has the advantage that it can be performed over any coronary wire so that control on the distal true lumen is maintained.
- Rotational atherectomy (1.25 mm burr). The microcatheter is abutted to the lesion and the guidewire is exchanged for Rota floppy wire. Rotational atherectomy is performed. The microcatheter is advanced and the RotaWire is exchanged with a workhorse wire. This technique has the disadvantage that guidewire position is given up and RotaWire might not be able to reach the distal true lumen.

Retrograde Approach

- The prerequisites to use the retrograde approach are the following:
  - Appropriate collaterals exist AND.
  - There is local experience with the retrograde approach AND.
  - The antegrade approach fails OR.
  - In the following scenarios:
    Ostial occlusion.
    Ambiguous proximal cap or stumpless occlusion.
    Long occlusion.
    Severe proximal tortuosity or calcification.
    Small or poorly visualized distal vessel.
    CTO vessel that is difficult to engage (e.g., anomalous coronary artery).
    Occlusion involving a major bifurcation.

Selection of Collaterals

- Septal channels are the safest and should always be tried first, as perforations of these vessels are almost always benign.
- Septal tortuosity can be an issue both for wiring and microcatheter advancement. Novel guidewires (e.g., Suoh03) have greatly improved crossing rates.
- In addition, invisible septal collaterals can be wired (the tapered-tip Fielder XT-R performs best in this case).
- Septal crossing can be guided by tip injection through the microcatheter or be "blind" ("septal surfing").
- Bypass grafts can be an excellent route for the retrograde approach, as they have no side branches and are large in size.
- Challenges might arise identifying the distal anastomosis (in case of occluded grafts) and wiring the native coronary artery in a retrograde fashion (as the anastomosis angle is often >90 degrees).

Retrograde CTO Steps

- *Step 1: Septal collateral wiring*. Selection of suitable septal collaterals: preference should be given to visible septal connections with little tortuosity. Usually, septal origin is profiled better in RAO view with slight cranial or caudal projections.
  - Profile the septal collaterals.
  - Enter the septal artery with a workhorse wire with a large curve.
  - Advance the microcatheter into the septal.
  - Exchange the workhorse wire with a dedicated retrograde wire (Sion, Sion Black, Suoh03, Fielder FC, Samurai RC)
  - Wire the septal using an iterative, gentle, back-and-forth spinning motion (see Fig. 19.4).
  - Once the wire reaches the distality of the occluded vessel, the microcatheter is advanced.
- *Step 2: Crossing the distal cap, traversing the length, and crossing proximal cap*:
  - Initial attempt with a low tip-load polymer-jacketed wire (e.g., Fielder family).
  - If unsuccessful (e.g., due to calcification), retrograde wire escalation can be performed (Pilot 200, Gaia family, Confianza Pro family, etc.) (Fig. 19.5).
  - The most commonly used final crossing technique in retrograde cases is, however, reverse controlled antegrade and retrograde subintimal tracking (reverse CART), with which a connection is created between the spaces occupied by the antegrade and retrograde guidewires by balloon dilatation on the antegrade wire.
- *Step 3: Externalizing the wire*:
  - Advance the wire into antegrade guide (Fig. 19.6).
  - Trap the wire with a 2.0/2.5 mm balloon in the antegrade guide (Fig. 19.7).
  - Advance the retrograde microcatheter into the antegrade guide.

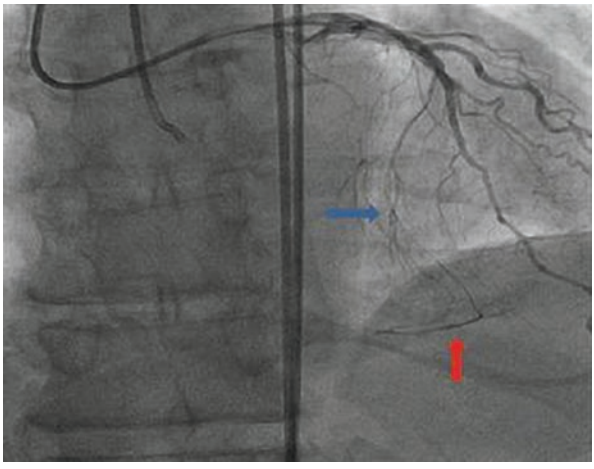

Fig. 19.4 *Red arrow*: Fielder FC wire used for surfing the septal collaterals and seen entering into distal vessel. *Blue arrow*: Corsair catheter

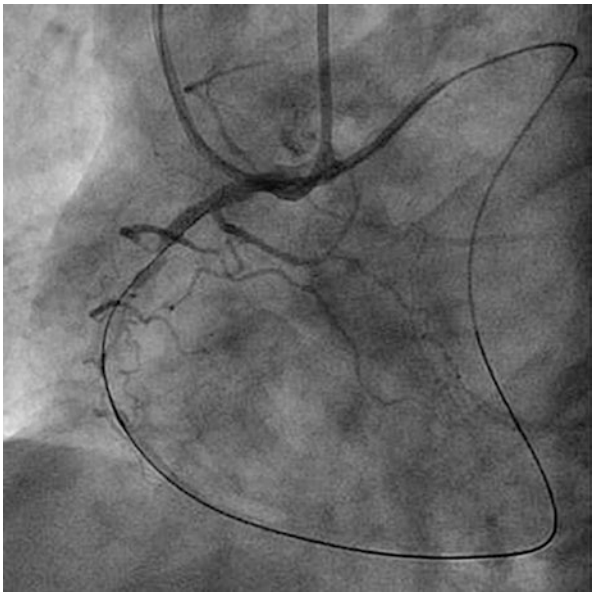

Fig. 19.5 Fielder wire™ escalated to Confianza Pro™ 12. Successful retrograde occlusion crossing

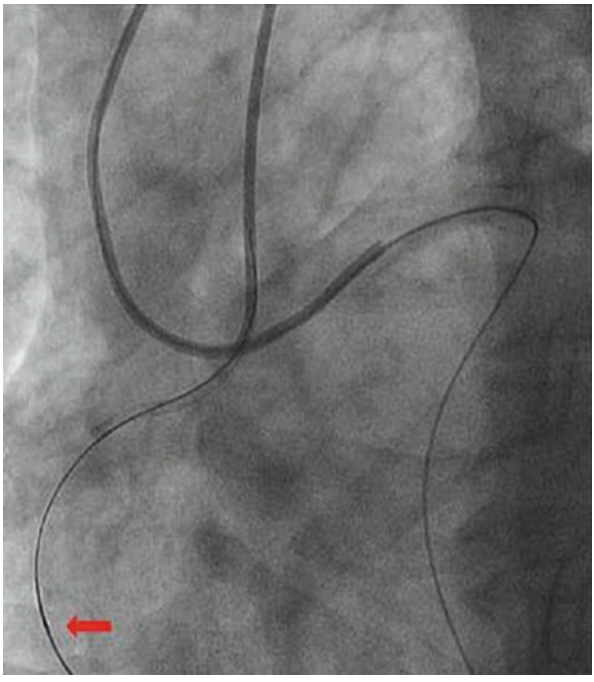

Fig. 19.6 Wire advanced into the antegrade guide catheter. *Red arrow*: Corsair catheter

– Exchange the wire for long, dedicated externalization wire (e.g., RG3, which is 330-cm long; or ViperWire, which is 325-cm long). Remove copilot from the antegrade guide. Insert wire introducer in the copilot. Thread the retrograde wire into the introducer and then attach the co-pilot to the antegrade guide (Fig. 19.8).

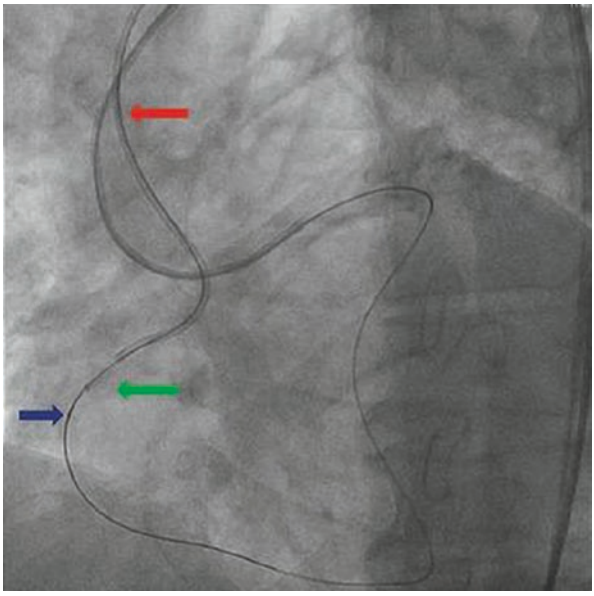

Fig. 19.7 *Red arrow*: 2.5/12 balloon used for trapping the wire in guide. *Green arrow*: Guideliner used as guide extension in difficult cases was Corsair cannot be advanced into the antegrade guide. *Blue arrow*: Corsair catheter

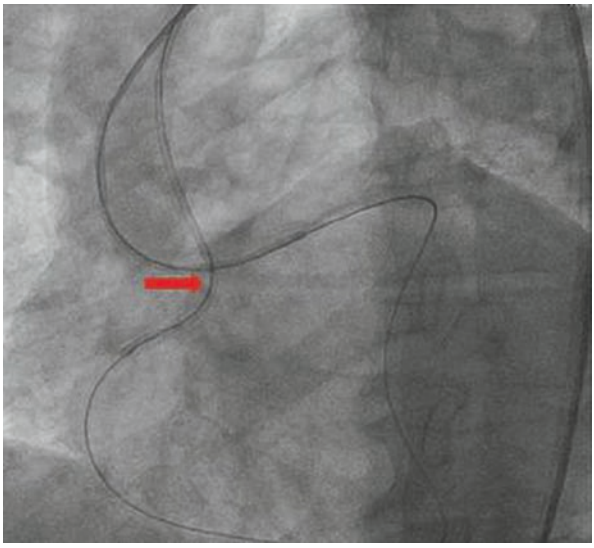

Fig. 19.8 Corsair (*red arrow*) advanced into the antegrade catheter. Retrograde wire removed and exchanged for a 330 cm ViperWire which is externalized from the copilot of antegrade guide

- *Step 3: Alternative to this (conventional) externalization are represented by:*
  - Tip-in: the retrograde wire is advanced into a microcatheter introduced into the antegrade wire, inside the guide catheter. This is more easily achieved in segments of the guide catheter where curves are present (e.g., aortic arch). The retrograde microcatheter is then pulled for a few centimeters, while the antegrade microcatheter is simultaneously advanced. This is repeated several times until the two microcatheters are close to one another (although not touching each other, to prevent entrapment) in the distality of the CTO vessel. At this point, the retrograde guidewire is pulled inside the retrograde microcatheter, and an antegrade workhorse wire is advanced through the antegrade microcatheter, thus achieving antegrade distal true lumen control.
  - *Rendez-vous*: a guidewire is advanced anterogradely into the antegrade guide catheter entering into the retrograde microcatheter (inside the antegrade guide catheter). Then the same process (pushing the antegrade microcatheter while pulling the retrograde microcatheter) detailed for the tip-in technique is performed.
  - *Snaring*: antegrade snaring of the retrograde guidewire should be performed only if the aforementioned three techniques have failed or cannot be performed. This sometimes happens in flush ostial occlusions and/or in coronary arteries with anomalous takeoff. A large snare (Gooseneck or EnSnare) is advanced through the antegrade guide catheter to capture the retrograde wire into the aorta. Then, the snare-retrograde wire system is held tight to facilitate forceful sliding of the antegrade guide catheter into the coronary ostium of the occluded vessel. This technique creates some strain onto the retrograde collaterals, which is why the role of snaring is currently marginal in retrograde CTO PCI.
- *Step 4: Removal of the retrograde microcatheter*:
  - Disengage the retrograde guide. This will prevent ostial dissection when the retrograde microcatheter is pulled out.
  - Pull the retrograde microcatheter proximal to the retrograde channel used, while leaving the externalized/retrograde wire in place. This will allow prompt treatment of a perforation in case one is observed.
  - Re-engage the retrograde guide catheter.
  - Perform a donor vessel angiogram, looking for dissections and thrombus.
  - Re-advance the retrograde microcatheter to the distal CTO vessel, so to protect the retrograde channel during extraction.
  - Remove the retrograde microcatheter-wire system.
  - Retrograde guide can be removed.
- *Step 5: Opening the CTO*:
  - Perform lesion preparation as per standard PCI practice. This might include atherotomy, atherectomy, and other adjunctive technique (e.g., imaging).
  - Stenting.
  - Post-dilatation.
  - Final intravascular imaging to confirm an optimal result is strongly encouraged.
  - Final angiogram to rule out any perforation, dissection, or other complications.

When to Stop a CTO Recanalization Attempt

- AK > 5 Gy, if CTO has not been crossed yet.
- Excessive contrast media volume administered (>3 times contrast volume/eGFR ratio)
- Patient or operator fatigue.
- Major complications (e.g., perforation, tamponade, donor artery dissection/thrombosis, equipment entrapment, etc.).
- Futility: the procedure is unlikely to be completed successfully for any reason (excessive tortuosity/calcification, distal vessel too short/small, etc.) and all available techniques have been utilized.

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Acute Coronary Syndrome: STEMI and Non-STEMI Interventions

20

Amit Hooda and Joseph Sweeny

Introduction

Acute coronary syndrome (ACS) is a spectrum ranging from ST-elevation myocardial infarction (STEMI) to troponin-negative unstable angina. Plaque rupture or erosion leads to thrombus formation resulting in ischemic myocardial damage. Current guidelines recommend primary angioplasty for patients with STEMI and early invasive strategy (<72 h) for patients with high-risk ACS.

Acute Coronary Syndrome Basic Management

Tips for the management of patients with acute coronary syndrome:

- Keep it simple. Avoid bifurcation stenting due to a higher risk of stent thrombosis.
- Ensure the lesion is adequately covered by the stent, use longer stents in thrombotic lesions.
- Treat the infarct-related artery at the time of primary PCI.
- Staged PCI of significant non-culprit lesions should be considered before hospital discharge.
- Minimize contrast use.

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Non-STEMI [1]

Dual Antiplatelet Therapy [2]

- ASA 162–325 mg plus.
- P2Y12 inhibitor (one of the following):
  - Clopidogrel: 600 mg loading dose (300 mg if increased risk of bleeding) followed by 75 mg daily.
  - Prasugrel: 60 mg loading dose, followed by 10 mg daily. Contraindicated in patients with prior CVA, weight < 60 kg, and age > 75 years.
  - Ticagrelor: 180 mg loading dose followed by 90 mg twice daily.
  - IV Cangrelor: consider in cases of cardiogenic shock, inability to take PO, and intubated patients.

Anticoagulation (One of the Following)

- Bivalirudin 0.75 mg/kg and then infusion of 1.75 mg/kg/h. If creatinine clearance is <30 ml/min, bolus dose remains the same, but infusion dose is reduced to 1 mg/kg/h or 0.25 mg/kg/h if on dialysis.
- Unfractionated heparin: Initial loading dose of 60 IU/kg (max 4000 IU) followed by 12 IU/kg infusion up to 1000 IU/h, continued for 48 h or until PCI is performed. Target ACT 300 s.

Adjunctive Medical Therapy

- Nitrates (sublingual/intravenous/transdermal) helps in relieving pain. Contraindicated if systolic BP < 100 mmHg or concerns for right ventricular infarction or recent use of sildenafil, vardenafil (<24 h), or tadalafil (<48 h).
- Beta-blockers (Oral/IV) (within the first 24 h). Contraindicated in patients who are at the risk of developing cardiogenic shock (>70 years of age, heart rate > 110 beats per minute, systolic BP <120 mm Hg, and late presentation).
- High-dose statins (within the first 24 h) for its pleiotropic effect.
- ACE inhibitors (within first 24 h), especially in patients with systolic LV dysfunction with LVEF <40%, and used with caution in patients with relative hypotension.
- Liberal use of vasodilators to avoid slow flow in presence of an obvious thrombotic lesion.
- IC Verapamil 250–500 μg, IC Sodium nitroprusside 50–200 μg.

Drug Eluting Stent (DES)

- Stenting with new generation DES is recommended over BMS.
- New-generation DES has shown superior safety and efficacy, with respect to lower risk of stent thrombosis and target vessel revascularization.

Duration of Dual Antiplatelet Therapy Post PCI

- ASA 81–162 mg daily lifelong.
- Clopidogrel/prasugrel/ticagrelor for 12 months.

STEMI [3]

Dual Antiplatelet Therapy

- ASA 162–325 mg plus one of the P2Y12 inhibitors as noted above (non-STEMI).

Anticoagulation

- Bivalirudin IV bolus and infusion is routinely used for coronary intervention in the cardiac catheterization lab. IV 4000 units of heparin at the first contact in ER may be administered with intermittent doses during the PCI to maintain ACT >300 s.

Vascular Access

- Radial access is recommended over femoral access as this approach has consistently shown a lower risk of access site bleeding, vascular complications, and mortality benefit in this group of patients. In cases of heart block and hemodynamic instability, femoral vascular access is recommended.

Diagnostic Angiogram

- If ST-segment elevation on ECG suggests LCA obstruction:
  - Obtain access, assess opening pressure, and give bolus dose of anticoagulation.
  - Advance JR4 diagnostic catheter into LV to measure LVEDP. IV furosemide if LVEDP >20 mm of Hg is given after finishing PCI.
  - Perform manual injection ventriculogram to assess LV function (limit dye use).
  - Perform single injection of RCA in LAO cranial view (15 LAO, 30 Cranial) and pan for late collateral filling.
  - To evaluate LCA, engage with VL3.5 guide catheter and take AP caudal and AP cranial projections to assess for lesion location.
- If ST-segment elevation on ECG suggests RCA obstruction (Fig. 20.1).
  - If there is any electric instability or conduction delay or bradycardia, consider placing a temporary pacing wire.
  - Take two views of LCA with JL4 catheter (AP caudal and AP cranial).
  - Advance IM/FR4 guide catheter (with side holes) into LV to assess LVEDP.

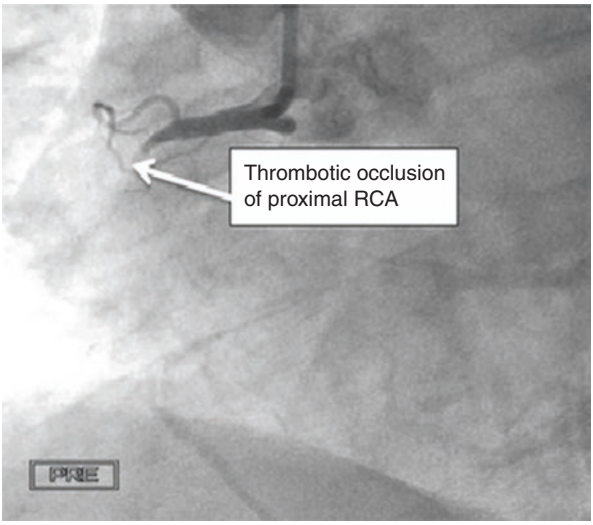

Fig. 20.1 Thrombotic occlusion of proximal RCA

  - Inject RCA using minimal dye.
  - If borderline BP (SBP <100 mm of Hg, HR >90 beats/min, or LVEDP >25 mm of Hg) consider IABP/Impella insertion and IV furosemide at the end of primary PCI.
- If a patient presents with STEMI and signs of cardiogenic shock
  - (SBP <90 mm of Hg, HR > 110 beats/min), consider IABP or Impella placement at the start of the case.
  - Short-term mechanical circulatory support in form of ECMO may be considered as in cases of severe refractory cardiogenic shock (SRCS), characterized by a systolic blood pressure of ≤90 mm Hg, a cardiac index of ≤2.0 l/(min·m^2), and evidence of end-organ failure despite inotrope/vasopressor support, with or without IABP [4].

Coronary Intervention [5]

- Check ACT to ensure adequate anticoagulation (goal >300).
- Use a workhorse wire with a soft tip such as Runthrough™ or BMW Universal™ to cross an occlusive thrombotic lesion to reduce the risk of dissection (Fig. 20.2). Use a 2 × 15 compliant balloon for balloon support if there is difficulty in crossing the lesion.
- Once the lesion is crossed with the wire, advance balloon across the lesion to break up the thrombus (Dottering or Fogarty technique). Confirm the wire position inside the distal vessel. If the distal vessel is not still visualized, consider low-pressure balloon inflation to establish flow inside the vessel.

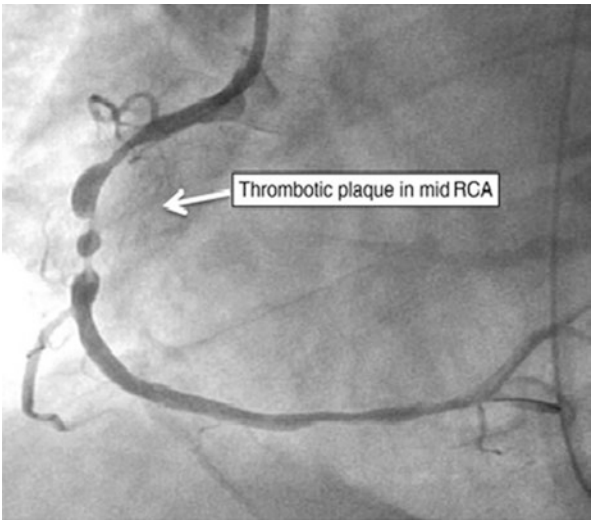

Fig. 20.2 Thrombotic plaque in mid-RCA

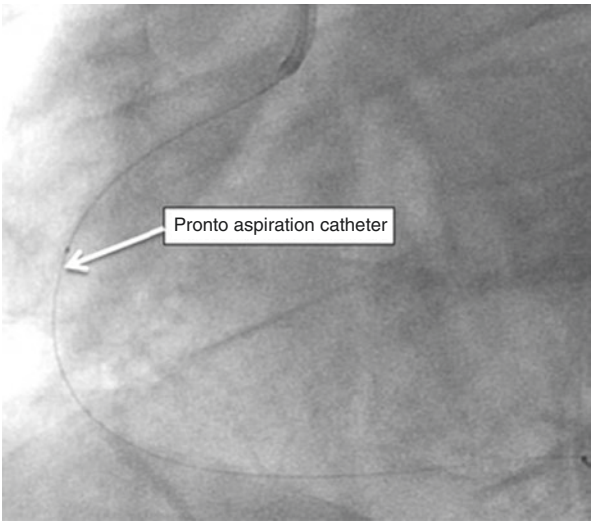

Fig. 20.3 Pronto™ (aspiration catheter) in mid-RCA

- Take a cine angiographic picture to document restoration of distal vessel flow and distal wire position.
- Look for distal embolization.
- Routine aspiration thrombectomy before primary PCI is not recommended. Consider using an aspiration catheter like a Pronto™ or Export™ in selective and bailout situations (Fig. 20.3). In the presence of extensive thrombus burden (grade ≥ to 4) is evident, then mechanical thrombectomy device like AngioJet™ and Penumbra Indigo™ system may be beneficial.
- Stenting: We attempt to direct stent without predilatation (Fig. 20.4).

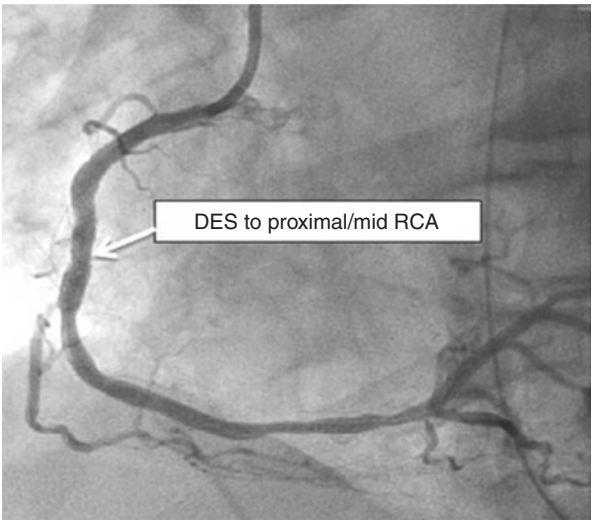

Fig. 20.4 DES deployed in proximal/mid RCA

- Predilatation can lead to distal embolization of the thrombus.
- If the stent is unable to cross the lesion, predilatation should be performed with a small compliant balloon (2–2.5 mm diameter and 12–15 mm length at 8–10 atm).
- For the same reasons, one should try to avoid postdilatation.
  However, if the stent does not fully expand, postdilate the stent with a 1:1 high-pressure balloon at no more than 16 atm.

Treatment of Distal Embolization/Slow Flow/No Reflow

- Medication to treat distal embolization and slow flow/no reflow in an infarct-related artery can be administered through the guide catheter or through a special delivery catheter that can be delivered to the target vessel using 0.014″ wire.
- Twin-Pass Catheter™: Allows the operator to deliver medication through the OTW lumen while leaving the original treatment lumen in place.
  - If Twin-Pass Catheter™ is not available, medication can be delivered through the aspiration lumen of Pronto™ or Export™ catheter. If this catheter has been used for thrombus suction, make sure that the lumen has been flushed thoroughly to remove any residual clots.
- Medication used to treat distal embolization/slow flow
  - Verapamil 250–500 μg bolus, max 2000 μg (vasodilator).
  - Adenosine 10–20 μg bolus (vasodilator), 30–40 μg.
  - Sodium nitroprusside 50–200 μg, max 1000 μg (direct NO donor).

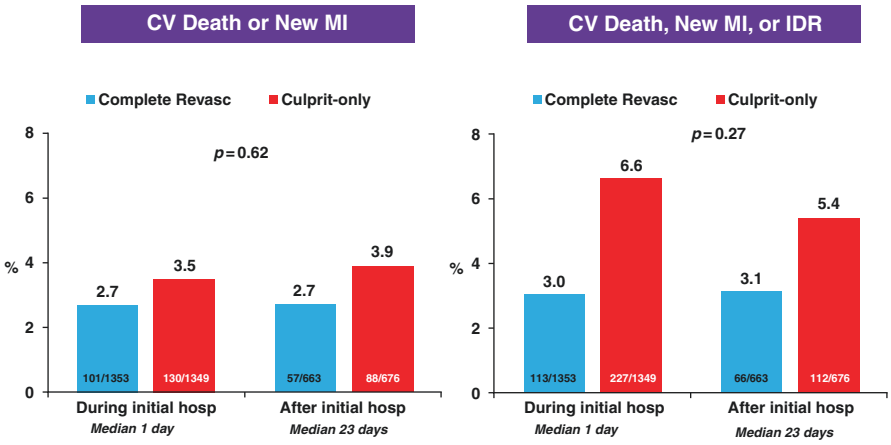

Fig. 20.5 Complete Trial: Timing of Non-Culprit Lesion PCI during or after Index Hospitalization. (Adapted from Mehta et al. [6])

Management of Non-culprit Lesion [5, 6]

- STEMI patients frequently found to have multivessel coronary artery disease, with additional angiographically significant lesions in locations separate from that of the culprit lesion that caused the acute event and recurrent ischemic events are an important predictor of long-term prognosis.
- Based on current available evidence (Class IIb recommendation), PCI of a non-infarct artery may be considered in selected patients with STEMI and multivessel disease who are hemodynamically stable, either at the time of PCI or as a staged procedure, with primary benefit resulting from decreased risk of spontaneous MI. Based on the COMPLETE trial, a second staged PCI to be done during index admission or within 45 days of discharge (Fig. 20.5).

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Coronary Artery Bypass Graft Interventions

21

Hemal Bhatt and Samin K. Sharma

Introduction

The ten-year patency rate after coronary artery bypass graft (CABG) surgery is approximately 90% for internal mammary artery (IMA) and 50% for saphenous vein grafts (SVG) [1, 2]. Redo-CABG is associated with reduced graft patency rates, incomplete symptom relief, and higher mortality compared with the first CABG [2, 3]. PCI of the native vessel is preferred over graft vessel PCI in symptomatic patients [4].

SVG Interventions

Basics

- Majority of randomized control trials favor using drug eluting stents compared with bare metal stents in SVG interventions [5–8].
- Successful revascularization is less likely in grafts >3 years old and aorto-ostial lesions which are more prone to distal embolization.
- Chronic total occlusion (CTO) PCI of SVGs in a symptomatic patient should not be considered unless there is an antegrade channel present with no possibility of native vessel PCI (class III ACC/AHA recommendation) [4, 9, 10].
- 2011 ACC/AHA coronary intervention guidelines recommend using embolic protection devices (EPD) (Class I indication) in SVG interventions; however, the recent data does not support the routine use of EPDs in SVG interventions, except in high-risk anatomy such as severely degenerated or thrombotic grafts [11–13].

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Equipment

- Sheath: 6 Fr long sheath.
- Guide catheter:
- For left-sided bypass grafts—AR2, IM, AL 0.75, AL1, or AL 2.
- For right-sided bypass grafts—MP1, AR2, AL 0.75, or AL 1.
- Wires: Runthrough or FilterWire for SVGs if technically feasible.
- Balloon/stent: Size the balloons and stents no larger than the reference diameter. Balloon catheters with extra-long (145 cm) shafts may be required.

Access

- Femoral or radial.

Fluoroscopic Views

- Initial view is straight LAO for all bypass grafts.
- Second view depends on which segment of the native vessel the graft is anastomosed.
  - Graft to LCX or OM: *RAO caudal or AP caudal*.
  - Graft to LAD or diagonal: *RAO cranial or AP cranial*.
  - Graft to RPDA: *LAO cranial or AP cranial*.

Steps by Step Approach

- Coaxial engagement.
- Intra-graft verapamil, nitroprusside, nicardipine, or adenosine to prevent no-reflow phenomenon.
- Wire the lesion with 0.014″ wire.
- EPDs could be considered in some cases if the distal embolization risk is high. Assess the distal landing zone to decide on the type of EPD
- Primary stenting is preferred for all SVG interventions if possible to minimize distal embolization.
- If difficulty is encountered in advancing the stent across the lesion, then perform the following steps.
  - Advance the stent while the patient performs deep breathing maneuvers.
  - Use a small 2.0–2.5/12–15 mm compliant balloon to predilate the lesion
  - Use the buddy wire technique (If EPD is used, then ensure that the stent is deployed over the EPD wire) and ensure that the buddy wire is removed prior to stent deployment.
  - If all the above techniques fail, consider using a Guideliner with extreme caution over the EPD wire.

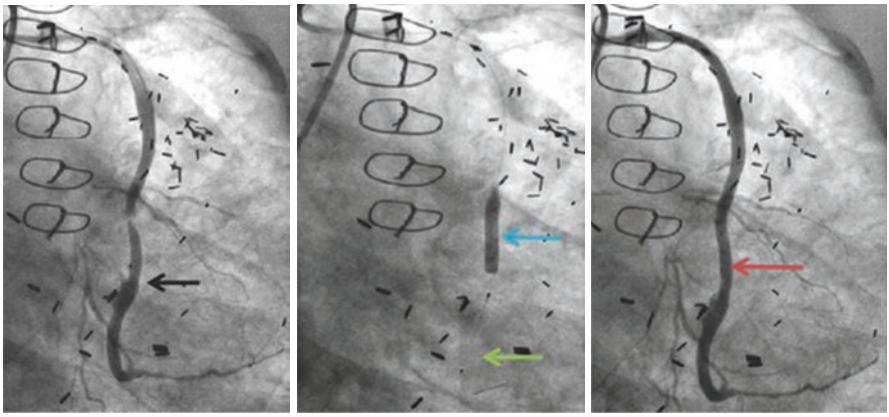

Fig. 21.1 SVG to LPL intervention. Black arrow: 90% stenosis at mid SVG to LPL, green arrow: Spider Filter. Blue arrow: angioplasty with a 3.0/18 noncompliant balloon, red arrow: deployment of DES [3.5/20]

- Never oversize the balloon or stent.
- Stent from "normal" segment to "normal" segment (Fig. 21.1).
- Avoid high pressure (>12–14 atm) or postdilation.

Complications

- No-reflow: etiology is complex but mainly caused by distal embolization of thrombotic particles. Delivery of nitroprusside or verapamil to the distal vessels using a Twinpass micro-catheter or EPD use may help prevent this phenomenon.
- Perforation: prevention of perforation by ensuring guidewire is intraluminal, not inflating a balloon in a subintimal location, and avoiding overexpansion of the graft.

Arterial Graft Interventions

Basics

- LIMA patency rates are high, around 90–95% at 10 years [1, 2].
- PCI of the LIMA or through the LIMA has high success rates.
- There is a low incidence of abrupt closure, distal embolization, acute myocardial infarction, or emergency CABG surgery.
- Usually, failure of PCI is due to inherent technical challenges because of the inability to navigate tortuous lesions.

Equipment

- Sheath: 6 Fr Long sheath.
- Guide catheter: use shorter guides to access distal lesions through long tortuous grafts.
  - LIMA: IM 90 cm (Fig. 21.2).
  - RIMA: 3DRC or no-torque (Fig. 21.3).
  - Free radial: usually AR2 for left-sided grafts and MP1 for right-sided grafts will suffice.
  - Gastroepiploic: engage with cobra diagnostic catheter and wire the lesion with a 300 cm wire and exchange for FR guide ± use of a GuideLiner (Fig. 21.4).

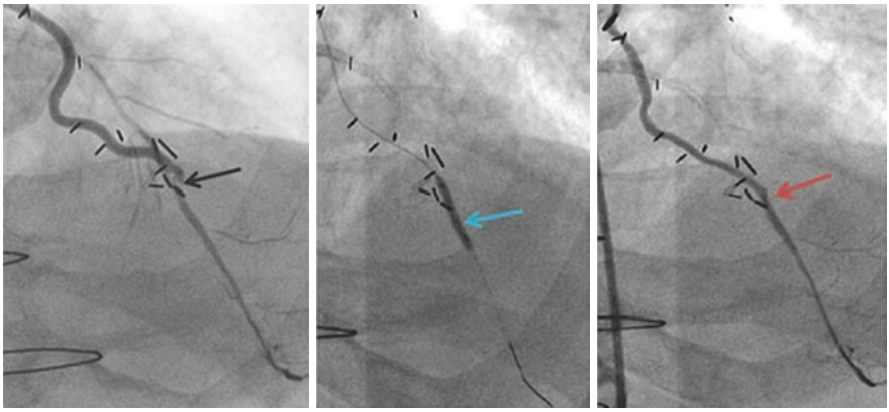

Fig. 21.2 LIMA intervention. Black arrow: 90% stenosis at site of LIMA anastomosis to distal LAD. Blue arrow: angioplasty with 2.0/15 noncompliant balloon. Red arrow: deployment of DES [2.5/18]

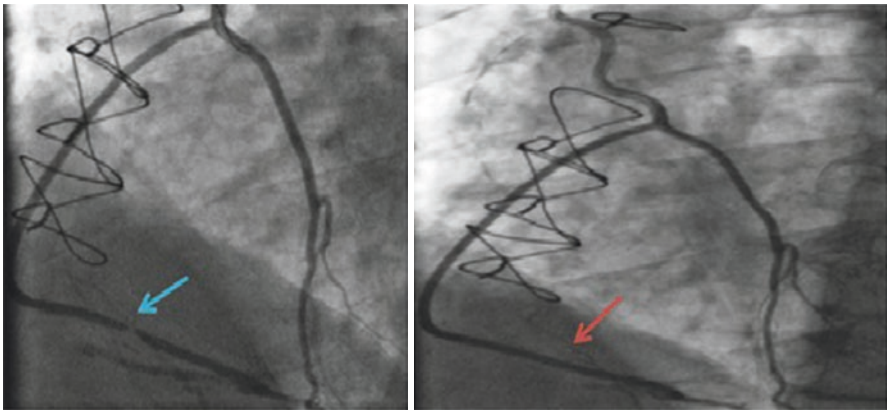

Fig. 21.3 RIMA graft intervention. Blue arrow: 90% stenosis RPDA limb of the RIMA Y graft. Red arrow: post direct DES stent deployment [3.0/15]

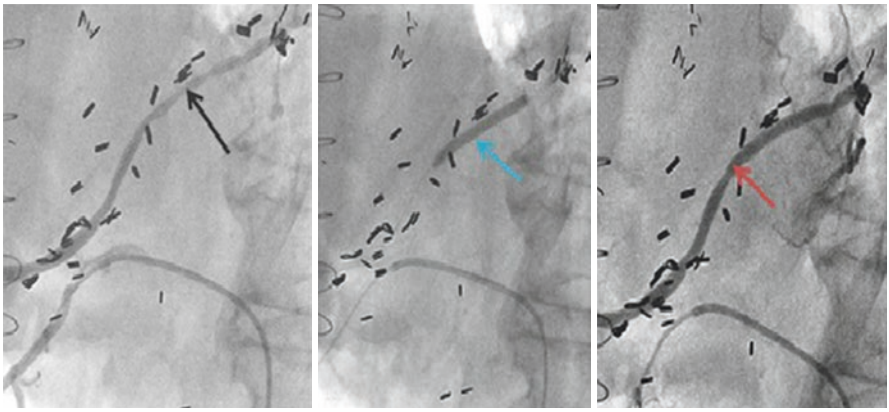

Fig. 21.4 Gastroepiploic graft intervention. Black arrow: 70% stenosis of mid gastroepiploic graft to distal LPL. Blue arrow: direct stenting with DES [3.5/33]. Red arrow: post stent deployment

- Wires:
  - Fielder or other hydrophilic and nitinol core guidewires are more flexible and unlikely to kink or straighten the IMA and less likely to cause pseudostenosis. In rare cases, extra support guidewires may be required.
- Balloon:
  - Size the balloons and stents no larger than the reference diameter.
  - Use longer shaft balloons and stents if needed.

Access

- LIMA: femoral/left radial for LIMA.
- RIMA: femoral/right radial for RIMA.

Fluoroscopic Views

- Initial view for engaging: AP straight.
- Second view depends on which segment of the native vessel the graft is anastomosed.
  - Ostium of LIMA: *AP straight or a 45° to 60° RAO or LOA.*
  - Anastomotic site of LIMA: *AP cranial, RAO straight and rarely 90° LAO.*
  - Anastomotic site of RIMA: *depends on artery bypassed.*

Step-by-Step Approach

- Engaging may be difficult. The key is to avoid dissection, as this may be disastrous especially if IMA is the primary vessel perfusing the heart.
- Watch out for spasm when engaging the vessel. Give intracoronary nitroglycerin or verapamil on engagement of the IMA.
- Avoid deep guide intubation of the IMA.
- Always monitor the pressure tracing closely.
- Hand inject contrast gently into the IMA to confirm the position of the guide and wire the lesion with a hydrophilic 0.014″ wire.
- If you have difficulty engaging LIMA:
  - Rule out groin tortuosity. Use a long sheath for access.
  - Use straight 45° to 90° projection to elongate the arch as it helps engage in the presence of a tortuous subclavian artery.
  - Wire the IMA using a hydrophilic 0.014″ wire with the guide close to the ostium and rail the guide into the IMA ostium with a transit catheter or balloon support (2.0/12 mm) the wire.
  - Use a diagnostic catheter to engage the IMA and exchange for a guide catheter over a long 300 cm 0.014″ wire.
- Lower threshold to use a GuideLiner for support, microcatheters, and movable and directional tip catheters (Venture or Supercross) to help wiring and stenting.

Complications

- Dissection or perforation of IMA:
  - Always have ready access to a covered stent and surgical backup.
- Spasm:
  - IMA is prone to spasm, which can be treated with intra-arterial nitroglycerin or calcium channel blockers (diltiazem or verapamil) (Fig. 21.5) [14].

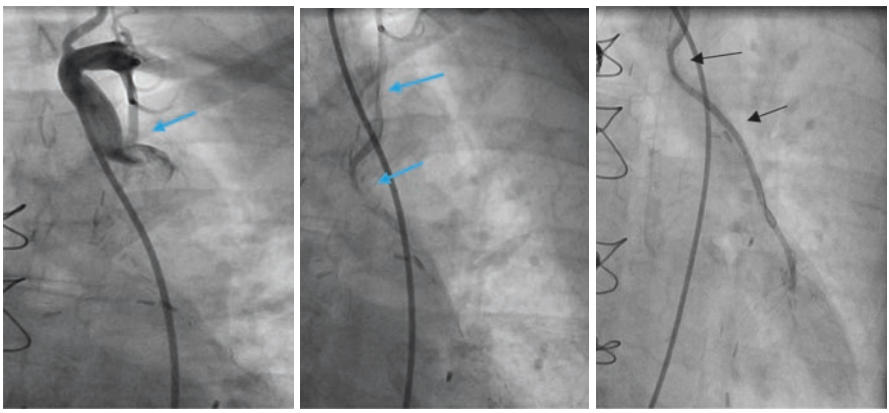

Fig. 21.5 Evidence of spasm in the proximal and mid to distal portion of left IMA (blue arrows). Resolution of spasm with intra-arterial nitroglycerin resulting in passage of coronary guidewire safely (black arrows)

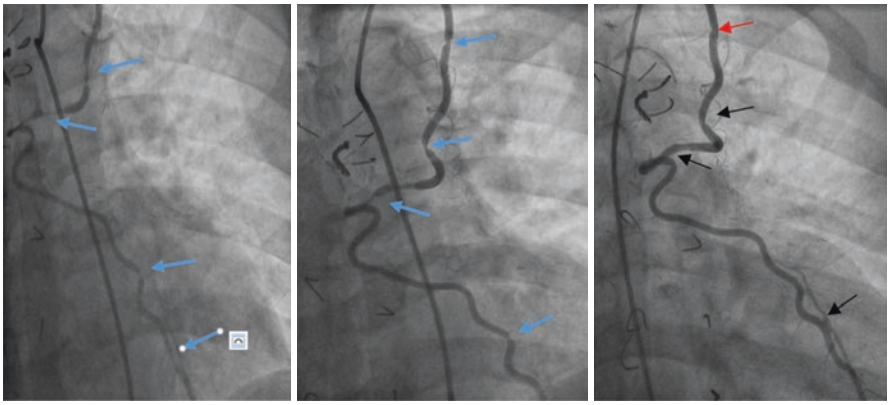

Fig. 21.6 Evidence of pseudostenosis after placement of stent in left IMA (blue arrows), relieved by pulling back the guidewire (black arrows). The guideliner (red arrow) was used to selectively engage IMA to achieve adequate contrast opacification

- Pseudostenosis (or accordion phenomenon):
  - Occurs due to straightening of highly tortuous vessels due to stiff guidewire leading to angiographically appearing constrictive defects of the vessel. Pseudostenosis could mimic an actual dissection, spasm, or closure of the IMA vessel. In such cases, pull the wire back so that the soft flexible portion of the wire is across the area of constrictive defects (Fig. 21.6) [15, 16]. The soft distal portion of the wire will conform to the tortuosity and eliminate pseudostenosis of these vessels, and will allow access to the vessel if it is a dissection or acute closure of the vessel.
- Immediate post-CABG ischemic symptoms:
  - Class I guidelines for PCI—Make sure that the wire is intracoronary and size the balloon 0.5:1 to avoid disruption of sutures and postoperative fatal bleeding.

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Severely Calcific Coronary Artery Lesion Interventions

22

Raman Sharma and Samin K. Sharma

Introduction

Calcific coronary lesions have been associated with multiple complications, including poor response to balloon angioplasty, dissection during PTCA/predilatation, difficulty in stent delivery and inadequate stent expansion [1–4]. This complexity associated with severely calcific lesion intervention has been associated with increase in-hospital (12.3% vs 1.5%) and 1-year MACE (24.2% vs. 5.4%) when compared to lesions with no/mild calcium [5]. Optimal stent delivery and apposition require adequate preparation and modification with super-high pressure noncompliant balloons, cutting balloon atherotomy or atherectomy devices.

Super-High Pressure Noncompliant Balloon

Conventional noncompliant balloon dilation confers a lower risk of the "dog-boning" effect seen with semi-compliant balloons with non-uniform balloon expansion and preferential over expansion proximal and distal to the lesion, together, which increase edge dissection coronary perforation [6, 7]. Noncompliant balloons, however, are limited in their ability to optimally modify plaque given the 20–22 atm inflation limitation.

The OPN NC Super-High Pressure Balloon (SIS Medical AG, Winterthur Switzerland) is a new rapid exchange PTCA catheter compatible with a 0.014" coronary wire. This twin-layer balloon construction is capable of very high pressure,

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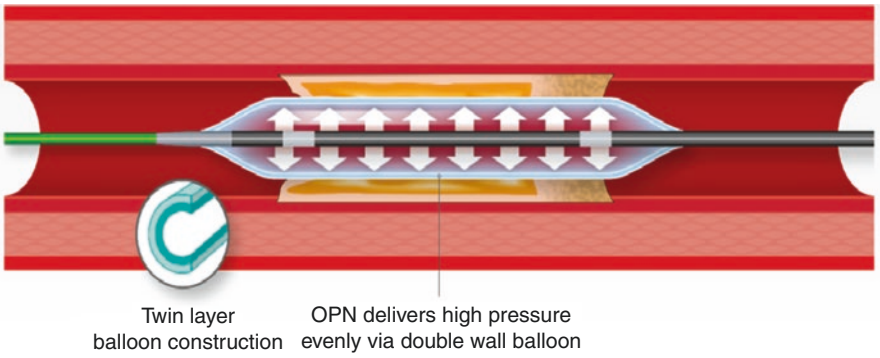

Fig. 22.1 OPN NC Super-high pressure balloon illustration of twin-layer balloon construction. Used with permission from SIS Medical AG

uniform inflations, with a rated burst pressure of 35 Atm and tested at >40 Atm (Fig. 22.1). These balloons are currently available outside the USA in diameters from 1.5 to 4.0 mm and lengths of 10, 15, and 20 mm.

Secco et al. demonstrated the use of the OPN balloon in severely calcific lesions unresponsive to conventional high pressure balloon dilatation, achieving adequate expansion of the lesion with OPN PTCA alone in 90.5% of cases and 46.9% of cases requiring >40 Atm for adequate lesion treatment [8].

Atherotomy: Cutting Balloon

Flextome™

Boston Scientific's Flextome Cutting Balloon™ Dilatation Device, which includes three (≤3 mm) or four (>3 mm) microsurgical blades (atherotomes) mounted lengthwise along the outer surface of a noncompliant balloon (Fig. 22.2). When inflated, these blades create intentional dissections within the plaque and intima without medial injury as seen with conventional noncompliant balloon PTCA, thereby limiting gross endothelial injury while maximizing lesion modification for subsequent balloon dilatation (Fig. 22.3). The Flextome™ device also has flex points every 5 mm allowing for more deliverability in increasingly tortuous vessels.

Wolverine™

Constructed very similarly to Flextome, Boston Scientific's Wolverine™ has essentially the same structure but with a smaller profile with a reduced T-slot height, allowing for improved deliverability and crossability (Fig. 22.4).

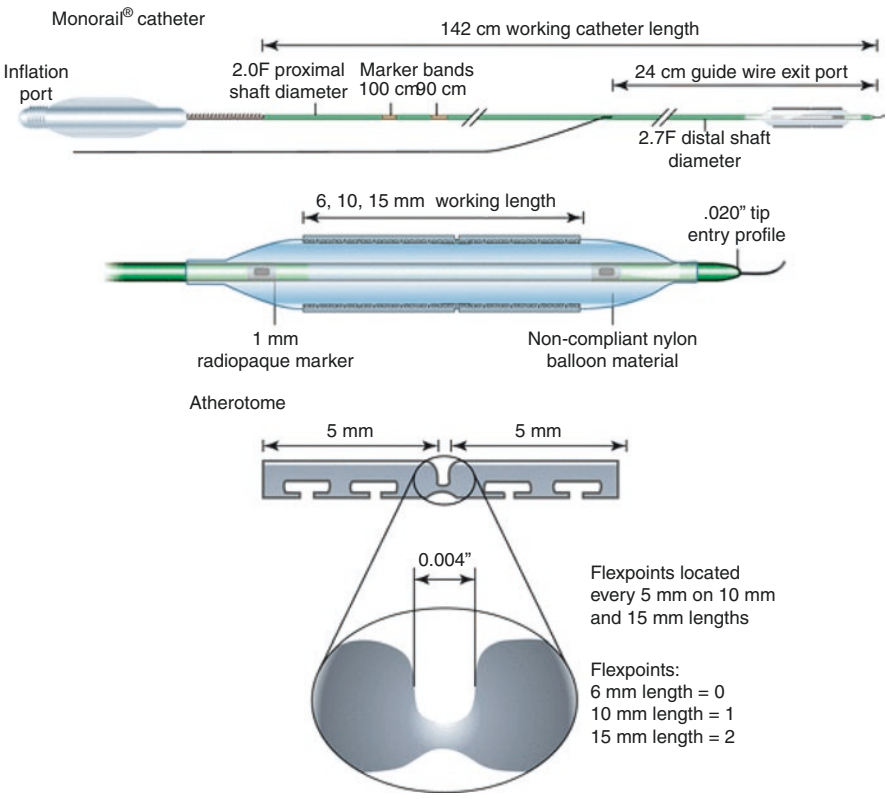

Fig. 22.2 Flextome ™ cutting balloon: blades mounted on a noncompliant balloon. Flex points are located every 5 mm on the balloon (© 2019 Boston Scientific Corporation or its affiliates. All rights reserved. Used with permission of Boston Scientific Corporation)

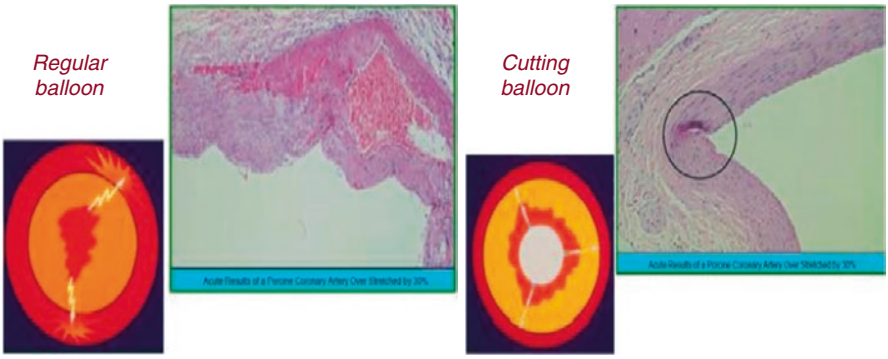

Fig. 22.3 Acute histopathologic features of porcine coronary artery over stretched by 30% by regular balloon vs. cutting balloon. Regular balloon inflation resulting in increased medial injury, increased arterial wall and balloon surface area contact resulting in endothelial injury and hematoma formation. Cutting balloon inflation resulting in localized scoring sites with decreased arterial wall/balloon surface area contact with less endothelial and medial injury

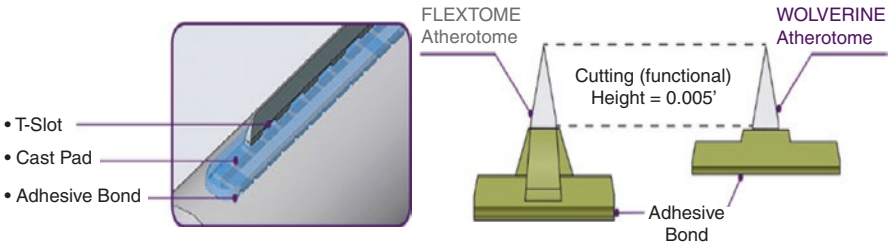

Fig. 22.4 Wolverine cutting balloon™ with markedly reduced profile to enhance deliverability and crossability (© 2019 Boston Scientific Corporation or its affiliates. All rights reserved. Used with permission of Boston Scientific Corporation)

Technical Considerations

- Cutting balloons create intentional dissections as a mechanism to prepare lesions for optimal stent delivery. Oversizing the device has expectedly demonstrated an increase in risk of perforation. To reduce the risk of this potential complication, the diameter should be selected to a ratio slightly less than 1:1 of the diameter of the vessel just proximal and distal to the stenosis. In ISR cases, cutting balloon size can be 0.25–0.5 mm greater than the stent size. Maximal pressure inflation should be limited to 8–12 atm. Cutting balloon inflation should be gradual and should be passed through the lesion 2–3 times after. The cutting balloon should be pulled back into the guiding catheter, which acts to reshape the atherotomy blades, and then delivered to the lesion again for another inflation in a difference axis of the vessel lumen.
- When treating lesions distal to a stent, extreme care should be practiced in the guidewire wiring. If the guidewire passes through a stent cell as opposed to down the axis of the stent, a deflated Flextome device will become entangled within the stent struts. Additionally, when treating bifurcation lesions, the Flextome can be used to pretreat lesions prior to stent deployment, but should not be taken through the stent struts to treat side branch lesions.

Atherotomy: Scoring Balloon

AngioSculpt™

This scoring balloon produced by Phillips is constructed differently than the Flextome of Boston Scientific. AngioSculpt RX has three flexible nitinol scoring elements arranged helically along a noncompliant balloon (Fig. 22.5). With a low crossing profile of approximately 2.7 F, this scoring balloon allows for large luminal expansion for optimal stent implantation with permissible inflation pressures up to 20 Atm.

Fig. 22.5 AngioSculpt scoring balloon™ (©AngioScore, A Spectranetics Company. All Rights Reserved. Used with permission of Cardiovascular Systems, Inc.)

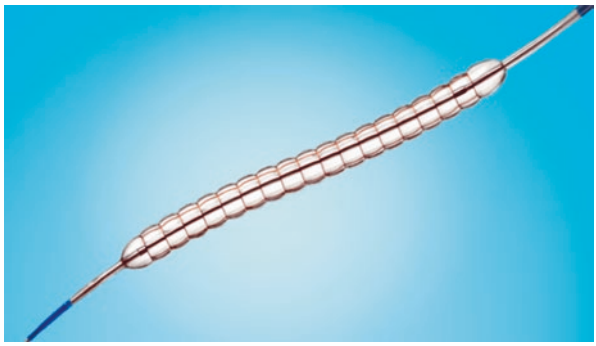

Fig. 22.6 Chocolate XD® PTCA balloon catheter (© Teleflex, All Rights Reserved, Used with permission of Teleflex, Inc.)

Atherotomy: Constrained Semi-compliant Balloon Angioplasty

The Chocolate XD® PTCA Balloon Catheter (© TriReme Medical, All Rights Reserved), is a specialty angioplasty balloon that may be used to pretreat heavily calcific lesions prior to stent delivery (Fig. 22.6). The balloon's design is unique with a proprietary nitinol constraining structure that create "pillows" and "grooves" that are engineered to result in predictable, uniform, and atraumatic lesion dilatation. The "pillows" provide vessel dilatation without cutting or scoring and reduce shear stress by balloon inflation. The plaque channeling "grooves" allow for stress relief zones further reducing the traumatic angioplasty effects. This device comes in diameters from 2.0 to 3.5 mm with a nominal pressure of 10 atm for all sizes.

Rotational Atherectomy

Rotational atherectomy (RA) from Boston Scientific's RotaPro™ is a system consisting of a high-speed rotating diamond-coated burr that behaves as an atheroablative surface on calcific plaque as well as in-stent restenosis [9, 10]. The elliptical

metallic burr is available in sizes ranging from 1.25 to 2.5 mm that provides differential ablation of fibrocalcific plaque over compliant elastic vessel tissue. The resulting ablated particles are pulverized down to 5–10 μm in size, which are presumed to be mechanism of the transient slow/no reflow phenomenon following RA seen in up to 24% in a recent randomized clinical trial [11]. The burr itself only has the diamond-coated abrasive surface on the distal edge and a smooth proximal surface, which is a critical fundamental principle in the rotational atherectomy technique High-speed revolution (140,000–160,000 RPM) minimizes friction with the vessel facilitating burr advancement, especially through tortuous vessels. Rotaglide is a lipid-based emulsion designed specifically for the RotaPro system to reduce friction and heat, improve tactile feedback, and limit sudden decreases in RPM due to lesion contact.

The classical RotaBlator rotational atherectomy is the original atherectomy system and is worthy to mention the notable differences (Fig. 22.7). The RotaBlator atherectomy system is operated by a foot pedal that controls the atheroablation as well as a separate knob that can activate/deactivate Dynaglide. In the RotaPro system, the foot pedal has been replaced with all the control functions of atheroablation and Dynaglide being added to the advancer device. The burrs, guidewire, and technique are entirely the same between the two, with a few exceptions in the preparation and steps.

The RotaPro burr is attached to a 135 cm long sheath with an outer diameter of 4.3 Fr, over a dedicate Rotawire, either Rotawire Floppy or Rotawire Extra Support

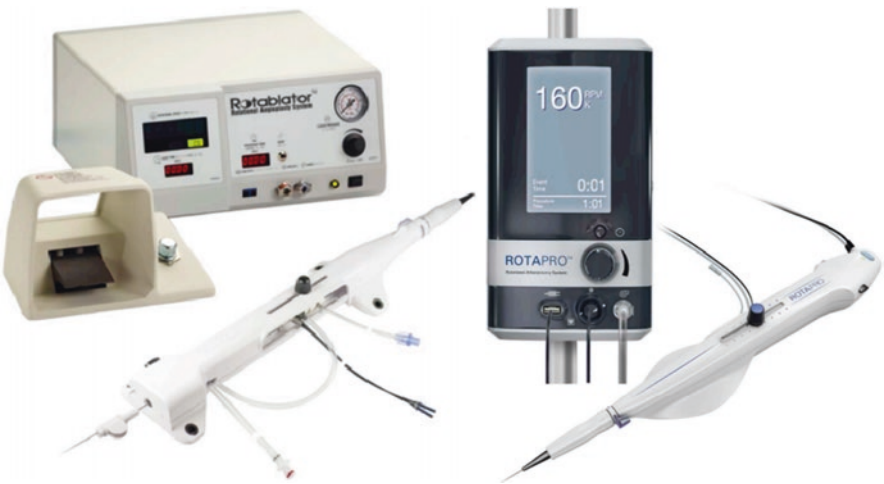

Fig. 22.7 RotaBlator rotational atherectomy system foot pedal, console, and advancer (left). RotaPro rotational atherectomy system, digital console with no foot pedal and Dynaglide mode control on advancer (right) (© 2019 Boston Scientific Corporation or its affiliates. All rights reserved. Used with permission of Boston Scientific Corporation)

depending on procedural requirements (Fig. 22.8). Both RotaWire guidewires are 0.009" stainless steel core with tapered distal ends and 0.014" distal platinum coil preventing the burr from traveling beyond the wire tip. The Extra Support wire is more supportive, with a shorter taper (5 cm) and a longer spring tip (2.8 cm). The Floppy wire is more flexible, with a longer taper (13 cm) and a shorter spring tip (2.2 cm) (Fig. 22.9). The Extra Support guidewire results in more vessel straightening, wire bias and preferential atheroablation on the lesser curvature of angulated segments. This design makes the Extra Support wire ideal for aorto-ostial lesions,

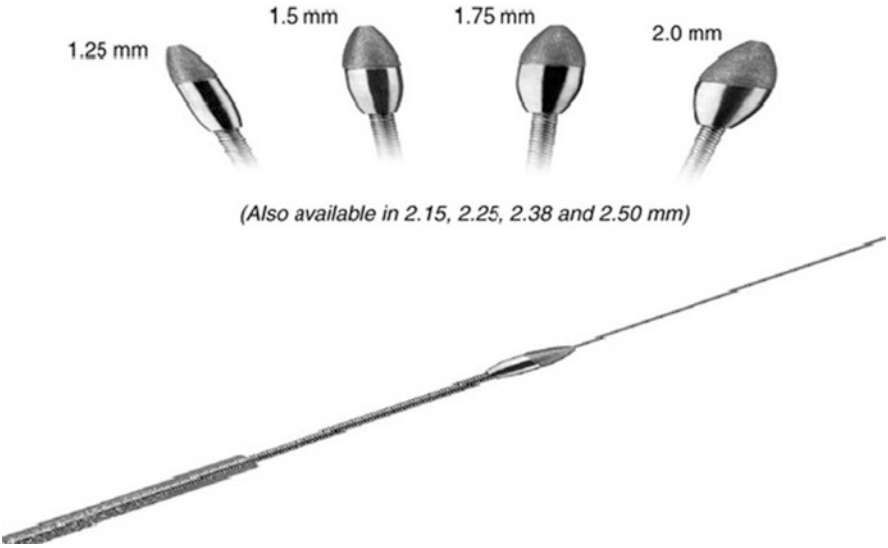

Fig. 22.8 RotaPro™ atherectomy burrs (© 2019 Boston Scientific Corporation or its affiliates. All rights reserved. Used with permission of Boston Scientific Corporation)

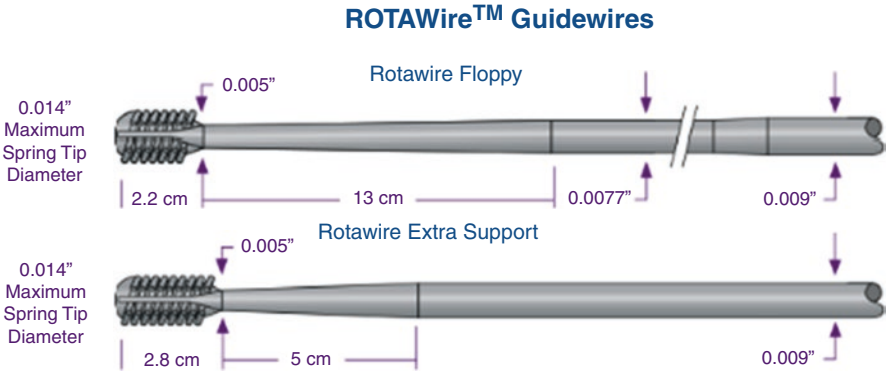

Fig. 22.9 RotaWire™ guidewires (© 2019 Boston Scientific Corporation or its affiliates. All rights reserved. Used with permission of Boston Scientific Corporation)

distal lesions, and in lesions where the calcific burden is primarily on the lesser curvature of angulated segments. The Floppy guidewire, on the other hand, causes less vessel straightening and wire bias, and preferential atheroablation of the greater curvature of angulated segments [12]. The RotaWire Floppy is suitable for most cases, but consideration should be taken to select the Extra Support wire in the appropriate cases to optimize rotational atherectomy success.

Rotational Atherectomy Steps

- Patient Preparation:
  1. Evaluate for egg allergy (RotaGlide contains egg).
  2. Evaluate if patient is adequately hydrated and is able to receive vasodilators.
  3. Consider temporary pacemaker for RCA or dominant LCx intervention cases.
  4. Dual antiplatelet therapy on board.
  5. Anticoagulation with bivalirudin or heparin.
- Console Preparation
  1. Connect the Rotaflush fluid (RotaGlide + 5000 units heparin + 5 mg nitroglycerin + 5 mg verapamil in 1 L normal saline) with a three-way stopcock to the RotaPro device.
  2. Obtain a nitrogen compressor tank and cylinder regulator capable of delivering at least 140 L/min at 90–110 psi. A psi of 500 is the minimum acceptable range to begin a procedure and >750 psi is preferred to ensure the tank will not run out of compressed nitrogen during the case.
  3. Connect the supply hose gas coupling to the outlet port of the cylinder regulator and the other end to the inlet connector on the back of the console. Connect the power cord. Open the compress gas cylinder valve to supply compressed gas to the console.
  4. Connect fiberoptic cable, advancer electrical cable and gasline to front of the console.
  5. Turn the console on. The front console has a display for a run time, tachometer, and a knob to control the RotaPro burring speed.
  6. If the RotaBlator system is being used, the foot pedal connection, gas connection, and fiberoptic cable are all connected to the back of the console.
- Equipment
  1. Burr size should be roughly 0.5:1 of the target vessel.
  2. A 6 F guide catheter system is capable of delivering a burr ≤1.75 mm, a 7 F for a 2.0 mm burr, 8 F for a 2.15 mm burr, and 9 F for 2.25 mm–2.38 mm burr and a 10 F for a 2.5 mm burr.
  3. Choose the appropriate Rotawire based on anatomic and lesion specific considerations.
  4. Choose and prepare a noncompliant balloon that is 1:1 to the target vessel.

5. Keep the packaged guidewire clip either at primary operator area to be used as a torquer device or at the back end by the Rotapro device where it must be clipped prior to starting the procedure to act as a second brake.
6. Carefully remove the distal tip of the desired Rotawire, making four large loops while wiping with wet gauze and set aside. Take great care to not create bends or kinks in this wire.

- Initial Procedure Steps
  1. Direct wiring of the lesion with a Rotawire is reasonable in certain cases, but the majority an exchange wiring will be necessary with the use of workhorse wire and microcatheter.
  2. Turn on Rotaflush solution and backload, advance the burr over the guidewire to the copilot, and immediately place the guidewire clip at the guidewire end that has exited from the back end of the Rotapro device. Confirm verbally
  3. Verbally request a speed check, ensuring the burr and guidewire are held in the air to prevent entanglement.
  4. Verbally countdown to advancing the burr over the guidewire through the copilot and towards the guide catheter tip while on fluoroscopy. The second operator should be pulling the guidewire to ensure the wire is not migrating distally and the guide is not moving aggressively in and out of the ostium. If the guidewire is advancing forward or the guide is moving aggressively, the second operate should pull the wire faster.
  5. Stop when the ostium of the artery has been reached.
- Removing Tension and Inertia from the System.
  1. Unlock advancer knob and move back and forth.
  2. Open copilot and move sheath back and forth.
  3. Active DynaGlide by pressing the Dynaglide button on the device, which turn on a solid green light. Briefly tap on the activator button. Deactivate Dynaglide by pressing the Dynaglide button again.
  4. If using the RotaBlator system, Dynaglide control is located on the foot pedal and is to be performed similarly. Dynaglide is activated by pressing the knob (Dynaglide light on console turns green) and press on the foot pedal to Dynaglide, under fluoroscopy. Deactivate Dynaglide by pressing the knob again.
- Rotational Atherectomy Steps
  1. Advance the burr slowly with a to- and fro-pecking motion.
  2. Use short burr times, roughly 20 s, speeds ranging from 140,000 to 160,000 RPM, avoiding drops in RPM >5,000 for >5 sec, and pause between runs.
  3. Keep SBP >100 bpm and stopping atherectomy temporarily in this setting to administer continuous fluids and IV doses of Neo-synephrine. Monitor for bradycardia.
  4. Once lesion has been crossed, a final polishing run should be performed, which should be smooth and without resistance.

5. To remove the burr, activate Dynaglide mode again. The second operator should be advancing the guidewire from the back end of the device while the first operator is removing the burr.
6. Remove the clip and turn off the Rotaflush.
7. Take a cine image to rule out any complications.
8. Advance a workhorse wire to across the lesion parallel to the Rotawire and perform PTCA with previously prepared noncompliant balloon.
9. Deliver the appropriately sized stent to the lesion, remove the Rota wire, and deploy the stent.
10. If using the RotaBlator system, still remove under Dynaglide, activating appropriately using the foot pedal.

Complications of Rotational Atherectomy

- Transient heart block (pacemaker if needed).
- Wire bias.
- Dissections (Complication chapter).
- Perforations (Complication chapter).
- Slow flow/no reflow (Complication chapter).
- Rota burr entrapment (See below).
  - The presence of the diamond-coated abrasive surface on the front, but not the back allows for the feared complication of lodging the burr within a lesion and become entrapped. During high-speed rotation, the heat may enlarge the space between plaque and since the coefficient of friction during motion is lower than that at rest, the burr may pass the calcific lesion easily without significant plaque debulking, allowing it to get stuck on the way back, called the "kokesi" phenomenon.
  - The best way to prevent burr entrapment is to employ careful and conservative technique throughout the entire rotational atherectomy procedure, especially diligent relief of system tension/inertia prior to atherectomy.
  - In the event of burr entrapment, take advantage of the 0.014"spring tip and pull it to dislodge the burr. If this fails advance the burr at 200,000 RPM into the distal lumen and attempt burr removal while at high-speed revolutions. Next steps include obtaining a second arterial puncture for a second guide catheter or upsize the guide catheter over the RotaPro system, which requires cutting the sheath, driveshaft and Rotawire proximal to the advancing device. A hydrophilic wire should be advanced distal to the trapped burr and balloon dilatation should be performed proximal to the burr to facilitate burr removal. A deep guide catheter intubation or child-in-mother catheter coronary intubation with subsequent pullback of all devices can be alternative method. By simultaneous traction on the burr shaft and countertraction on the child catheter, the catheter tip can serve as a wedge between the burr and entrapping plaque, allowing a large and direct retracting force to retrieve the burr. If all above techniques fail, the patient needs to be referred to CT surgery for

surgical removal of the entrapped burr. For more details, refer to chapter on Complications and Management of Percutaneous Coronary Interventions.

Orbital Atherectomy

Diamondback 360 Coronary Orbital Atherectomy System by Cardiovascular System Inc. (CSI, St. Paul, MN) has been recently added to the market as another successful atherectomy device for severely calcific lesions (Fig. 22.10). Orbital atherectomy combines the use of centrifugal force and differential sanding to modify calcified lesions. Unlike the RotaPro rotational atherectomy system, the orbital atherectomy device uses an eccentrically mounted, diamond-coated crown that orbits over specialized guidewires, ViperWire Advance and ViperWire Advance with Flex Tip (both 0.012”), at high speeds while minimizing trauma to healthy tissue that can flex away from the orbiting crown. As treatment proceeds and multiple runs are performed, both forward and backwards, the crown’s orbital diameter expands radially via centrifugal force, generating a channel 1.25–1.75x greater than the crown size. The main advantage observed of orbital atherectomy over rotational atherectomy is the generation of particles less than 2 μm, compared to the 5–10 μm generated by rotational atherectomy, resulting in less slow flow/no reflow. Additionally, the elliptical orbit as opposed to a concentric burr allows for antegrade blood flow, decreasing potential thermal injury to the vessel, and microdebris to freely flow distally for absorption by the reticuloendothelial system.

The ViperWire Advance has a stainless steel core with higher tip load than the ViperWire Advance with Flex Tip (1.4 g vs 1.0 g). The ViperWire Advance with Flex Tip has a nitinol core, which is more flexible allowing it to reduce wire bias and less straightening of angulated segments. The stainless steel support coil provides a

Fig. 22.10 Orbital atherectomy equipment and diamond-coated crown (© Cardiovascular Systems, Inc. All rights reserved. Used with the permission of Cardiovascular Systems Inc.)

shapeable tip that may facilitate direct wiring, as well. The ViperWire Advance, on the other hand, has a more supportive body and results in more wire bias, vessel straightening, which usually requires a microcatheter delivery system and exchange for appropriately delivery.

Orbital Atherectomy Steps:

- Patient Preparation
  1. Evaluate if patient is adequately hydrated and is able to receive vasodilators.
  2. Dual antiplatelet therapy on board.
  3. Anticoagulation with bivalirudin or heparin.
- Console Preparation
  1. Connect the spike end of saline tubing to a 1 L bag of normal saline with 20 mL of ViperSlide™ lubricant and connect the other end of the tubing to the orbital atherectomy device luer. Hang flush bag on to the saline pressure sensor and make sure the pressure sensor is plugged to the back of the console. Continuous infusion of the flush solution is critical for safe atheroablation.
  2. Open the door located on the front of the orbital atherectomy pump and place the saline tubing in between the saline tubing positioners, making sure that there are no kinks and lock the door.
  3. Switch on the master power switch located at the back of the pump. Press the green "Start" button and hold the blue "Prime" button until the purge solution exits the sheath near the crown. Ensure that there are no air bubbles in the tubing prior to beginning the atheroablation procedure.
- Initial Procedure Steps
  1. Direct wiring of the lesion with a ViperWire is reasonable in certain cases, but the majority an exchange wiring will be necessary with the use of workhorse wire and microcatheter.
  2. Unlock the control knob and ensure that the crown moves freely in agreement with movement of the control knob.
  3. Move the crown control knob to 1 cm proximal (away from the shaft) and release the guidewire brake (pull up). Ensure flush is running and back feed the ViperWire through the crown, advancing the crown to the copilot.
  4. Lock the control knob and guidewire brake. Elevating the crown to prevent entanglement and press the on/off button on the device to perform speed check. Release the guidewire break and verbally countdown for crown advancement, having the primary operator advancing the crown towards the ostium of the artery and the second operatory pull the wire from the back of the device under fluoroscopy. The second operator should be pulling the guidewire to ensure the wire is not migrating distally and the guide is not moving aggressively in and out of the ostium. If the guidewire is advancing forward or the guide is moving aggressively, the second operate should pull the wire faster.
  5. Position the crown approximately 1 cm proximal to the lesion. This step can be performed on GlideAssist, by pressing and holding the low RPM button

until it flashes. While having the guidewire brake locked, unlock the crown, press the on/off button, and advance on GlideAssist to the lesion. Turn off GlideAssist by pressing on/off and the low RPM button again.
6. Remove tension and inertia from the system as per steps 7–9.
7. Unlock advancer knob and move back and forth.
8. Open copilot and move sheath back and forth.
9. Active GlideAssist by pressing the low RP button on the device, which turn on a flashing green light. Briefly tap on the on/off button. Deactivate GlideAssist by pressing the low RPM button again.

- Orbital Atherectomy Steps
1. Lock the guidewire brake as the orbital atherectomy device will not work with the brake unlocked.
2. Slowly advance and retract the crown advancer knob to begin atherectomy of the lesion at a rate of 1 mm per second. If treating a tight lesion, advance and retract slowly to verify 1:1 movement. The device works both antegrade and retrograde so avoid rapid movements while the crown is orbiting.
3. For every 20 s of treatment, a rest period of equal time is recommended. The pump console will beep after every 25 s interval of treatment time. Maximum total treatment time should not exceed 5 min.
4. Keep SBP >100 bpm and stopping atherectomy temporarily in this setting to administer continuous fluids and IV doses of Neo-synephrine. Monitor for bradycardia.
5. Using a series of intermittent treatment intervals and rest periods, slide the advancer crown knob back and forth across the lesion, always stopping the crown in healthy vessel segment.
6. After treatment, activate GlideAssist mode again. The second operator should be advancing the guidewire from the back end of the device while the first operator is removing the crown.
7. Take a cine image to rule out any complications.
8. Advance a workhorse wire to across the lesion parallel to the guidewire and perform PTCA with previously prepared noncompliant balloon.
9. Deliver the appropriately sized stent to the lesion, remove the ViperWire and deploy the stent.

Rotational Atherectomy vs. Orbital Atherectomy (Table 22.1)

Table 22.1 Comparison of rotational atherectomy vs orbital atherectomy

	Rotational atherectomy	Orbital atherectomy
Setup	Longer setup	Easier setup
Learning curve	Steep	Short
Device size	1.25–2.5 mm, can be changed outside of body	1.25 mm, single crown capable of treating varying diameters with increasing orbiting speed

(continued)

Table 22.1 (continued)

	Rotational atherectomy	Orbital atherectomy
Guide catheter	6 F (1.25 mm–1.75 mm) 7 F (2.00 mm) 8 F (2.15 mm) 9 F (2.25 mm–2.38 mm) 10 F (2.5 mm)	6 F
Aorto-ostial lesion	Yes	No
CTO/Subtotal lesions	Yes	No
ISR lesions	Yes	No
Distal lesions	Yes	Yes
Angulated lesions >90°	Yes	No
Vessels >3.5 mm	Yes	No

Excimer Laser Coronary Atherectomy

The clinical utility of coronary laser is limited and is currently rarely used as a first line strategy for severely calcific lesions. The current excimer laser system (CVX-300 ELCA System, Spectranetics Inc., Colorado Springs, Colorado) is based on a XeCl laser catheter system (X-80, 0.9 mm) consists of concentrically arrange fibers around a guidewire that is capable of generating energy up to 80 mJ/mm^2, compatible with a 6 F system. Ablation is achieved through photochemical, photothermal, and photomechanical effects. The latter of these effects is the result of the interaction of the laser with liquids, i.e. saline, blood, or contrast. This photomechanical effect is amplified when interacting with contrast and is therefore typically performed with continuous intracoronary saline infusion [13].

Clinically, laser atherectomy is the only option when a microcatheter or Rotawire/ViperWire are unable to cross the lesion. However, in these scenarios the relative efficacy of the laser atherectomy alone is lower than that of rotational atherectomy in severely calcific lesions [14, 15]. Increased efficacy has been demonstrated employing the RASER technique, which combines laser atherectomy to facilitate rotational atherectomy [16]. Laser atherectomy, however, does seem to have a unique role in-stent restenosis due to underexpanded stent with modification of the calcium behind stent struts with intentional contrast infusion and amplified atherectomy result.

Intracoronary Lithotripsy: Shockwave C² ™

One of the newest technologies available has been recently adapted nephrolithiasis treatment to treat heavily calcific coronary artery lesions. Shockwave Medical Inc. (© 2020 Shockwave Medical Inc., All Rights Reserved) has previously engineered intravascular lithotripsy treatment systems for calcific peripheral arterial disease (Shockwave M⁵ ™) and recently been conducting clinical trials of the new intracoronary lithotripsy calcium modification system Shockwave C² ™, which uses sonic pressure waves to crack calcium in situ [17] (Fig. 22.11). The lithotripsy device has an integrated 12 mm semi-compliant balloon with 2 emitters (6 mm between the emitters and 4 mm/2 mm between emitters and marker bands), and is delivered over a 0.014" guidewire through a 6 F guide catheter. After inflating to only 4 atm, a small spark at the emitters vaporizes the saline-contrast solution and creates a bubble, which rapidly expands and collapses within the balloon, thereby creating a sonic burst pressure wave. While reflecting off and cracking calcium with an effective pressure of roughly 50 atm, the emitters create a localized field effect 3 mm deep within the vessel to fracture both intimal and medial calcium. Each cycle consists a single pulse every second for 10 s, with a maximum 80 bursts recommended in a single segment. For maximal optimization of the energy profile, it is also recommended to reposition the lithotripsy device with 2 mm overlap across the length of the lesion. The integrated balloon, after the lithotripsy is then used to dilate the lesion for maximal luminal gain and subsequent stent delivery. This device is currently available internationally in 2.5–4.0 mm, with a crossing profile 0.043"–0.046" (Fig. 22.12).

Fig. 22.11 Shockwave C² ™ intracoronary lithotripsy system, including a generator, connector cable for button activation of lithotripsy, and the lithotripsy catheter (© 2020 Shockwave Medical Inc., All Rights Reserved. Used with permissions from Shockwave Medical Inc.)

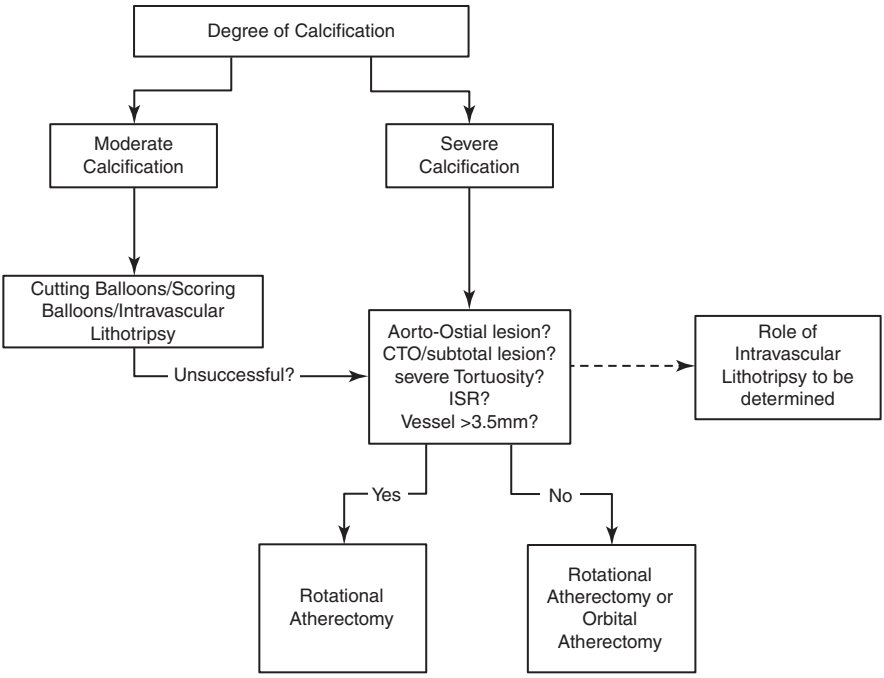

Fig. 22.12 Severely calcified lesion intervention algorithm

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Transradial Coronary Interventions

23

Tarun Jain and Nitin Barman

Introduction

Transradial angiography and intervention continue to become increasingly common as an access site for coronary procedures due to decreased rates of access site complications, improved patient comfort and reduced adverse cardiac events in certain subsets. This chapter provides an overview on radial interventions.

Basic Catheter Techniques for Diagnostic Angiography

Left or Right Radial Artery Access

- Transradial angiography and PCI are predominantly performed from the right radial artery due to cardiac catheter laboratory set, operator comfort, and preferences [1].
- Advantages of using the left radial artery approach include a significantly shorter learning curve and progressive reduction in fluoroscopic and coronary arterial cannulation times when compared with the right radial artery approach. Left radial artery is also preferred in CABG cases with in situ LIMA grafts. It is also preferred in patients in whom tortuous anatomy is suspected and in whom coronary engagement may be more difficult (i.e. age>80 years, short stature, severe hypertension, etc.) [1].

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Navigation

- The standard 0.035″ (150–180 cm) J wire is most commonly utilized to introduce catheters. The 0.035″ J wire has the advantage of avoiding most side branches in the forearm and arm and providing better support. The use of fluoroscopy can aid in the proper passage of these wires into the ascending aorta without engaging side branches (carotids, vertebrals, mammary, etc.) but can be avoided while traversing the arm in most cases.
- The 0.035″ Angled Glide Terumo Wire (Terumo®, Tokyo, Japan) can be used to navigate tortuous vasculature but should always advance under fluoroscopic guidance.

Pearls

- The 0.035″ J wire is the safest wire to use and will be effective in over 90% of cases. If a glide wire is needed, be sure to use a curved-tip wire and utilize fluoroscopy to guide the progress of the wire. In difficult cases, a 0.014″ wire may be used.
- If the wire or catheter does not progress easily, be aware of several possibilities:[1]
  - Spasm.
  - Small radial artery.
  - Loops.
  - High takeoff.
  - Recurrent radial artery.
  - Stenosis.
  - Side branch.
  - Dissection.

Radial Diagnostic Catheters

- Judkins Left (JL)[2] or Judkins Right (JR).
- Specialized radial catheters—Tiger and Jacky (Terumo®, Tokyo, Japan).

Cannulation

- Note that described maneuvers to engage catheters in this section are performed in the left anterior oblique (LAO) 20–30°.

[1] See “Troubleshooting.”

[2] Note: When utilizing the Judkins catheters, successful cannulation of the left main coronary artery will more easily be achieved with a Judkins catheter that is 0.5 size smaller than that chosen for the transfemoral approach.

- Left Main (LM) Coronary Artery
  - After obtaining access, gently advance a J wire (standard length or exchange length J wire), and with the aid of fluoroscopy, traverse the aortic arch and advance to the level of the aortic arch.
  - Advance the JL 3.5 catheter to the level of the transverse aortic arch; ask the patient to take a deep breath and advance the J wire to the level of the aortic valve. If the J wire does not automatically advance into the left coronary sinus, use the JL catheter to direct the J wire into the proper sinus. Advance the JL catheter into the left coronary sinus and withdraw the J wire.
  - Gentle clock or counter clock rotations combined with a gentle forward push will result in successful cannulation.
  - In patients with subclavian tortuosity, the following technique may aid in LCA engagement, Fold the catheter in the mid ascending aorta, rotate and advance towards the left coronary sinus and advance the wire while simultaneously withdrawing the catheter. As the catheter unfolds, withdraw the wire to allow catheter tip engagement.
  - After completion of LCA angiography, the JL 3.5 catheter is disengaged from the left main coronary artery, and an exchange length 0.035″ J wire is advanced to the level of the aortic valve, and the JL catheter is extracted over the wire.
- Right Coronary Artery (RCA)
  - Advance the JR4 catheter over the exchange wire to the right or left coronary sinus and remove the J wire.
  - If the catheter is in the left coronary sinus, pull and turn the catheter clockwise to place it in the right coronary sinus.
  - A clockwise rotation of the JR4 catheter will enable proper cannulation.
- LIMA and RIMA
  - Obtaining access on the ipsilateral side of the bypass graft is the easiest approach.
  - Advance J wire across the subclavian.
  - Advance JR4 catheter or internal mammary catheter beyond LIMA graft or RIMA graft.
  - Pull back wire into the catheter, pull back catheter, and engage graft. Clock the catheter for LIMA engagement via the left radial artery approach and counter-clock the catheter for RIMA engagement via the right radial artery approach (opposite from femoral access).
- Left Ventricular (LV) Angiography
  - Left ventriculography is performed in the same approach as that of the femoral artery.
  - No special sizing adjustment for the pigtail catheter is necessary.

Specialized Catheters

- Catheters made specifically for radial procedures include the Tiger and Jacky.
- Advantage: They can be used to engage both the left main and right coronary arteries (universal catheters) and thus obviate the need for an exchange to a second catheter.

Pearls

- When using the right radial approach, left-sided catheters have to be downsized. Catheters to engage the right coronary artery often have to be longer (i.e., Amplatz right catheters instead of Judkins right catheters may have to be used). Generally, no size adjustment is necessary when using the left radial approach.
- If there is trouble engaging a coronary artery, it is often easier to try another catheter using an exchange wire.

- Disadvantage: Require specific experience in maneuvering to use them effectively and safely.

Transradial PCI

Guiding Catheter Selection

- When using the right radial access, choose a left guiding catheter that is 0.5 smaller than a guide that would be normally chosen had femoral artery access been utilized (i.e., if a Mach 1 VL 3.5 guide would have been used for a femoral approach, the radial equivalent is a Mach 1 VL 3.0). A longer right coronary guide may be required. Amplatz guides may be useful when performing right coronary artery interventions to facilitate support.
- Generally, no size adjustment is necessary if the left radial access is used.
- Specialized guides for radial interventions include the Ikari guides (Terumo®, Tokyo, Japan). These specialized guides are designed for optimal support during radial interventions and may often be used for both left and right coronary interventions.

Other Equipment

- The same guidewires, balloons, and stents that are typically used for the femoral artery are also utilized for transradial interventions.
- Specialized interventional devices including rotational atherectomy devices can also be used safely during transradial interventions.

Pearls

- Size adjustments in guide selection are required when using the right radial artery. Generally, no guide size adjustment is necessary when using the left radial artery.

Post-Procedure Care

- Hemostasis: Terumo® TR Band™ (Fig. 23.1) is a device designed to achieve patent hemostasis for the transradial approach.
  - Choose appropriate TR Band size (regular or large).
  - Wrap the TR Band around the wrist and secure in place via Velcro band.
  - A small green box indicates where the band should be placed proximal to the radial percutaneous site.
  - Instill 15 cc of air. Then remove air 1 cc at a time until bleeding occurs at which time 1–2 cc of air is reinjected into the balloon. The idea is to achieve hemostasis without completing occluding the radial artery.
  - Save the syringe for future deflation.
- TR Band Removal
  - Air removal can begin 1 h after the sheath is pulled (2 hrs for PCI).
  - Once it is time to remove the TR Band, the operator should withdraw 1 cc of air at a time that was initially instilled into the balloon and observe for any bleeding.
  - If bleeding occurs, reinject air that was removed until hemostasis is achieved, and reattempt deflation in 30 min.
  - If no bleeding is observed, the operator should remove all the air, deflate the balloon completely, and observe for bleeding. If there is no bleeding, the operator can remove the TR Band and place a protective covering (i.e., Tegaderm™) over the radial percutaneous site.
  - Vital signs and pulse check q 15 min × 4 for the first hour while TR band is in place, q 30 min × 2 during the second hour, and q 1 h thereafter until post-sheath removal. Assess for presence of feeling in the fingers and capillary refill in distal fingers and nail beds until TR Band is removed. Radial artery patency should be assessed using reverse Barbeau test.

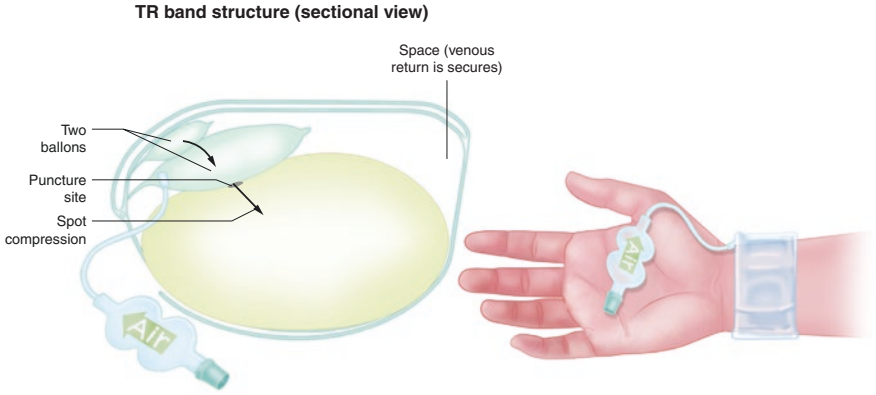

Fig. 23.1 TR band structure

Pearls

- When placing a hemostatic device after a radial intervention, avoid occlusion of the radial artery. Applying just enough pressure to achieve hemostasis is essential to avoid post-procedure radial artery occlusion. Use reverse Barbeau test to confirm.

Discharge Instructions

- Limit bending of the affected wrist for 24 h.
- No lifting of greater than 5 lbs with affected hand for 5 days.
- No bathing showering or driving for 24 h.

Troubleshooting

Spasm

- Minimize pain and administer additional sedatives.
- Repeat administration of intra-arterial nitroglycerin (200 mcg) and/or verapamil (250 mcg).
- Apply a warm compress over the forearm.
- If sheath removal is not possible due to radial artery spasm around it, wait 1 h, administer additional sedatives/analgesics, and reattempt.
- Persistent radial artery spasm preventing sheath removal may require deep sedation with the assistance of an anesthesiologist.

Subclavian Loop

- Navigate the loop with 0.035″ angled glide wire.
- Advance diagnostic or guide catheter over the wire. Once the catheter has passed beyond the loop, a counterclockwise rotation while pulling back will straighten the loop. Deep inspiration with breath holding may allow further negotiation of subclavian artery tortuosity by modifying the angulation of the brachiocephalic trunk.
- For extreme tortuosity, it may be necessary to cannulate the LCA/RCA with the stiff shaft 0.035″ wire within the catheter.
- Only attempt to manipulate the catheter for cannulation with loop straightened. If the loop was reformed, repeat the above step to straighten the catheter. It will be extremely difficult to manipulate the catheter with a loop present.

Radial Artery Tortuosity and Loops

- Perform selective angiography to identify the problem.

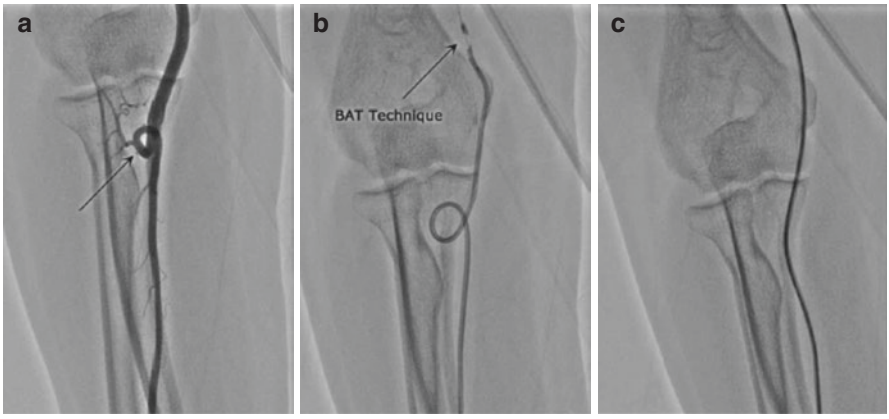

Fig. 23.2 (**a**) Right radial artery loop (**b**) Balloon inserted half in and half out of the guide catheter and inflated at low pressure while advancing the entire system as a unit (**c**) Successful advancement of the guide catheter past the radial loop resulting in straightening of the radial loop

- Utilize hydrophilic 0.035″ Terumo glide wire or 0.014″ coronary guidewire to navigate the loop or tortuosity. Advance a hydrophilic catheter over the wire and then apply a gentle counter-clock rotation while pulling back the catheter to straighten the loop. If this technique does not work, balloon-assisted tracking technique can be used as described below.
- Balloon assisted tracking (Fig. 23.2) is a technique that may be used to overcome radial tortuosity, spasm, or loops. A regular 0.014″ hydrophilic coronary angioplasty wire is passed through the difficult area under fluoroscopy. Then a diagnostic or guide catheter is loaded with a long compliant balloon (20 mm) positioned half protruding beyond the tip of the catheter. A 5 Fr catheter will accommodate a 2.0 mm balloon, whereas a 6 Fr guide may require a 2.5 mm diameter balloon. Once correctly positioned, the balloon is inflated to 8–10 atmospheres. The entire system is then advanced as a unit post the obstruction. Once the catheter has reached the ascending aorta the 0.014″ coronary angioplasty wire and balloon catheter may be removed and replaced with a standard 0.035″ guide wire, providing greater support to further position the catheter into the aorta root [2].
- An exchange length J-tip (260 cm) guidewire is then used for subsequent exchange catheter exchanges to avoid loss of radial access.

Arteria Lusoria (Lusorian Artery)

- The innominate artery arises from the descending portion of the thoracic aorta.
- It is difficult to pass a catheter into the ascending aorta. Other problems include difficult cannulation of the coronary arteries.
- Advance the catheter into the ascending aorta (LAO 60).
- Ask the patient to take a deep inspiration and attempt to advance wire into ascending aorta.

- If the wire only goes down the descending aorta, push the catheter into the descending aorta, pull the wire and slowly withdraw the catheter, and direct it into the ascending aorta with rotation.
- Be prepared to choose an alternate site of vascular access. This anatomy is associated with one of the highest rates of transradial intervention failure.

Catheter Kinking

- Rotate under fluoroscopy in a direction opposite of what caused the kink.
- Gentle forward or backward movement of the catheter will facilitate unkinking rotation.
- If the kinked portion of the catheter can be safely brought to the elbow, then applying and inflating a manual blood pressure cuff on the arm may facilitate unkinking.
- Whenever possible, pass a 0.035″ stiff Terumo® guidewire beyond the kinked portion to facilitate removal of the catheter in one piece.

Complications [3–6]

Radial Artery Occlusion

- Radial artery occlusion (RAO) following transradial access occurs in 2–10% of patients.
- RAO is usually asymptomatic due to ulnar–palmar collateralization vascular blood supply of the hand. However, RAO precludes the use of radial artery access in any future coronary interventions.
- Procedural duration, arterial diameter-to-sheath ratio, compression time, and pressure all are risk factors for RAO.
- Prevention: Administration of intravenous or intra-arterial unfractionated heparin 5,000 U or 50 U/kg or a higher dose as a bolus is recommended following placement of radial artery introducer sheath.
- Use of lowest profile sheath and/or catheter system required for procedural success, with attention to sheath/catheter-to-artery ratio.
- Patent hemostasis should be the default strategy, regardless of the method or device used for compression of the arteriotomy. Concomitant ipsilateral ulnar artery compression is recommended to further maximize radial artery patency if there is acute loss of radial pulse.

Radial Artery Pseudoaneurysm

- Rare and most likely with antecedent oral anticoagulation.
- Treatment: US guided compression bandage.

- Treatment: US guided thrombin injection.

Brachial Artery (BA) or Subclavian Artery (SA) Dissection

- Prevention: Never advance the catheter without a guidewire.
- Do not push wire or catheter against resistance.
- Prevention: Be cautious in excessive brachial or subclavian tortuosity.
- Treatment: Prolonged balloon dilation and stenting of flow-limiting dissections, however, is rarely required.

Reflex Sympathetic Dystrophy

- Very rare—likely a result of prolonged and aggressive compression.
- Treatment: Multimodality approach utilizing medical pain management and physical therapy.

Hematoma, Perforation, and Compartment Syndrome

- Predisposing Factors
  - Anticoagulated state.
  - Small vessels.
  - Low BMI.
  - Women.
  - Diabetic.
  - Older patients.
  - Spasm.
  - Tortuosity and remnants of the radial artery.
  - Use of hydrophilic guidewires.
- Avoidance
  - Avoid the use of force when wiring.
  - Utilize fluoroscopic or angiographic guidance (especially when using glide wires).
  - Avoid forceful injections as it may cause hydraulic perforation.
  - Attempt to straighten tortuosity with 0.014″ guidewire first.
  - Use a J wire whenever possible.
- Perforation Management: During Procedure
  - If a perforation occurs, one can continue to use the guiding catheter and complete the coronary intervention, by which time perforation usually seals off. However, be aware of severe spasm.
  - Position BP cuff after catheter passage and inflate BP cuff 20 mmHg below SBP. Monitor finger O_2 saturation during inflated BP cuff. Deflate BP cuff every 15 min.

- Perforation Management: Post-Procedure
  - In addition to above, consider a second compression device (i.e., elastic band).
  - Reverse anticoagulation.
  - Manual compression/blood pressure cuff.
  - Consult vascular surgery for large perforations or hematomas to help manage potential compartment syndrome and circulatory compromise.

Pearls

- The cornerstone to a successful radial artery percutaneous intervention involves a systematic and stepwise approach. Increasing operator experience and knowledge of pitfalls will lead to easier cannulation and shorter procedure times.

Hematoma Classification after Transradial or Transulnar PCI

- Grade I—Local hematoma, superficial.
- Grade II—Hematoma with moderate muscular infiltration.
- Grade III—Forearm hematoma and muscular infiltration below the elbow.
- Grade IV—Hematoma and muscular infiltration extending beyond the elbow.
- Grade V—Ischemic threat (compartment syndrome).

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24 Coronary Complications and Management of Percutaneous Coronary Interventions

Raman Sharma, Samin K. Sharma, and Annapoorna Kini

Introduction

Percutaneous coronary intervention has seen vast progress with advancement in stent designs, adjunctive technologies including atherectomy systems as well as development in more potent antiplatelet agents. With continued advancements in these procedures, periprocedural complications, albeit at a reduced frequency, still occur during PCI in a minority of cases. Management of these complications is largely based on the timely recognition and treatment.

Coronary Perforation

Coronary perforation has an estimated incidence in the contemporary era of 0.5%, which is associated with nearly a 13x fold increase in in-hospital major adverse events and 5x fold increase in 30-day mortality [1]. The most common etiology is balloon or stent size mismatch, which become more common with device-artery ratios larger than 1.2:1, as well as high-pressure inflation of semicompliant balloons. However, coronary perforation can also occur in the setting of appropriately sized device but in the context of severe coronary calcification, extensive dissection or unintentional coronary wire migration, especially with the use of hydrophilic guidewires. Use of atherectomy devices, female sex, CTO intervention, advanced age, and previous CABG are also associated with increased incidence of coronary perforation.

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Type	Definition
I	Crater extending outside the lumen only and in the absence of linear staining, angio-graphically suggestive of dissection.
II	Pericardial or myocardial blush without >1 mm exit hole.
III	Frank streaming of contrast through >1 mm exit hole.
III cavity spilling (III CS)	Perforation into an anatomic cavity chamber or coronary sinus.

Fig. 24.1 Ellis Classification of coronary artery perforations. Images printed with permissions [4]

Classification

- Two classification schemes have been proposed for coronary perforations. The Ellis classification scheme divides perforations into 3 different subtypes (Fig. 24.1) [2]. Type I perforations are defined as the development of an extraluminal crater without extravasation, Type II are defined as the presence of a pericardial of a myocardial blush without contrast jet extravasation and without a ≥1 mm exit hole, and Type III defined as extravasation through a frank perforation ≥1 mm exit hole. The Type III perforation has an alternative subtype, Type III-CS, which is defined a perforation into an anatomic chamber, such a coronary sinus, atria or ventricle. The Kini classification schemes simply divide coronary perfusions into 2 categories. Type I is described as "myocardial stain" with no frank dye extravasation whereas Type II as a "myocardial fan" with dye extravasation into pericardium, coronary sinus or cardiac chamber [3].

Outcomes

- Patients with coronary perforations of either Ellis Type I or Kini Type I subtype, generally have an excellent overall prognosis. On the other hand, those with Ellis Type II/III or Kini Type II coronary perfusions have a significantly higher rate of complications as well poor long-term outcomes.

Treatment

- Management of coronary perforations largely depends on severity and the patient's hemodynamic status. The most important aspect of management is the prompt recognition of a perforation. The onset of sudden, acute chest pain during balloon inflation or stent deployment should immediately raise the suspicion of coronary perforation and the balloons should never be removed the guide catheter until angiography has confirmed or excluded the presence of coronary diagnosis.

Ellis Type I Coronary Perforations

- For confirmed Ellis Type I, or Kini Type I perforations, the following steps will most often be sufficient for treatment of the coronary perforation:
  1. An appropriately sized balloon (often the same balloon that caused the perforation) should be inflated to the lowest possible pressure just proximal to the perforation verified by contrast injection, usually 2–4 atm, for 10 minutes
  2. If the anticoagulant is bivalirudin, stop the infusion. In cases anticoagulated with heparin, consider reversal with intravenous protamine (1 mg protamine/100 U heparin administered for goal ACT >150 s). This decision should be based on the potential subsequent risk of acute stent thrombosis.
  3. Deflate the balloon and obtain a cineangiogram. If continued extravasation is observed, repeat balloon inflation for another 10 minutes, reposition the balloon and consider going to high-pressure inflation, while keeping special attention to development of myocardial injury and hemodynamic instability.
  4. Obtain a state transthoracic echocardiogram while patient is still on the table and plan to monitor the patient in the cardiac intensive care unit post-procedure
  5. In the event of worsening pericardial effusion leading to cardiac tamponade confirmed by transthoracic echocardiogram, emergent pericardiocentesis should be performed with aspirated blood being auto-transfused into a vein for hemodynamic stability. Vasopressors should be used as necessary.

Ellis Type II/III Coronary Perforations

- For confirmed Ellis Type II/III or Kini Type II perforations, the same initial steps as above should be performed prior to moving down the following pathway. PTFE stents (Fig. 24.2) and microcoil embolization (Fig. 24.3) have reduced the rates of cardiac tamponade, need for emergent surgery as well as mortality. However, despite progress in the design of PTFE stents, they still are bulky devices and their delivery may be challenging.
  1. For persistent contrast extravasation, consider the use of occlusive coils or beads or the use of polytetrafluoroethylene (PTFE) stents.

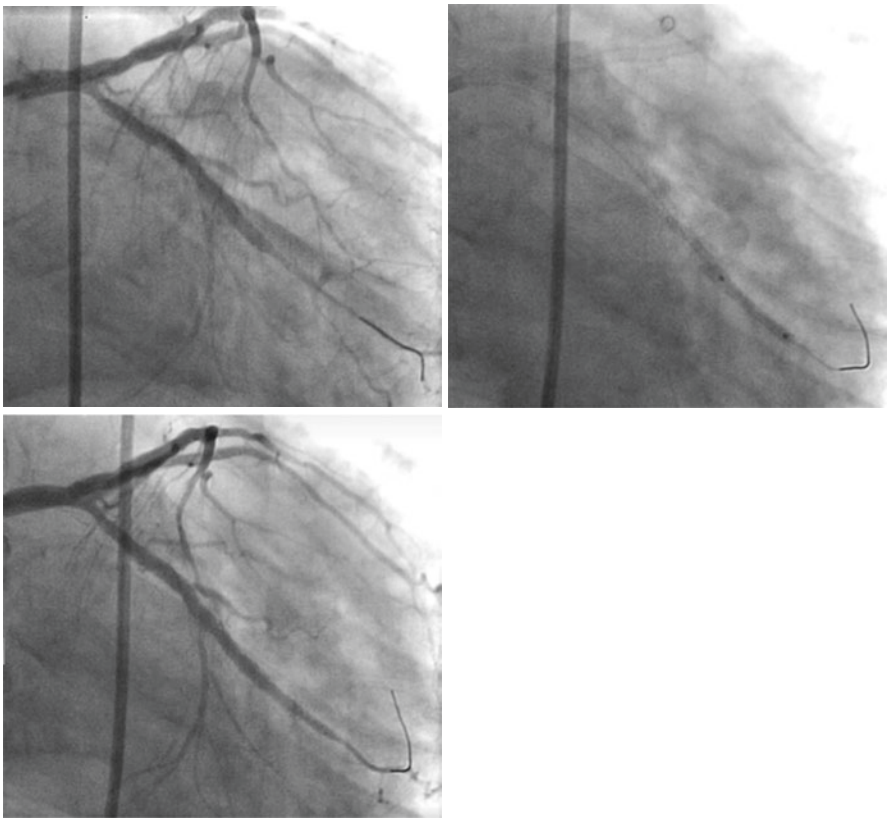

Fig. 24.2 PTFE stent deployment for Type III coronary artery perforation. Top left: Type III coronary perforation of LCx-OM1 after pLCx stent deployment. Top right: After unsuccessful balloon tamponade, not shown, persistence of contrast extravasation requiring PTFE stent delivery to lesion with single guide catheter system. Bottom: After high-pressure post dilatation of PTFE stent, well expanded LCx-OM1 PTFE stent with appropriate sealing of coronary perforation

2. If using a PTFE stent, a 6Fr guide catheter will accommodate stents <4.5 mm in diameter, whereas ≥4.5 mm stents will require a 7Fr guide catheter. A second access can be obtained for a "ping-pong" guiding catheter technique can be performed to facilitate rapid PTFE stent delivery and deployment, especially in tortuous anatomy. If anatomy is forgiving, a single guide catheter system can be used.
3. Momentarily deflate the angioplasty balloon to allow passage of the guidewire of the second guiding catheter and quickly advance the PTFE stent.
4. Remove the angioplasty balloon and deploy the stent at the appropriate location to seal the perforation. PTFE stents require high-pressure post-dilation (minimum 20 atm) for adequate expansion and minimizing ISR.

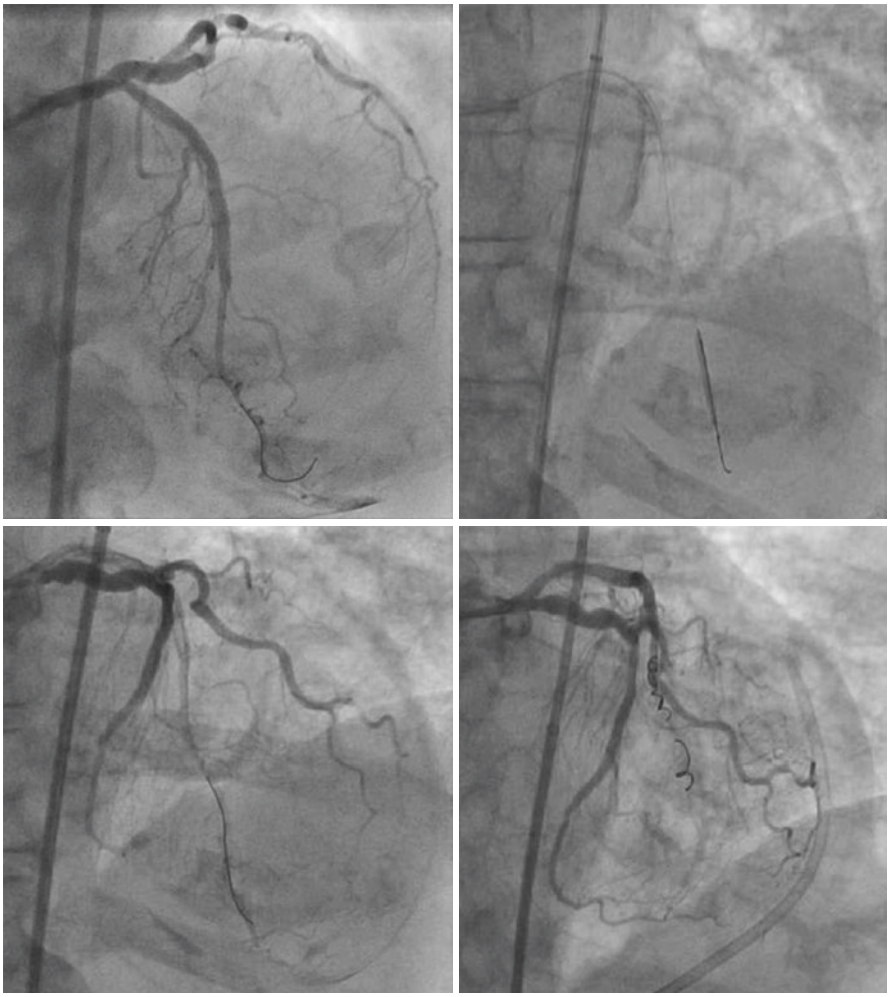

Fig. 24.3 Microcoil embolization to coronary perforation. Top Left: Evidence of distal LAD Type III coronary perforation. Top Right: Balloon tamponade for hemostasis of distal perforation. Bottom Left: Persistence of coronary perforation with persistent contrast extravasation. Notice the enlarging contrast shadow in the pericardial space indicating rapidly enlarging pericardial effusion. Bottom Right: Microcoil embolization deployment (x2) achieving hemostasis of distal perforation and interval pericardial drain placement and improvement of pericardial effusion

5. Confirm with angiography that perforation has been sealed, followed by TTE for assessment of pericardial effusion and pericardiocentesis if necessary, as above.
6. If using microcoil embolization, you will need microcoils, microcatheter delivery system and a 0.018" wire for delivery.
7. Once the microcatheter has reached to the appropriate microcoil delivery location, apply forward tension to avoid proximal migration of the

microcatheter and thereby proximal microcoils delivery. Repeat of delivery of up to 3 microcoils to achieve hemostasis of distal perforation.

Abrupt Vessel Closure

Abrupt vessel closure is one of the most common complications during PCI and has dramatically decreased in incidence to 0.3% in the contemporary era. The potential mechanisms for abrupt vessel closure are dissection, intracoronary thrombus formation, native thrombus or atheroma embolization, air injection or spasm.

General Treatment

- The immediate priority in the management of abrupt vessel closure is to ensure the intraluminal wire position. Once confirmed, proceed down the following algorithm.
  1. Perform Fogarty maneuver with an angioplasty balloon or stent balloon. This will dislodge any thrombus present at the site of intervention and facilitate the reestablishment of flow.
  2. Recheck the ACT and administer additional anticoagulation to maintain ACT>300.
  3. If abrupt vessel closure is due to a dissection, perform balloon angioplasty to seal the dissection flap and reestablish the flow and stabilize hemodynamics with inotropes, atropine or intravenous fluids. Treat arrhythmias with anti-arrhythmic and cardioversion if indicated. Consider IABP insertion if necessary.
  4. Perform re-stenting if dissection occurs in medium to large size vessels. For small size vessels, balloon angioplasty may be sufficient.
  5. If thrombus is present, can consider the use of the Pronto® Extraction Catheter (Teleflex) for aspiration thrombectomy or Twin-Pass® Dual Access Catheter (Teleflex) for distal vasodilator administration. Consider GP IIb-IIIa inhibitors as well.
  6. If etiology appears to be air embolism, this should be managed by increasing systemic blood pressure with vasopressors and repeated aggressive intracoronary saline flushing to move the air to distal microvasculature.
  7. If the above measures are ineffective, obtain multiple angiographic views to rule out left main dissection or distal embolization. Selective injection of contrast through the central balloon lumen or intravascular ultrasound (IVUS) may also be helpful in such cases.
  8. Administer intracoronary nitroglycerin if patient has stable hemodynamics (to rule out the possibility of spasm).
  9. After successful reestablishment of antegrade flow, admit these patients for close monitoring in the cardiac intensive care unit.

10. If none of these approaches are effective, these patients may warrant urgent CABG.

- The above treatment algorithm is a general sequence of steps to follow to accurately and rapidly identify the etiology of abrupt vessel closure and provide treatment. Below are some special considerations regarding the etiologies of abrupt vessel closure.

Coronary Artery Dissection

- *NHLBI* Classification (Fig. 24.4) [5]
  1. *Type A*: Minor radiolucent areas within the coronary lumen during contrast injection with little to no persistence of contrast after the dye has cleared.
  2. *Type B*: Parallel tracts or a double lumen separated by a radiolucent area during contrast injection, with minimal to no persistence of contrast after dye have cleared the lumen.

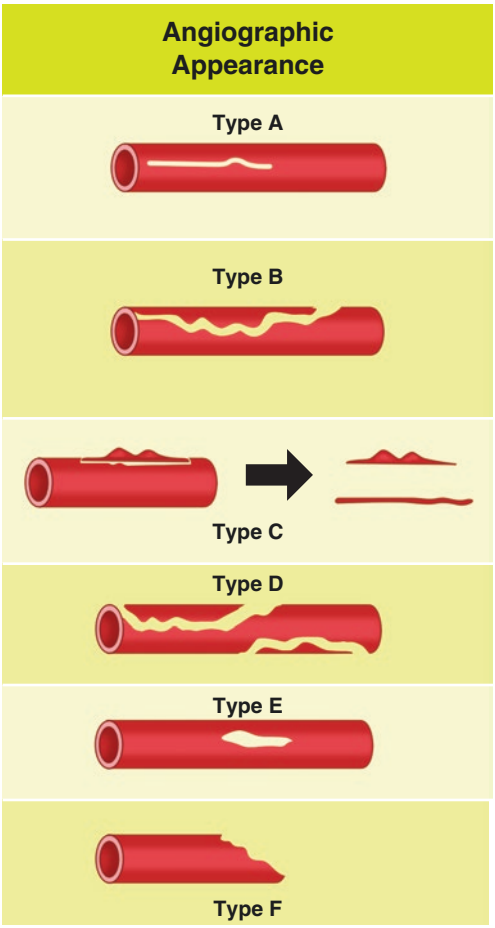

Fig. 24.4 NHLBI Classification of dissection

3. *Type C*: Contrast outside of the coronary lumen (extraluminal cap) with persistence of contrast after dye has cleared the lumen.
4. *Type D*: Spiral ("barber shop pole") luminal filling defects, frequently with excessive contrast staining of the dissected false lumen.
5. *Type E*: Appear as new, persistent filling defects within the coronary lumen.
6. *Type F*: Lead to total occlusion of the coronary lumen without distal antegrade flow.

- Generally, Type A coronary dissections are rarely flow limiting, infrequently have hemodynamic or clinical consequences and can usually managed medically. Type C-Type F are more likely to require further intervention, with the most crucial step being to never lose wire access of the true lumen, the most important for Type F dissections. For Type B dissections, however, use of intravascular OCT may provide insight on whether or not to stent the dissection flap. On OCT, if the dissection flap is >2 mm, it is reasonable to cover this flap with a stent, whereas less than 2 mm can be left alone and management with maximal medical therapy (Fig. 24.5).
- In the cases in which it is appropriate to intervene, there are a few pitfalls to be weary of. When advancing a balloon through the dissection and there is some resistance noted, or if the balloon "watermelons" backwards proximally, it is important to recognize the likely subintimal passage of the guidewire. In this case, do not perform further angioplasty at the risk of furthering the dissection plane and leave this guidewire in its current position. Using a second guidewire, cross the dissection in a different plane using the original wire as a marker of the flap and false lumen. Proceed to treat the dissection if the wire in the true lumen with balloon angioplasty and stenting if appropriate. A similar sequence of steps utilizing a second guidewire should be performed if false lumen position of the guidewire is confirmed on contrast angiography.

Air Embolism

- Intracoronary air embolism is a potentially lethal but rare complication that can result in hypotension, hemodynamic collapse, cardiac arrest and in rare cases death. Coronary air embolism is nearly always iatrogenic and it is imperative to take the necessary steps to prevent it. Inadequate aspiration and flushing of catheters, introduction/withdrawal of guidewire or device, balloon rupture or intracoronary medication injection all may result in air embolism.
- Diagnosis

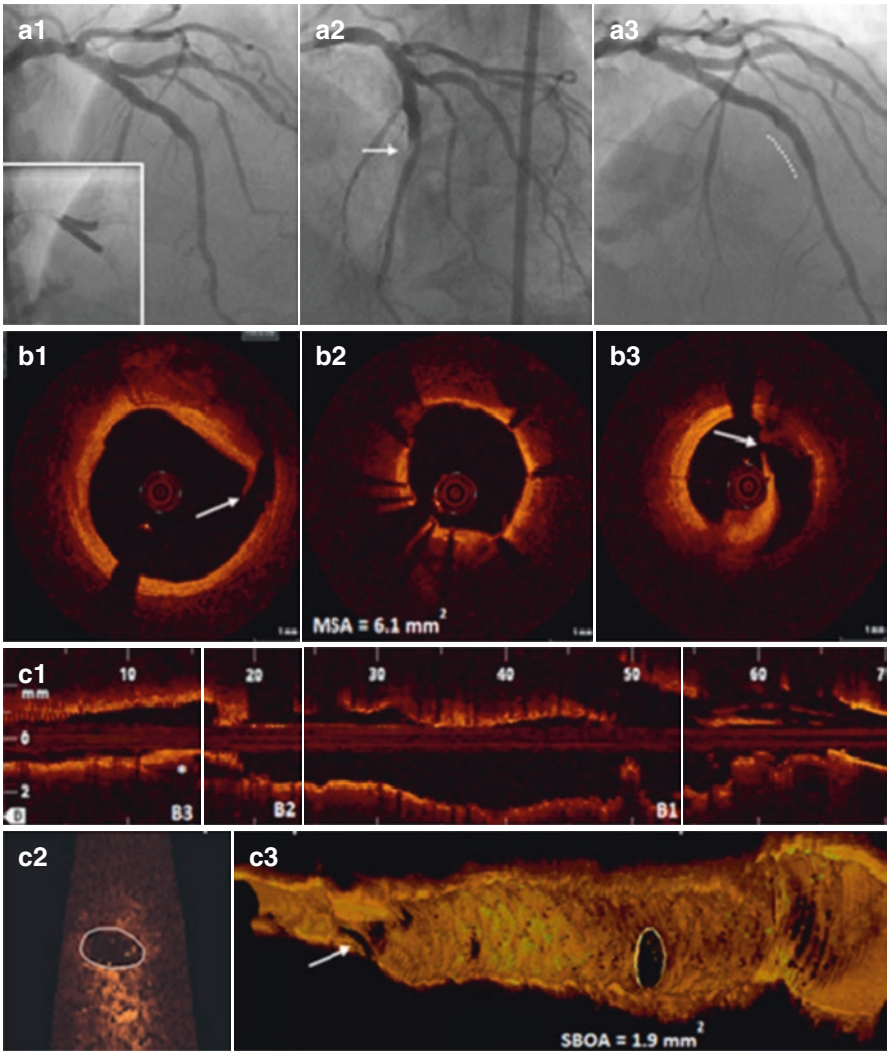

Fig. 24.5 After rotational atherectomy in the LAD with a 1.75 mm burr and 1.25 mm burr in D1, two DES placed in LAD and D1 mini-crush stenting technique with subsequent KBI (a1). Small distal edge dissection was visualized post KBI (a2, arrow). OCT of LAD revealed well apposed and expanded stent with MSA of 6.1 mm^2 (b2), a 2 mm long proximal stent sedge dissection (b1, arrow, and a large 5 mm distal edge dissection (b3, arrow). The distal edge dissection can be seen in longitudinal OCT image (c1, asterisk), with a significant decrease in SBOA from 3.8 to 1.9 mm^2 by 3-D OCT post stenting (c2, c3, circled). The arrow in c3 points to 3-D reconstruction of distal stent edge dissection visualized by both angiography (a2) and OCT (b3). Distal edge dissection was successfully treated with an additional DES (a3, dotted line) and no residual dissection

1. Coronary air embolism is detected under fluoroscopy as intracoronary filling defects during contrast injection. Occasionally this may present itself as an abrupt cutoff of a vessel, which is secondary to an occlusion of distal vessel with an air column. Clinically, a small air embolism may be asymptomatic, with larger air embolism resulting in more catastrophic outcomes.

- Prevention
  1. Do not engage the left main coronary when pulling out the guiding wire unless the patient has excessive aortic tortuosity or an enlarged aortic root.
  2. Do not connect the manifold to the catheter with the flush running. This may lead to an air embolism if the catheter already has a column of air inside it.
  3. Draw back at least 2 cc of blood into the injection syringe and make sure that the interface is free of air prior to injection.
  4. Inject some dye into the ascending aorta prior to engaging left main.
  5. Always ensure that all the catheters and tubings are aspirated, flushed and free of air.
  6. Taking adequate care when prepping stents or balloons and ensure that the syringe tip is facing downwards.
  7. Always inject with the syringe tip facing downwards.
- Treatment
  1. Put patient on 100% oxygen.
  2. Flush air free saline vigorously into the coronary arteries. Aspirate blood via Guide catheter and reinject saline forcefully back into coronary arteries.
  3. Administer IV phenylephrine 200 μg for hypotension. Repeat as needed every minute. If significant hypotension or hemodynamic collapse is present, push IV 1 cc epinephrine (1:10,000 dilution).
  4. Intracoronary injection of vasodilators (adenosine, nitroprusside, verapamil) may be attempted.
  5. Guide wire to disrupt the air bubbles or aspiration of air bubbles using thrombectomy aspiration catheters may be attempted.
  6. Supportive measures should be instituted and patient admitted to intensive coronary care unit for further monitoring.

Guide Catheter Dissection

- Guide dissection quite often be overlooked as it involved the coronary ostium or a segment quite proximal to the target lesion. Unexpectedly slower flow or dampening/ventricularization of the pressure waveform should immediately direct attention to the guide catheter and the status of the ostium of the coronary tree. Visible dissections, in conjunction with EKG changes and new chest pain symptoms are sufficient to confirm this diagnosis, and further contrast injection should be performed judicially or avoided entirely if possible. Intravascular imaging with IVUS is an acceptable method to confirm the diagnosis of guide catheter dissection, again with special attention and care on not losing wire position of the true lumen. Once confirmed, immediate stenting to limit the further dissection propagation is recommended.

No-Reflow Phenomenon

- After prolonged coronary occlusion, the restoration of blood flow to epicardial coronary arteries does not result in prompt, brisk flow down the once occluded coronary artery. There is sufficient structural damage to the microvascular circulation to prevent the restoration of normal blood flow, including a combination of endothelial damage, platelet and fibrin embolization, vasospasm, extracellular/intracellular tissue edema, leading to deposition of neutrophil plugs and myocardial platelet infiltration, and eventual abrupt vessel closure. This etiology of abrupt vessel closure should be differentiated from dissection, as stent placement for no-reflow may potentially worsen the situation.
- Treatment
  1. The no-reflow phenomenon can be diligently prevented and treated with adjunctive pharmacotherapy and mechanical precautions. If no-reflow is suspected, i.e. after rotational atherectomy for heavily calcific lesions, adenosine, verapamil, nitroprusside, or nicardipine can be injected directly into the coronary tree through the guiding catheter. In cases in which these agents are ineffective to result in adequate reversal of no-reflow and other etiologies have been ruled out, use of a microcatheter or dual-lumen catheter for direct distal intracoronary administration of the same agents is recommended. An important consideration should also be made for saphenous vein graft intervention, in which abrupt vessel closure due to no-reflow is not an uncommon occurrence. In these cases, use of distal embolization protection devices upfront prior to angioplasty and stent delivery may mitigate the development of no-reflow.

Periprocedural Myocardial Infarction

The Fourth Universal Definition of a periprocedural MI differentiates between procedural myocardial injury that can be diagnosed by a stand-alone increase in cardiac troponin (cTn) level versus periprocedural MI (Type 4a MI). Periprocedural MI requires an elevation of cTn values greater than five times the 99th percentile URL in patients with normal baseline levels or, in patients with elevated pre-procedural cTn in whom the cTn levels are stable (<20% variation) or falling, the post-procedural cTn must rise >20% to an absolute value more than five times the 99th percentile URL. In addition, there should be evidence of new myocardial ischemia, either from ECG changes, imaging evidence of new loss of viable myocardium or new regional wall motion abnormality in an ischemic pattern, or from procedural-related complications associated with reduced coronary blood flow such as coronary dissection, occlusion of a major epicardial artery or a side branch occlusion/thrombus, disruption of collateral flow, slow flow or no-reflow, or distal embolization [6].

Large periprocedural MIs are associated with an adverse long-term prognosis and has been a focus of ongoing research. With the newer high-sensitivity assays, it was thought to have adjusted the formerly proposed 5x elevation in cTn as per the Third Universal Definition of MI, but recent studies also demonstrated a similar 5-fold increase of high-sensitivity cTn threshold to predict cardiovascular outcomes

at 30 days and 1 year [7]. Recently, Society of Angiography and Interventions (SCAI) has proposed a new definition which recommends using elevation of isolated CK-MB ≥ 10 × local laboratory upper limit of normal (ULN) or troponin ≥ 70 × ULN with emphasis on CK-MB to classify periprocedural MI [8].

Common causes of periprocedural MI include acute closure, distal embolization, no-reflow, or side branch closure. It can be diagnosed by measuring cardiac biomarkers before the procedure and 3–6 h post-procedure. Procedural and lesion characteristics associated with periprocedural MI include intervention on degenerated vein grafts, presence of thrombus, CTOs, long lesions, use of rotational or orbital atherectomy, prolonged balloon inflation, aggressive stent expansion, acute closure, no-reflow, and side branch closure.

- Prevention
  1. The use of intravenous GPIIb-IIIa agents, P2Y12 inhibitors, and statins has been shown to decrease the incidence of post-procedural MI. The use of distal embolic protection devices (EPD) for saphenous vein graft (SVG) interventions significantly reduces the periprocedural MI. For native coronary interventions, EPD have not been shown to be beneficial.
- Treatment
  1. Depends on the underlying cause. If there is angiographic evidence of vasospasm or slow flow, then use of IC verapamil, nitroglycerin, nitroprusside, or adenosine can be helpful. Most cases of periprocedural MI are silent. Conservative management is adequate for modest elevations in such cases. Serial enzyme measurements should be done. Patient could be discharged once the enzyme values start declining even if it remains above the baseline. For patients with persistent ischemic symptoms, EKG changes, or Q wave infarcts, repeat angiography should be considered to rule out stent thrombosis or dissection and to decide of further treatment course.

Complication of Rotational Atherectomy: Burr Entrapment

The presence of the diamond coated abrasive surface on the front, but not the back allows for the feared complication of lodging the burr within a lesion and become entrapped. During high-speed rotation, the heat may enlarge the space between plaque and since the coefficient of friction during motion is lower than that at rest, the burr may pass the calcific lesion easily without significant plaque debulking, allowing it to get stuck on the way back, and called the “kokesi” phenomenon.

The best way to prevent burr entrapment is to employ careful and conservative technique throughout the entire rotational atherectomy procedure, especially diligent relief of system tension/inertia prior to atherectomy.

In the event of burr entrapment, take advantage of the 0.014” spring tip and pull it to dislodge the burr. If this fails, advance the burr at 200,000RPM into the distal lumen and attempt burr removal while at high-speed revolutions. Next steps include obtaining a second arterial puncture for a second guide catheter or upsize the guide catheter over the RotaPro system, which requires cutting the sheath, driveshaft and

Rotawire proximal to the advancing device. A hydrophilic wire should be advanced distal to the trapped burr and balloon dilatation should be performed proximal to the burr to facilitate burr removal. A deep guide catheter intubation or child-in-mother catheter coronary intubation (guide extension catheter) with subsequent pullback of all devices can be alternative method. By simultaneous traction on the burr shaft and countertraction on the child catheter, the catheter tip can serve as a wedge between the burr and entrapping plaque, allowing a large and direct retracting force to retrieve the burr (Fig. 24.6). If all above techniques fail, the patient needs to be referred to CT surgery for surgical removal of the entrapped burr.

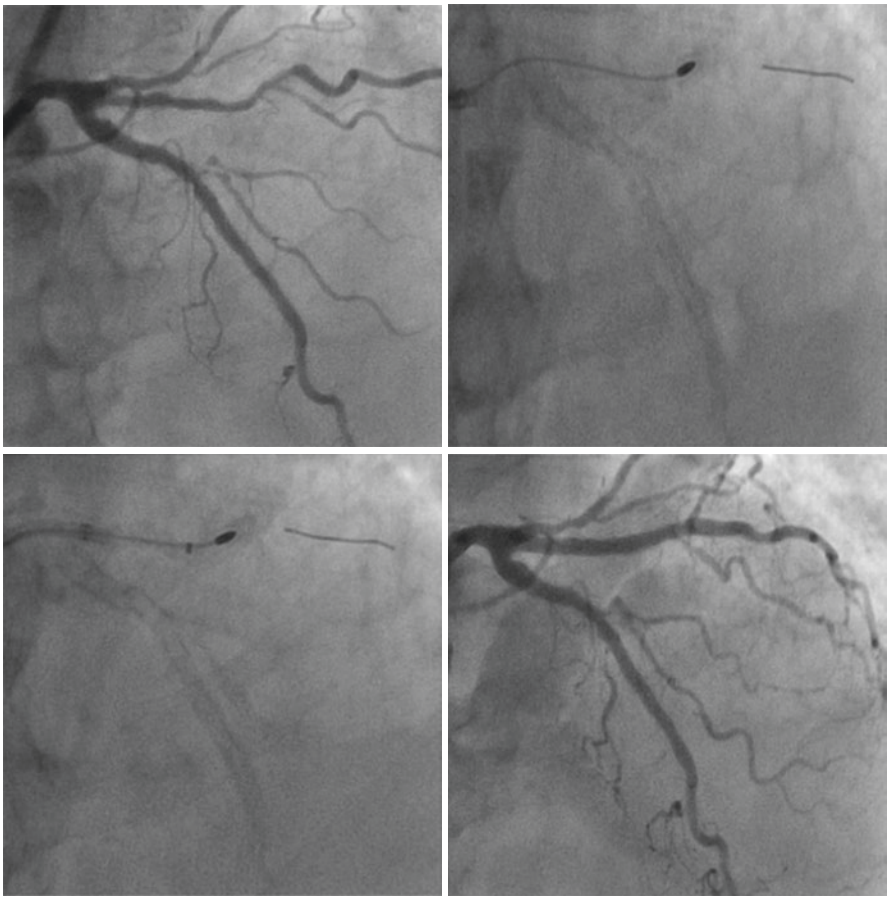

Fig. 24.6 Rota burr entrapment management. Top Left: Highly tortuous and heavily calcific Ramus Intermedius. Top Right: 1.25 mm Rota burr entrapped after crossing highly calcific lesion, unable to be retracted. Bottom Left: "Child-in-mother" catheter system with guide extension catheter advanced immediately proximal to the entrapped Rota burr for direct negative traction for successful Rota burr removal. Bottom Right: After Rota burr removal, post-high-pressure balloon angioplasty and stenting of Ramus Intermedius

Device Embolization

Stents Embolization

- Stents are the most commonly embolized devices (0.32%) and are usually the result of extreme tortuosity, angulation and severely calcific disease, all of which increase the risk of dislodgement of the stent from the delivery system. This may result in system or intracoronary embolization, with the former potentially resulting in CVA and the latter intracoronary thrombosis and subsequent MI.
- In the cases in which stent delivery is difficult, caution should be taken to avoid aggressive stent delivery as this can result in stent deformation and subsequent dislodgement. At this point, the stent should be gently retrieved through the guide catheter and further lesion modification should be performed, including high-pressure dilatation of noncompliant balloon, atherotomy or atherectomy as appropriate.
- Management of stent embolization should be categorized by whether or not the stent is still on the guidewire. In cases in which the stent is dislodged from the delivery system but still on the guidewire, it is often possible to advance a small balloon beyond the stent, inflate it, and retrieve the stent by "dragging" the balloon back. Another option is to pass a second guidewire through the stent struts and intentionally entangle the wires together, thereby entangling the dislodged stent between the two wires and allowing for stent retrieval. In cases in which the stent is not on the wire, use of gooseneck snares or guide extension catheters may facilitate stent retrieval.
- Not uncommonly, stent retrieval is simply unsuccessful and in these cases "crushing" the stent into the stent wall with another stent is the only solution. This, however, is not without risk, as it comes with an increased risk of periprocedural MI, death and need for urgent CABG. Alternatively, if the stent is irretrievable in large coronary artery, surgical removal of the stent can be considered upfront.

Wire Embolization

- When the embolized device is a small guidewire fragment, the first consideration of the operator is to leave the wire within the coronary artery. Current literature does not report any increase in coronary artery thrombotic occlusion due to retained guidewire fragments. However, unraveling of the guidewire should be carefully investigated with the use of IVUS, as this unraveled segment may behave as a nidus for thrombus formation. In some scenarios, the location of guidewire segment, it may be reasonable to consider exclusion of the wire by stenting to prevent distal migration and subsequent perforation and tamponade.

Longitudinal Stent Deformation

New generation cobalt-chromium/platinum-chromium stents with thinner stent struts allow for much better trackability and deliverability. In order to engineer these maneuverability improvements, the number of fixed links between cells and alteration in geometry comes at the price of potential longitudinal stent deformation, defined as the distortion or shortening of the stent along its longitudinal axis (Fig. 24.7). Most commonly, heavily calcific lesions, severely tortuous vessels, guide extension catheter usage, lesion lengths >28 mm, ostial and bifurcation lesions are angiographic features that may predict the possibility of longitudinal stent deformation. The major risk conferred from longitudinal stent deformation is stent thrombosis. For ostial disease, especially, operators must pay extreme caution of the guide positioning through stented segments. Similar caution should be taken in cases with deliberate "underexpansion" of the proximal segment of a long stent in a tapered vessel.

When longitudinal stent deformation is suspected, radiographic assessment of the stented segment with an image enhancement program should be done. Once confirmed, a small compliant balloon, followed by high-pressure inflation of a non-compliant balloon should be performed in order to ensure adequate expansion of the deformed stent struts and their apposition to the arterial wall. The use of IVUS/OCT to confirm appropriate expansion of the deformed stent is recommended to avoid further potential deformation.

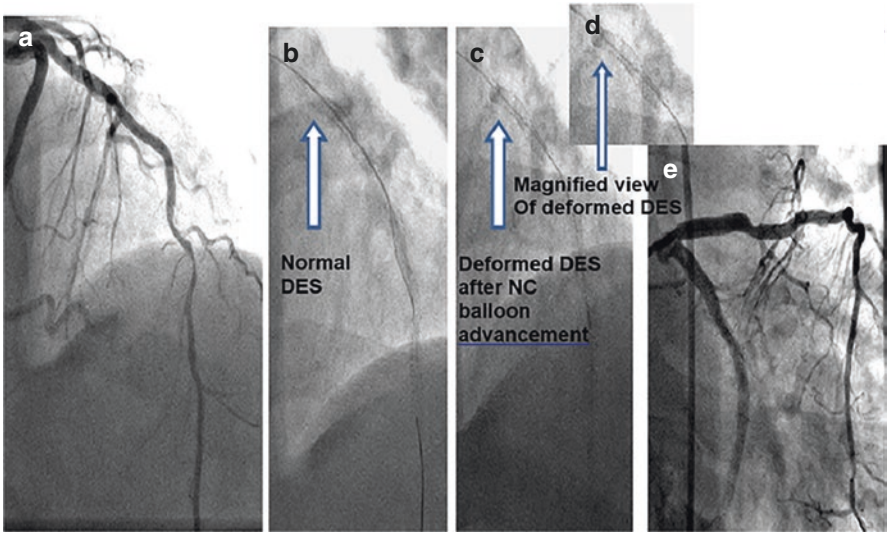

Fig. 24.7 Longitudinal Stent Deformation. Lesion noted in mLAD segment (**a**), and s/p DES placement (**b**). Proximal portion of stent demonstrating longitudinal stent deformation due to non-compliant balloon advancement seen under fluoroscopy (**c**, **d**), and confirmed with angiography (**e**)

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Part III

Special Procedures

Contrast-Induced Nephropathy Post Percutaneous Interventional Procedures

25

Hemal Bhatt, Lorenzo Azzalini, and Samin K. Sharma

Introduction

Contrast-induced nephropathy (CIN) is associated with increased morbidity, mortality, prolonged hospitalization and consequently increase healthcare costs. The pathophysiology of CIN is complex, and although contrast-induced kidney injury plays a central role, other compounding factors (such as hypotension, bleeding, and atheroembolic showers into the renal arteries) are also involved. Risk stratification prior to catheterization can help identify patients at high risk for CIN and to implement preventive strategies.

Definition

The incidence of CIN after cardiac catheterization has been reported as 3–15% [1–5]. Several definitions of CIN have been proposed. The most recent international consensus is represented by the Kidney Disease Improving Global Outcomes (KDIGO) statement, which defines CIN as any of the following [6].

- Rise in serum creatinine (SCr) by ≥0.3 or 0.5 mg/dl within 48 h [7].
- Rise in SCr to ≥1.5 times from baseline within 7 days.
- Urine volume <0.5 ml/kg/h for ≥6 h.
- Usually, creatinine levels peak at 3–5 days and return to normal in 2–4 weeks [7, 8].

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Epidemiology

Data from the National Cardiovascular Data Registry (NCDR) indicates that CIN complicates 7% of all PCIs in the USA. However, the incidence is much higher in patients with advanced chronic kidney disease (up to 27% in patients with an eGFR <30) and in subjects presenting with an acute coronary syndrome, heart failure, or cardiogenic shock. The incidence of need new for dialysis is 0.3% in all-comers but can increase up to 4% in patients with an eGFR <30 and in those with an unstable presentation [9].

Pathophysiology

The exact pathophysiology of CIN is not completely understood but is believed to be multifactorial. After a transient phase of vasodilation, contrast media induces prolonged arteriolar vasoconstriction, which creates the basis for reactive oxygen formation and ischemia of the outer medulla of the kidney, which is particularly prone to ischemic injury. At the same time, contrast media exerts a direct toxic effect on the tubular epithelium, leading to osmotic nephrosis. Besides contrast-induced acute kidney injury, other factors related to percutaneous coronary intervention also play a role: atheromatous embolization into the renal arteries due to catheter manipulation (particularly with femoral access), hypotension, and bleeding [4, 5]. Figure 25.1 summarizes the pathophysiology of CIN.

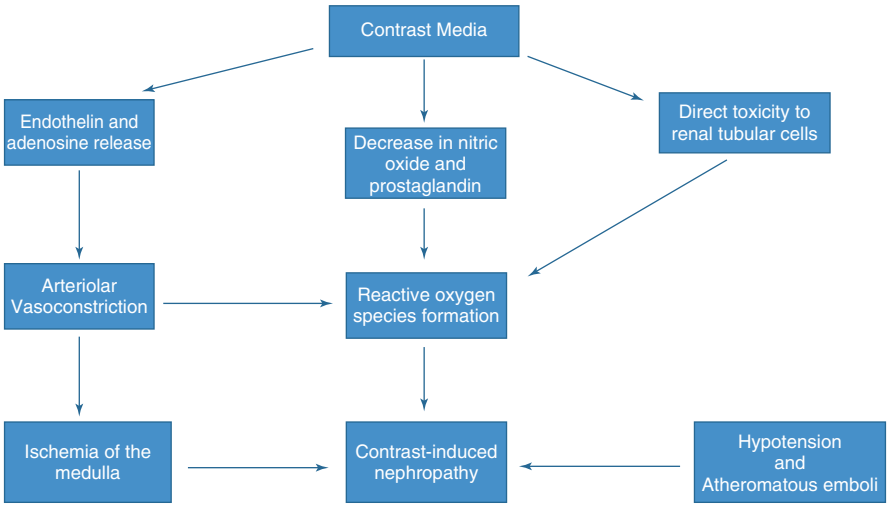

Fig. 25.1 Pathophysiology of contrast-induced nephropathy

Identification of High-Risk Patients

Once CIN occurs, treatment is mainly supportive. Identification of high-risk patients for CIN is therefore critical to establish preventative measures already at the pre-procedural stage [5]. Table 25.1 lists the risk factors for developing CIN.

- Risk assessment must be performed in all patients who are considered for cardiac catheterization.
- Risk models predicting the estimated risk of CIN after cardiac catheterization have been proposed by Mehran et al. and Gurm et al. and can assist in the identification of at-risk subjects [5, 10].
- In high-risk patients, do not perform ad hoc PCI after diagnostic angiogram: schedule elective PCI at least one week later.

Prevention

Figure 25.2 summarizes strategies supportive. Identification to prevent CIN.

- Minimizing contrast use during the procedure is vital [4].
  1. Use of iso osmolar contrast media like iodixanol (Visipaque™) or low-osmolar contrast media like iopamidol (Isovue™)..

Table 25.1 Risk factors for supportive. Identification developing CIN

Modifiable risk factors	Non-modifiable risk factors
First-generation hyperosmolar ionic contrast agents (rarely used)	Preexisting renal disease, CKD defined as a SCr >1.5 mg/dl or an eGFR of <60/ml/min
High contrast media dose	Diabetes
Nephrotoxic agents	Advanced age (>75)
Low hematocrit	Dehydration
Low serum albumin (<35 g/L)	Renal transplant
Intra-aortic balloon pump	Acute myocardial infarction
Hypotension	LVEF <40%
Hypovolemia	Advanced congestive heart failure and cardiogenic shock

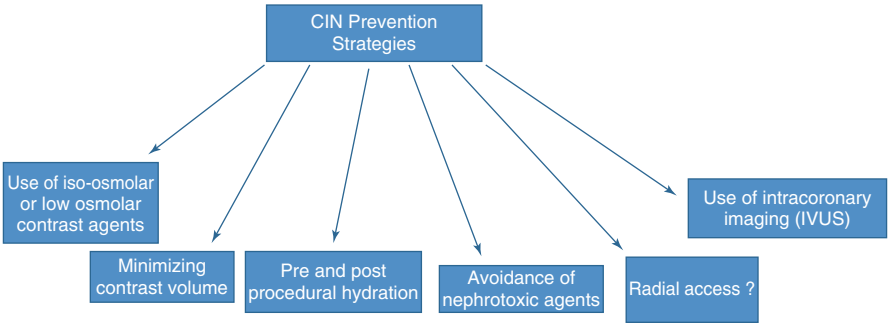

Fig. 25.2 CIN prevention strategies

2. Avoid taking test injections or using side-hole catheters: every drop of contrast media matters.
3. Use smaller French size catheters.
4. Allow for elimination of contrast from guiding catheter by back-bleeding or aspirating before entering the equipment.
5. Use additional guidewires to create a roadmap of the target vessel and its side branches or use dedicated software (e.g., Dynamic 3D Roadmap, Philips).
6. Extensive use of intracoronary imaging devices such as intravascular ultrasound or dextran-based optical coherence tomography to guide all steps of PCI: lesion assessment and preparation as well as stent sizing and optimization.
7. Use "dry cine" co-registration and intravascular ultrasound to guide stent placement.

- Avoid nephrotoxic agents:
  1. Nephrotoxic agents include metformin, nonsteroidal anti-inflammatory drugs (NSAID), and aminoglycoside antibiotics among others.
  2. There is insufficient evidence to support either continuation or suspension of diuretics, angiotensin-converting enzyme inhibitors, or angiotensin receptor blockers.
- Volume supplementation:
  1. Intravenous hydration with isotonic saline remains the cornerstone for prevention of CIN; however, in patients with cardiovascular disease, we prefer 0.45 NS.
  2. Liberal oral hydration 24 hr prior to the procedure may help.
  3. Individualize treatment by symptoms and volume status. To this extent, left ventricular end-diastolic pressure-guided hydration strategies have been successfully implemented, as they usually allow to maximize intravenous hydration volume in hypo- or euvolemic individuals [11].
  4. In the absence of acute decompensated heart failure, follow Tables 25.2 and 25.3.
- N-Acetylcysteine:
  1. Though no conclusive data exists regarding its benefit, we prescribe it as it has no harmful side effects and is inexpensive [12].
  2. 1200 mg orally prior to and q 12 h × 2 doses after cardiac catheterization.
- Use of radial compared with femoral access has been shown to be associated with decreased risk of CIN [13, 14].

Table 25.2 Pre-procedural hydration

	Start hydration with normal saline (0.9 NS) 3 h prior to procedure if outpatient and 12 h prior if inpatient
LVEF %	For all patients but especially if eGFR <60
>50	1–2 mL/kg/h
31–50	0.5 mL/kg/h
<30	0.3 mL/kg/h for 3 h only for all patients

Table 25.3 Intraprocedural and post-procedural hydration

	Continue hydration with normal saline (0.9 NS) during procedure and for 6–8 h post procedure if outpatient and 12 h if inpatient
LVEF %	For all patients but especially if eGFR <60. If eGFR >60, intravenous hydration can be given for a shorter duration with instructions for liberal discharge oral hydration
>50	1–2 mL/kg/h
<50	Measure LVEDP in the catheterization lab. Give a bolus of NS based on LVEDP
	<12: 500 ml bolus 12–18: 250 ml bolus
	>18: no bolus
31–50	Subsequent infusion of 0.5 mL/kg/h
<30	Subsequent infusion of 0.3 mL/kg/h

- The data regarding the benefit of automated contrast injector systems has been conflicting.
- A periprocedural course of high-dose potent statins (e.g., rosuvastatin 40 mg on admission followed by 20 mg daily) has been shown to decrease the risk of CIN in high-risk subjects [15].
- No conclusive data exists that ascorbic acid, prostaglandin E1, post-procedure diuretics, mannitol, atrial natriuretic peptide, dopamine, calcium channel blockers, theophylline, and fenoldopam are preventive.

Post-Procedure Monitoring and Treatment

The identification of high-risk individuals, aggressive volume repletion, avoidance of concurrent nephrotoxic drugs, use of low-osmolar or iso-osmolar contrast media, contrast-sparing strategies making ample use of intravascular imaging, and close post-procedural monitoring are vital in prevention, early recognition, and treatment of CIN.

- Check SCr at 24, 48, and ideally 72 hr post contrast media exposure.
- Hydration with 0.9% normal saline using the above strategies.
- Hold all nephrotoxic agents until renal function is established to be at baseline.
- Evaluate for other causes of acute kidney injury.
- Consider hemodialysis if a patient is oliguric or anuric as per nephrology recommendations.
- Patient may be discharged with close outpatient monitoring once the SCr starts returning down towards the normal baseline level.
- Outpatient follow-up with repeat SCr in 2–3 days.

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Intracoronary Stent Restenosis

26

Keisuke Yasumura, Annapoorna Kini, and Samin K. Sharma

Introduction

Despite contemporary drug-eluting stent (DES) technology, intracoronary stent restenosis (ISR) occurs in up to 10% of patients, increasing with intervention of complex lesions. The presentation of ISR is still challenging for optimal treatment [1].

Angiographic Definition and Classification

ISR is defined as ≥50% angiographic diameter stenosis within a coronary stent (intrastent) or 5 mm stent proximal and distal margins (in-segment). Mehran et al. proposed angiographic classification of bare metal stent (BMS) ISR, which is based on prognostic predictors of repeat revascularization for BMSs (Table 26.1) [2].

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Table 26.1 Angiographic restenosis and classification

Diameter stenosis ≥50%
Type I focal: ≤10mm in length
IA articulation or gap
IB margin
IC focal body
ID multifocal
Type II diffuse: >10mm intrastent
Type III proliferative: >10mm extending beyond the stent margins
Type IV total occlusion: Restenotic lesions with TIMI flow grade of 0

Table 26.2 Comparison of principal features of restenotic tissue after BMS and DES implantation

	BMS-ISR	DES-ISR
Mechanisms	Neointimal proliferation with vascular smooth muscle cells and extracellular matrix	Hypersensitivity to the polymer and the drug, local inflammation, and delayed healing
Angiographic morphology	Diffuse pattern more common	Focal pattern more common
OCT tissue properties	Homogeneous, high-signal band most common	Layered structure or heterogeneous most common
Time course of late luminal loss	Late loss maximal by 6–8 months	Ongoing late loss out to 5 years

Mechanisms

The mechanism of ISR is multifactorial, including biological, mechanical, and technical factors. There are significant differences between BMS-ISR and DES-ISR in terms of clinical presentation, pathogenesis, lesion morphology, and response to intervention [3] (Table 26.2).

Optical coherence tomography (OCT) study revealed that early DES-ISR was characterized by homogeneous neointimal hyperplasia, and late DES-ISR was neoatherosclerosis, which is with thin-cap fibroatheroma and lipid-rich neointima or with calcification, particularly in first-generation DES's [4, 5].

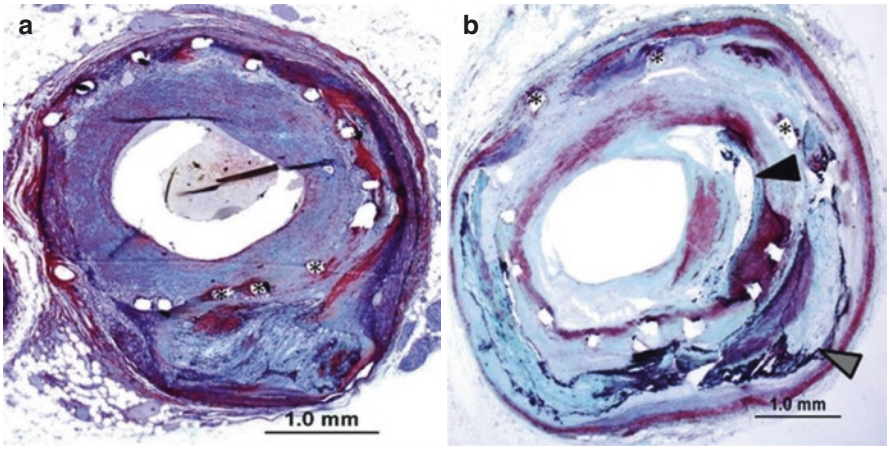

Fig. 26.1 Images of restenosis within (**a**) BMS and (**b**) DES, both implanted 5 years antermortem. In the BMS, the dominant pathology is smooth muscle cell-rich neointimal hyperplasia. In the DES, there is presence of neoatherosclerosis with formation of a necrotic core (black arrowheads) and calcification (gray arrowheads). Original table and figure modified for this publication. From: Fernando Alfonso, et al. Current Treatment of In-Stent Restenosis. J Am Coll Card. 2014; 63:2659–73. Table and Figure used with the permission of Springer All rights reserved

Pathological Images of ISR

Representative pathological images of ISR within BMS and DES are shown below (Fig. 26.1).

Role of Intravascular Imaging

Intravascular imaging is useful in ISR to evaluate the pattern and mechanisms of ISR, thereby providing guidance for treatment [3]. The frequent use of intravascular imaging during ISR treatment is recommended, especially in angiographic diffuse, proliferative, and occluded lesions [6]. Intravascular ultrasound (IVUS) is limited in its ability to evaluate neoatherosclerosis; OCT is the preferred imaging modality to detect neoatherosclerosis [7].

Representative OCT Images of ISR

Representative OCT images of ISR are shown below (Fig. 26.2).

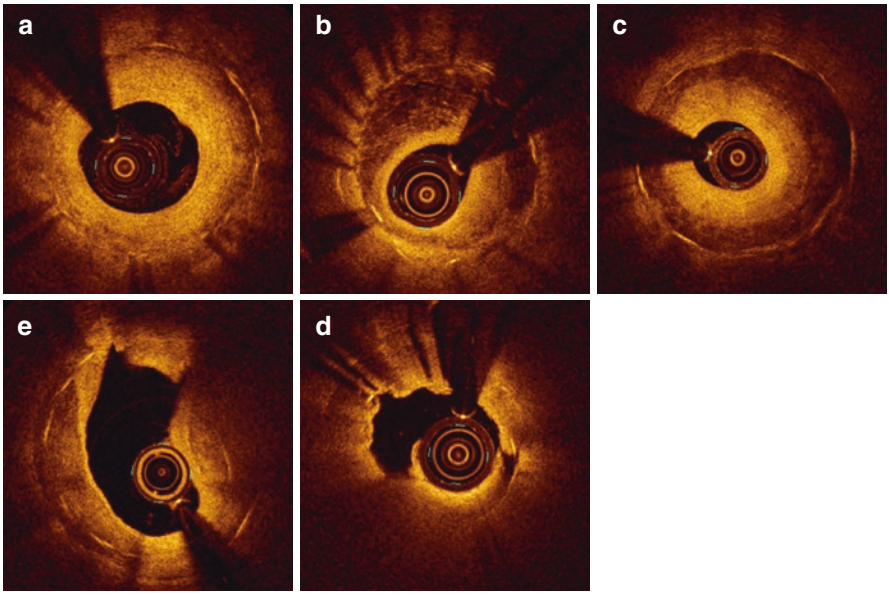

Fig. 26.2 Representative OCT Images of ISR. (**a**) Homogeneous neointima. This may represent neointimal hyperplasia with smooth muscle cell growth. (**b**) Heterogeneous neointima. This may represent proteoglycan-rich neointima or granulation tissue or fibrin deposition. (**c**) Layered pattern neointima. The relatively well-demarcated borders and the lack of attenuation. This may represent granulation tissue with overlying neointimal hyperplasia rich in smooth muscle cells. (**d**) Calcified neoatherosclerosis. In-stent signal poor region with sharply delineated borders. (**e**) In-stent plaque rupture from noncalcified neoatherosclerosis. Signal rich region close to the luminal surface accompanied by signal attenuation (Obscuring stent struts) with evidence of plaque rupture

Treatment for ISR

High-Pressure Noncompliant Balloons

- High-pressure noncompliant balloons can be used for mechanical ISR, such as stent underexpansion [8]. One of the limitations of balloon angioplasty is balloon slippage ("watermelon seeding" phenomenon) and that subacute tissue reintrusion back to the lumen tends to occur within minutes after the last balloon inflation [9].

Cutting and Scoring Balloons

- The lateral blades of a cutting and scoring balloon enable neointimal modification and anchor the balloon within the target lesion, preventing balloon slippage-related problems. The ISAR-DESIRE 4 randomized trial showed that in patients

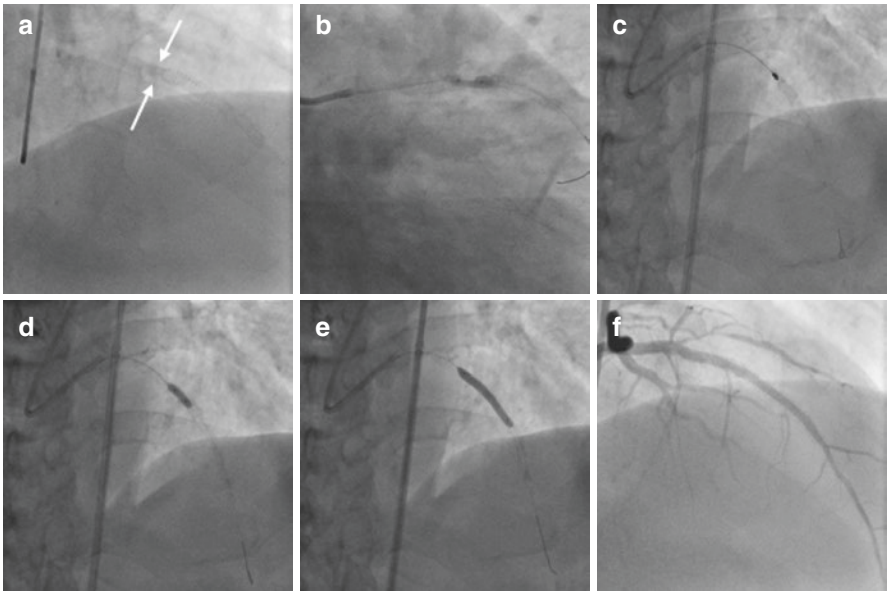

Fig. 26.3 (**a**) ISR due to underexpanded stent; (**b**) Undilated NC balloon; (**c**) Rotational atherectomy to ISR lesion; (**d**) Well-expanded NC balloon; (**e**) Re-DES implantation; (**f**) Final angiogram

with DES-ISR, neointimal modification with scoring balloon improves the antirestenotic efficacy of drug-coated balloon therapy as demonstrated by lower late lumen loss [10].

Debulking Techniques

- Both rotational atherectomy (RA) and excimer laser coronary atherectomy (ELCA) have been used for ISR to debulk the lesion and modify plaque compliance. Current data indicate that atherectomy techniques are best reserved for pretreatment of underexpanded stents that are resistant to high-pressure balloons or ISR of calcified lesions. Caution should be used for RA for ISR to prevent shaving metal particles or entrapping the burr within the underexpanded lesion [7]. Representative RA for ISR due to underexpanded stent case is shown below (Fig. 26.3).

Repeat DES

- Repeat stenting with DES has been established as the treatment of choice in DES-ISR. There is no consensus on whether the different drug type of stent is better. In patients with recurrent ISR, implantation of new DES would result in a

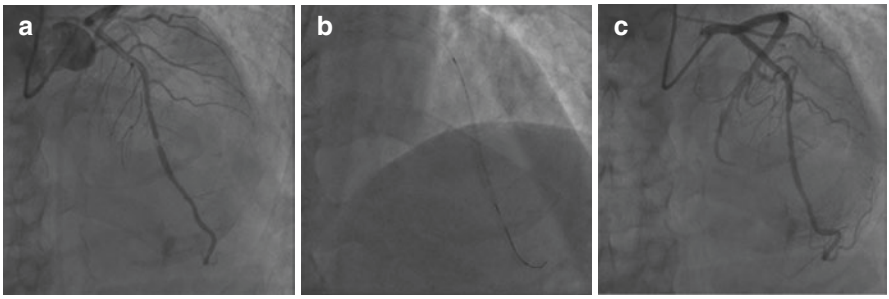

Fig. 26.4 (**a**) ISR with multiple stent layer; (**b**) IVBT for ISR lesion on the next day of cutting balloon angioplasty; (**c**) Final angiogram.

vessel with multiple layers and this patient cohort seems to be at a higher risk for additional recurrences.

Drug-Coated Balloon (DCB)

- DCBs, which is not commercially available for use in coronary arteries in the USA, inhibit neointimal proliferation and are recommended by European Society of Cardiology guidelines as a treatment option for ISR. Comparison of the results of DCBs and additional DESs for DES-ISR have been reported (3).

Intravascular Brachytherapy (IVBT)

- Brachytherapy inhibits neointimal formation within the stent, decreasing the proliferative tissue response [11]. VBT is primarily used for refractory ISR, and its use should be considered when multiple layers of stent are present. Treatment with VBT cannot be repeated during the procedure. After treatment with VBT, because of delayed endothelialization, patients should be maintained on a minimum of 3 years of antiplatelet therapy. Representative IVBT for an ISR case is shown below (Fig. 26.4).

New Classification for DES-ISR Based on Intracoronary Imaging

Waksman et al. proposed a new classification for the treatment of DES-ISR guided by intracoronary imaging [7] (Table 26.3).

Algorithm for the Treatment of DES Restenosis at Mount Sinai Hospital

Algorithm for the treatment of DES restenosis at Mount Sinai Hospital is shown below (Fig. 26.5).

Table 26.3 Waksman in-stent restenosis classification

Type	Definition		Treatment Options
I	Mechanical	Underexpansion (Type I A)	High-pressure balloon
		Stent fracture (Type I B)	DES
II	Biologic	Intimal hyperplasia (Type II A)	Balloon, DCB, DES, and VBT
		Neoatherosclerosis, noncalcified (Type II B)	DCB and DES
		Neoatherosclerosis, calcified (Type II C)	Scoring balloon, ELCA, and RA
III	Mixed pattern: Combined mechanical and biologic etiology		High-pressure balloon with DCB, DES, or VBT
IV	Chronic total occlusion		DCB or DES, VBT for multiple layers, CABG as needed
V	>2 layers of stent		Balloon, DCB, VBT, and CABG

CABG indicates coronary artery bypass graft; *DCB* drug-coated balloon; *DES* drug-eluting stent; *ELCA* excimer laser coronary atherectomy; *RA* rotational atherectomy; and *VBT* vascular brachytherapy

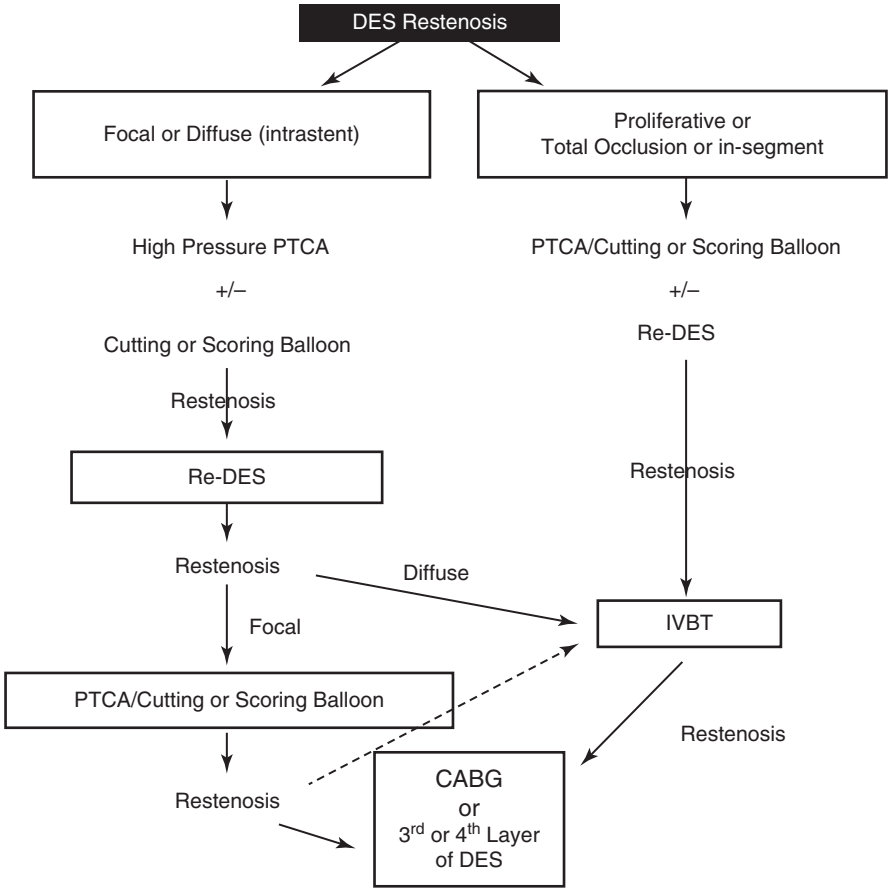

Fig. 26.5 Algorithm for the treatment of DES restenosis at Mount Sinai Hospital

Conclusion

Treatment of ISR remains a challenging clinical problem. Because of the various pathologies associated with ISR, there is not a universal treatment for all ISR. Intracoronary imaging provides unique insights into the underlying etiology of ISR, and new classification can guide treatment tailored to the specific lesion characteristics.

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Advanced Hemodynamic Support

27

Hemal Bhatt, Gurpreet S. Johal, and Gregory Serrao

Introduction

The advances in interventional cardiology have resulted in the shift towards PCI as opposed to CABG in high-risk complex lesions, especially in surgically non-amenable lesions. The use of LV assist devices has increased the odds of success and has decreased mortality, morbidity, and overall healthcare costs [1–4]. Cardiogenic shock complicates 5–8% of STEMI and is a major cause of in-hospital mortality up to 50% [5]. Unloading of the myocardium mechanically may limit infarct size, maintain end-organ perfusion, and decrease myocardial oxygen demand [6–8].

Commonly Used Advanced Hemodynamic Support

A good percutaneous circulatory support device should be placed without significant complications and provide an output of >2 L/min for hours to days without an external blood circuit. The two commonly used are Intra-Aortic Balloon Pump (IABP) (Fig. 27.1) and Impella 2.5 LP/CP systems (Abiomed). Tandem Heart is now infrequently used. Table 27.1 highlights the indications of hemodynamic support in PCI. Table 27.2 shows our selection algorithm for the type of hemodynamic support device.

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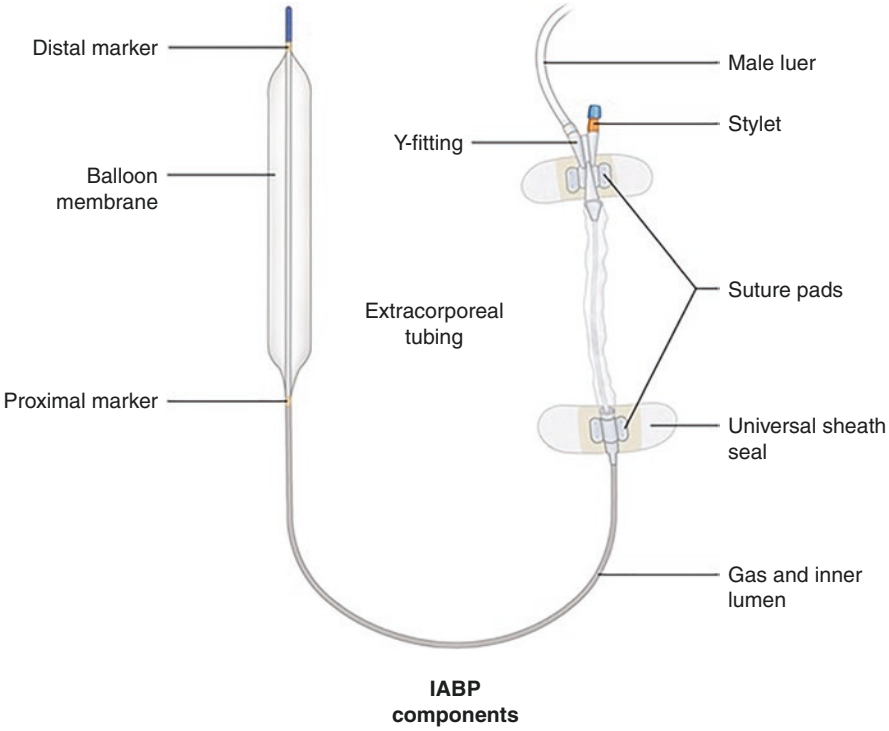


Fig. 27.1 Intra-aortic balloon pump (IABP)

Table 27.1 Indications for hemodynamic support in PCI

Indications
Cardiogenic shock
Severe LV dysfunction
Mechanical complications of an AMI
Complex coronary lesions (such as unprotected left main disease, complex multivessel disease, last remaining conduit vessel, and bypass graft disease)
As a bridge to further therapy:
Prophylaxis in patients with severe left main coronary artery stenosis
Intractable myocardial ischemia
Refractory heart failure
Intractable ventricular arrhythmia

Table 27.2 Algorithm for LV support device use

LVEF	Simple PCI	Complex PCI
>35%	No support	IABP
20–35%	IABP	Impella
<20%	Impella	Impella

Intra-Aortic Balloon Pump (IABP)

IABP is used in 30% of patients undergoing complex PCIs in the USA and requires a patient to have a certain level of residual left ventricular function to be effective. It is an over-the-wire balloon catheter with a fiber optic sensor for beat-to-beat adjustment and accuracy. It could be implanted with relative ease and low cost with a low complication rate.

Contraindications

- Severe aortic insufficiency.
- Aortic aneurysm.
- Aortic dissection.
- Limb ischemia.

Complications

- Table 27.3 details potential complications associated with IABP use.

Balloon Size

- 35 cm balloon if patient's height is <5 ft.
- 40 cm balloon if patient's height is between 5 and 6 ft (most commonly used).
- 50 cm balloon if patient's height is >6 ft.

Table 27.3 Potential complications of IABP

Access site complications	Aortic dissection	Limb ischemia	Thrombocytopenia
Bleeding	–	–	Check CBC daily
Infection	Prevent by inserting balloon over wire	Avoid by using the smallest sheath size and use the limb with the strongest pulse	Asses for HIT
Vascular complications	Assess daily for pain between shoulder blades	Use Xylocaine for spasm	Decrease or stop heparin
Compartment syndrome	Remove IABP and surgery for repair if needed	Bypass graft for the affected extremity if needed and change insertion site to the opposite limb	Transfuse platelets as needed

Access

- Obtain optimal femoral access or use preexisting access to place the 7 Fr or 8 Fr sheath that comes with a balloon pump kit.
- Sheathless in morbid obesity or scarring of the groin.
- Use a shallow angle of insertion <45°.
- Advance catheter in small steps of less than 1 in. at a time to avoid kinking.
- If kinking is suspected, reposition by pulling back half an inch.

Balloon Preparation Before Insertion

- Open the tray and do not remove the balloon from the T handle sheath.
- Attach the one-way valve to male Luer fitting of IABP catheter (Fig. 27.2) and a 60 cc syringe.
- Negative pressure on the balloon (about 30 cc) × 2 with the balloon tip remaining inside the sheath and detach the syringe only leaving the one-way valve in place of the IABP (Fig. 27.3).
- Remove stylet, and flush the inner lumen with 3–5 cc of flush solution. Elevate flush bag at least 3 ft above transducer and connect to pressure @ 300 mmHg to maintain a 3 cc/h continuous flow through the inner lumen of the IABP (Fig. 27.4).

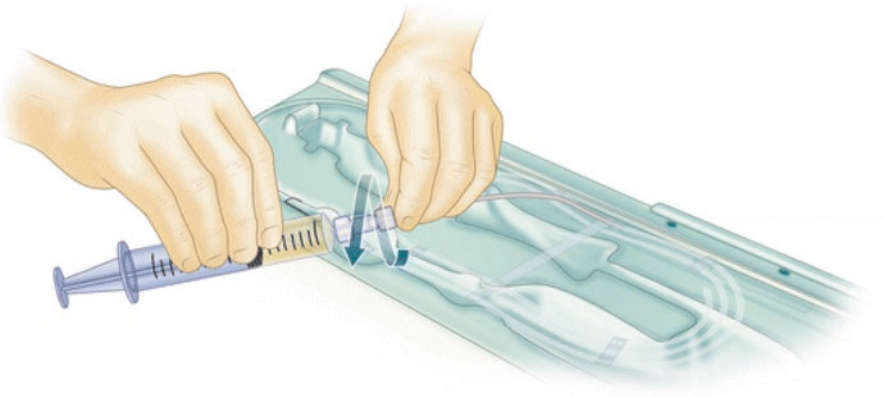


Fig. 27.2 Attach the one-way valve to the main Luer fitting of the IABP catheter and a 60 cc syringe

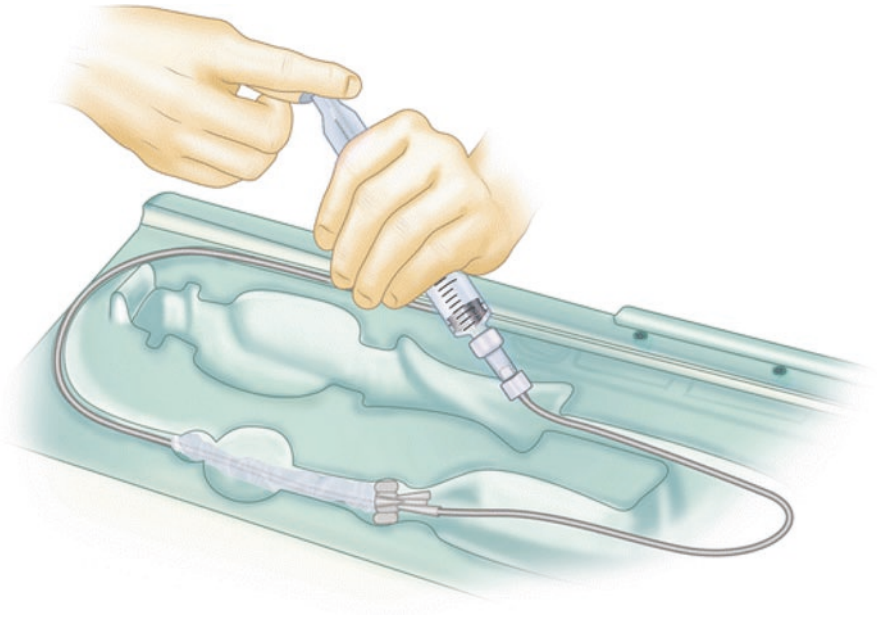


Fig. 27.3 Negative pressure on the balloon

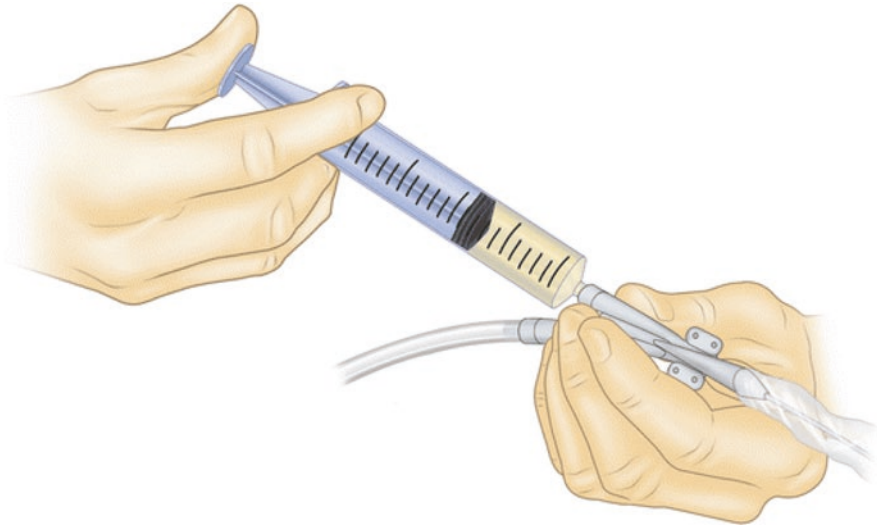


Fig. 27.4 Flush the inner lumen

Balloon Insertion

- Place an 8Fr catheter in the femoral artery with needle insertion at <45° angle (Fig. 27.5).
- Remove IABP by pulling straight from the T handle. Do not dip, wipe, or handle the membrane before insertion (Fig. 27.6).
- Advance 0.018″ wire to the level of the aortic arch. Using small short movements, advance the balloon over the wire until the distal tip is at the level of the carina. The proximal tip should be above the renal arteries.

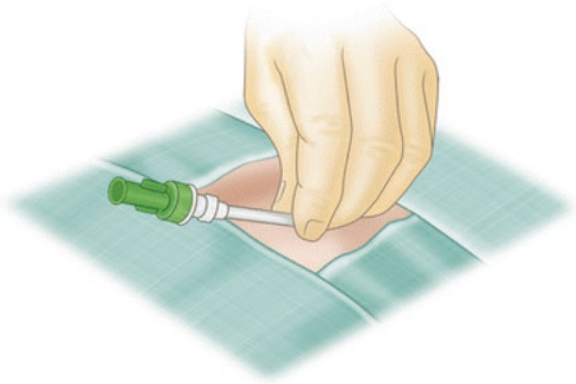


Fig. 27.5 Insert 8 Fr sheath

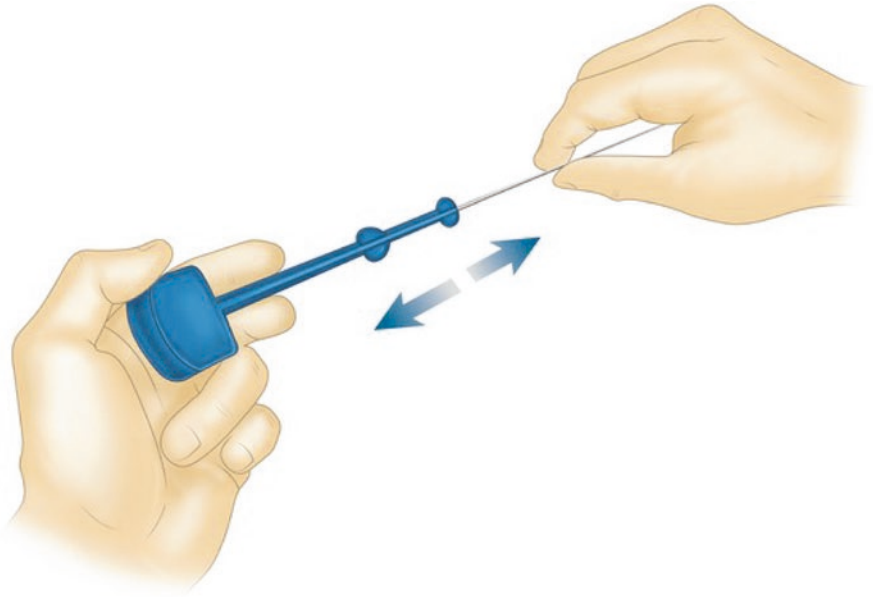


Fig. 27.6 Remove IABP by pulling straight

- Advance sheath seal as far as possible into the hub of the sheath and secure the IABP catheter to the patient's leg using STATLOCK® or sutures (Fig. 27.7).
- Remove guidewire, aspirate back 3–5 cc of blood from the inner lumen and flush with another 5 cc of flush solution (Fig. 27.8).

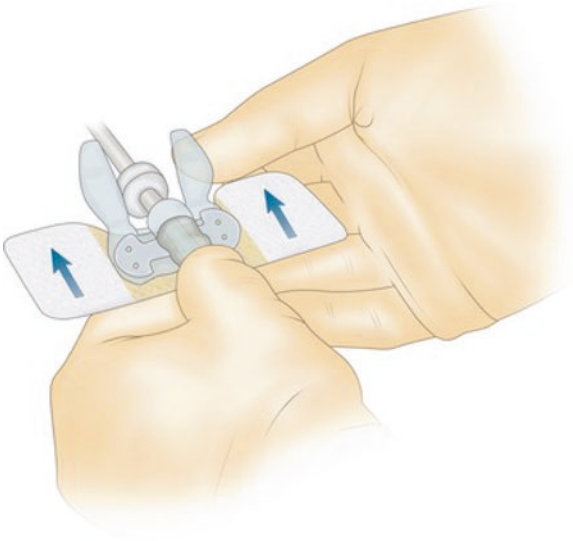


Fig. 27.7 Secure IABP with STATLOCK® or sutures

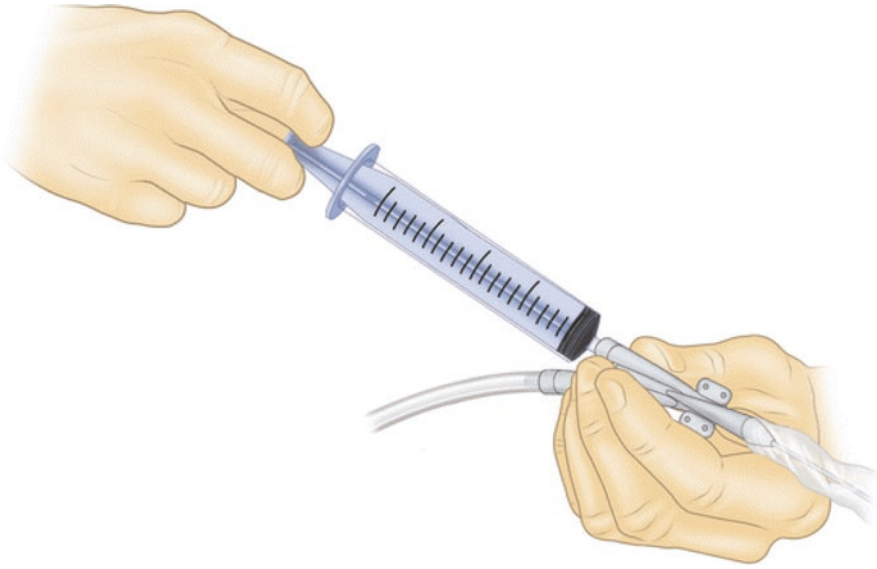


Fig. 27.8 Flush catheter

- Remove one-way valve from the IABP catheter and attach male Luer to female Luer fitting of Pneumatic Module of IABP (Fig. 27.9). Hand over fiber optic cable, if IABP is fiber optic, to the technician (Fig. 27.10).
- Press the "start button."
- Select mode:

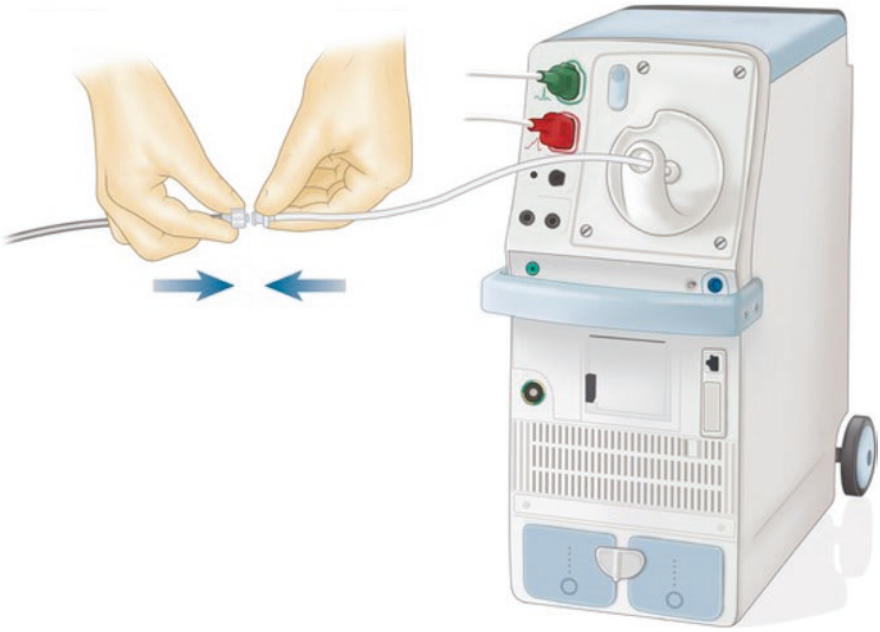


Fig. 27.9 Attach male Luer to female Luer fitting of Pneumatic Module of IABP

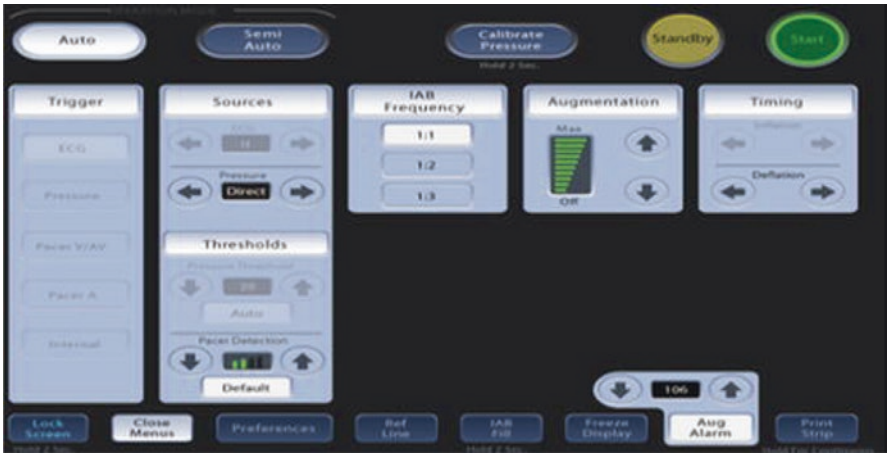


Fig. 27.10 Press "start button," select mode, and set the frequency at 1:1

Fig. 27.11 IABP frequency confirmed on the console

  - Auto Mode: Automatically selects the most appropriate lead and trigger and sets the inflation and deflation timing.
  - Asynchronous: Rate of 80/min and only used when a patient has no cardiac output
  - Select frequency: at 1:1 (Fig. 27.10).
- Confirm fluoroscopic balloon inflation and balloon waveforms on the console (Fig. 27.11).

Troubleshooting

- Inflation occurs at the dicrotic notch as a sharp "V" and ideally, diastolic augmentation rises above systole (Fig. 27.12). Deflation occurs just before systolic ejection and results in a reduction in assisted end-diastolic and end-systolic pressure (Fig. 27.12).
- See Fig. 27.13 for variations with heart rate and rhythm.

Timing Errors: Adjust the Onset of Inflation/Deflation

- Early inflation before aortic valve closure or dicrotic notch.
- Diastolic augmentation encroaches onto systole and causes AI and an increase in LVEDP and MVO_2 demand (Fig. 27.14).
- Early deflation during the diastolic phase.
- Deflation is seen as a sharp drop following diastolic augmentation (Fig. 27.15) and causes suboptimal coronary perfusion, retrograde coronary and carotid blood flow, and increased MVO_2 demand.
- Late inflation markedly after the dicrotic notch.
- This causes suboptimal diastolic augmentation and coronary artery perfusion (Fig. 27.16).

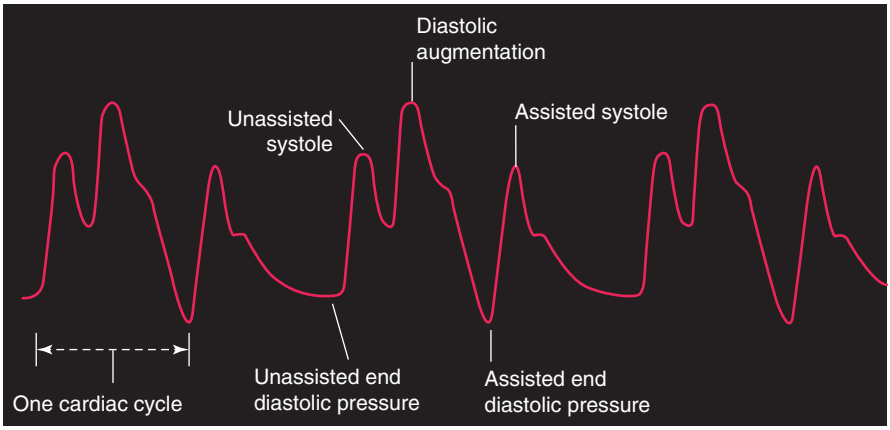


Fig. 27.12 Inflation of the IABP occurs at the dicrotic notch and deflation occurs just before systolic ejection

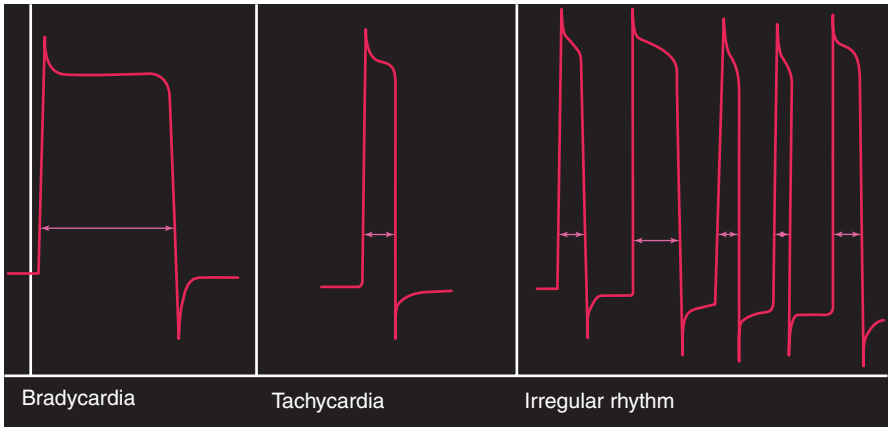


Fig. 27.13 Normal variations in balloon pressure waveform

- Late deflation after the aortic valve has opened.
- Afterload reduction is essentially absent (Fig. 27.17) and MVO_2 consumption is increased can impede left ventricular ejection.

Gas Loss

- If blood observed:
  - Stop pumping and remove IABP.
- If blood not observed
  - Check if connections are tight, perform an autofill, and press start.
- Fig. 27.18 shows the hemodynamic changes in the setting of gas loss.

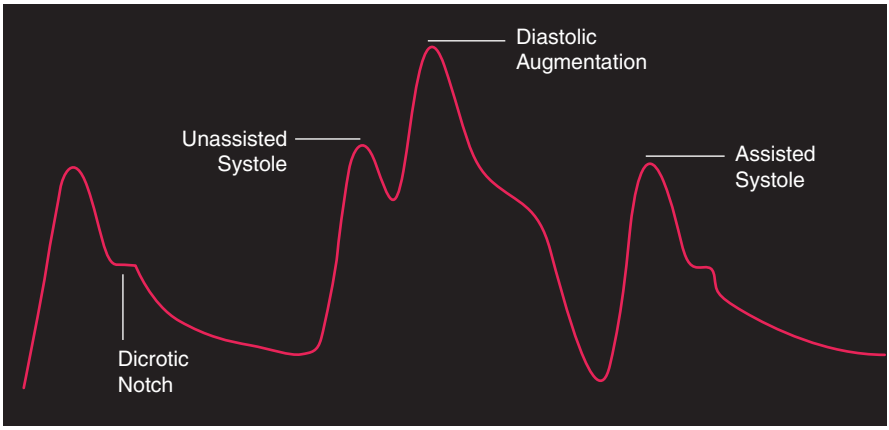


Fig. 27.14 Early IABP inflation before aortic valve closure or the dicrotic notch

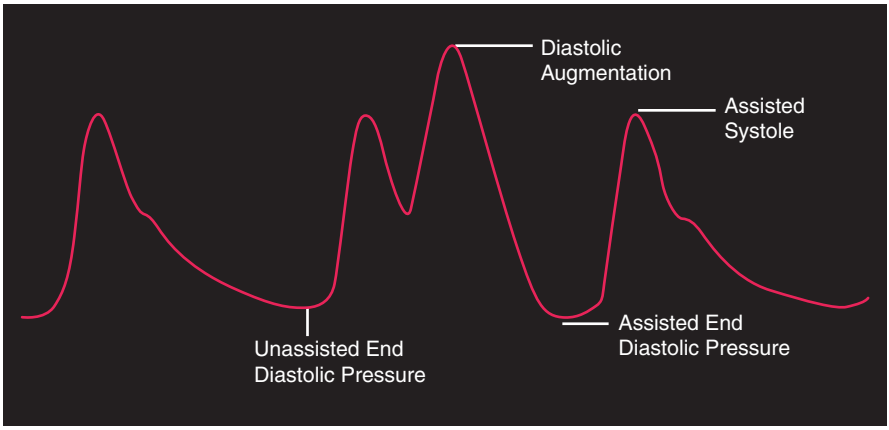


Fig. 27.15 Early IABP deflation during the diastolic phase

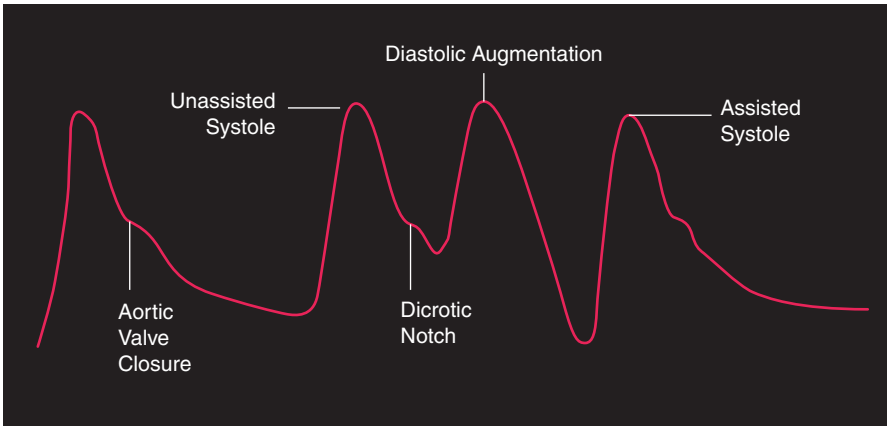


Fig. 27.16 Late inflation markedly after dicrotic notch

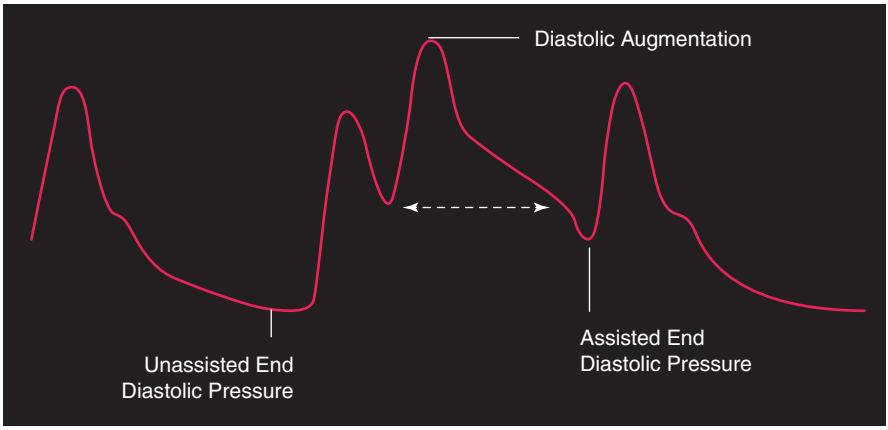


Fig. 27.17 Late deflation after the aortic valve has opened

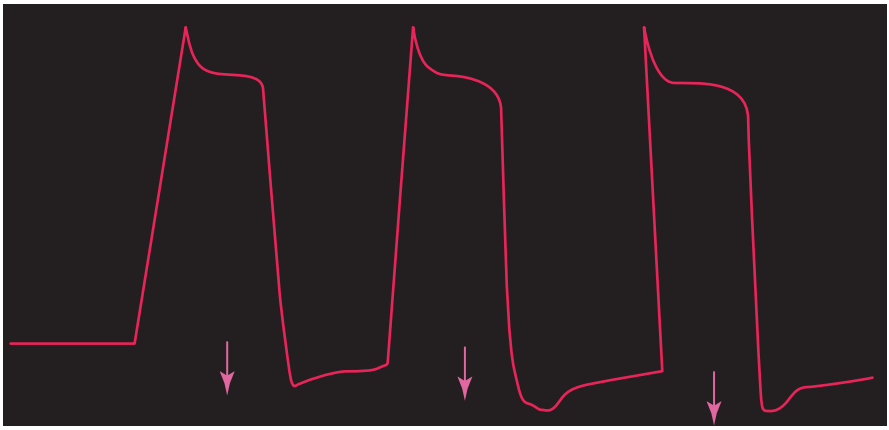


Fig. 27.18 Gas loss

Catheter Restriction

- Restriction in IABP or tubing
  - Relieve restriction.
- Membrane not unfolded
  - Try to manually inflate/deflate.
- IABP remains in the sheath
  - Make sure IABP is in position.
- Figure 27.19 shows the hemodynamic changes in the setting of catheter restriction.

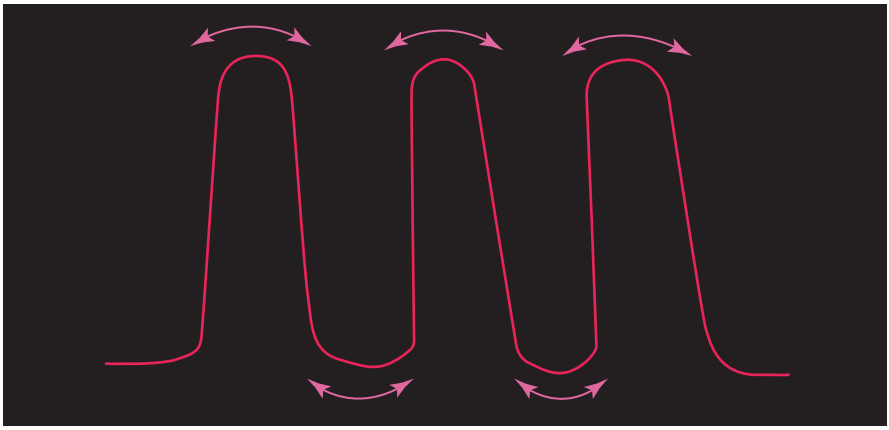


Fig. 27.19 Catheter restriction

Impella

Catheter-based, impeller-driven, axial flow pump which pumps blood directly from the left ventricle into the ascending aorta, contributing a cardiac output of up to 2.5 L/min for Impella 2.5 and 4 L/min for Impella CP.

Contraindications

- Mural thrombus in the left ventricle.
- Mechanical aortic valve or severe aortic stenosis (aortic valve area ≤ 1.5 cm^2 in PROTECT I and II trials; ≤ 0.6 cm^2 as per ABIOMED) [9–11].
- Moderate to severe aortic insufficiency (echocardiographic grade > 2+).
- Severe peripheral arterial disease or extreme tortuosity.
- Blood dyscrasia or coagulopathy predisposing to increased bleeding risk ($\leq$ 75000/mm^3 or INR ≥ 2.0 or fibrinogen ≤ 1.5 g/L).

Preimplantation

- Iliofemoral angiography to rule out severe PVD.
- Deploy two preclose sutures (see vascular closure devices).
- Press the power side button of the console for 3 sec. The console automatically performs a system check (Fig. 27.20).
- Open the Impella kit under sterile conditions (Fig. 27.21).
- Press "menu" and "start case."
- Auto prime (performed by the nonsterile technician).

Fig. 27.20 Press power button for 3 s

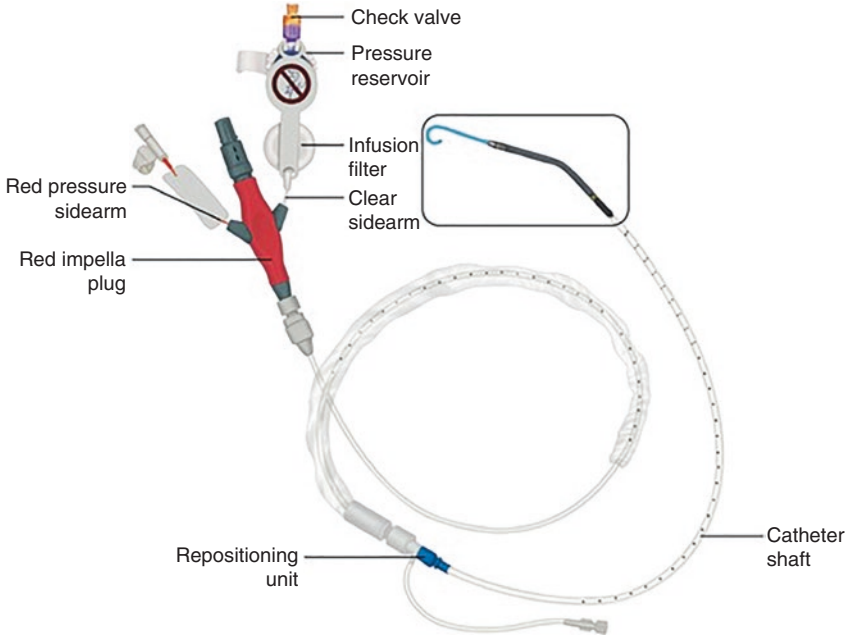


Fig. 27.21 Open Impella kit

- Connect the purge fluid spike of the purge cassette to the purge fluid bag (500 ml of 20% dextrose + heparin 50 U/ml) (Fig. 27.22).
- Press open the purge cassette door on the left side of the console, and snap the purge cassette into the slot, then slide the purge pressure transmitter to the right till you hear a snap (Fig. 27.23).
- The controller automatically starts to prime the purge cassette.

• Auto-detect
- Connect the black end of the connector cable to the red Impella catheter (Fig. 27.24).
- Snap the clear plastic clip on the sidearm to the connector cable (Fig.27.25).
- Hand the white end of the white connector cable to the technician to connect to the console with a click as shown. The controller automatically recognizes the catheter type (Fig. 27.26).

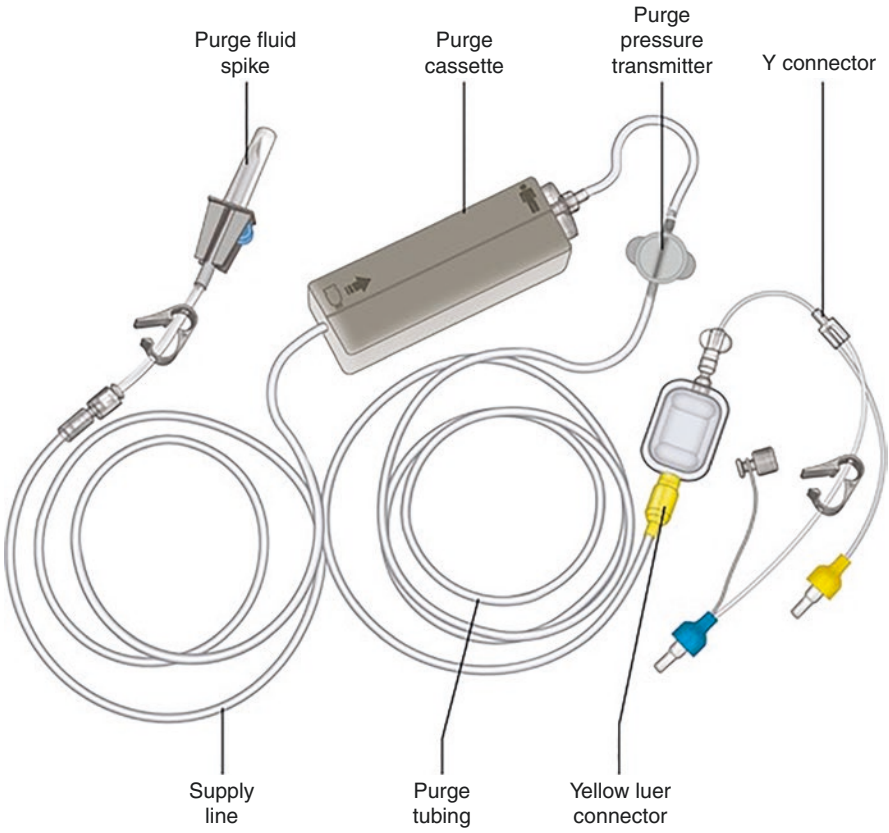


Fig. 27.22 Connect purge fluid spike to the fluid

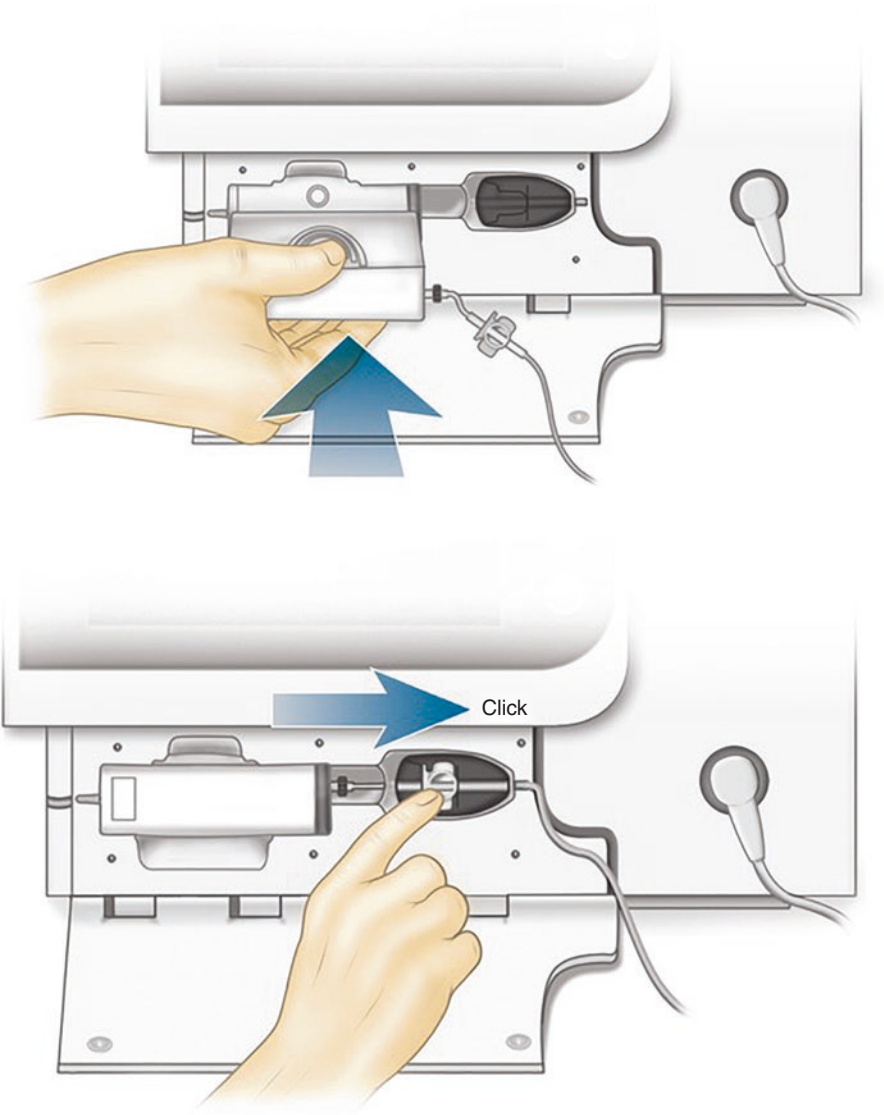


Fig. 27.23 Snap purge cassette into the slot

Fig. 27.24 Connect connector cable to Impella

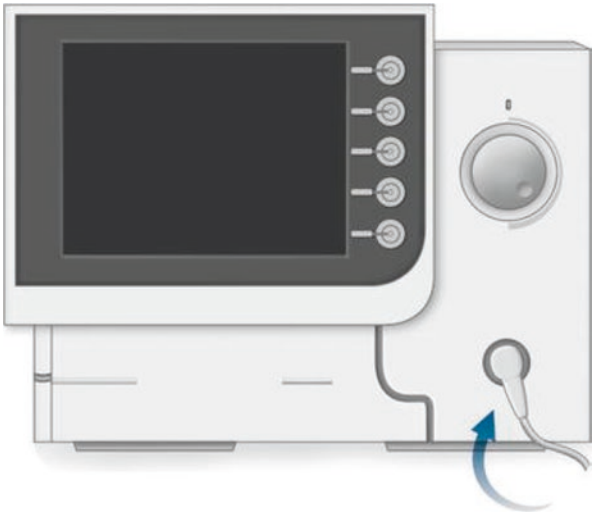


Fig. 27.25 Snap clear plastic clip on the sidearm

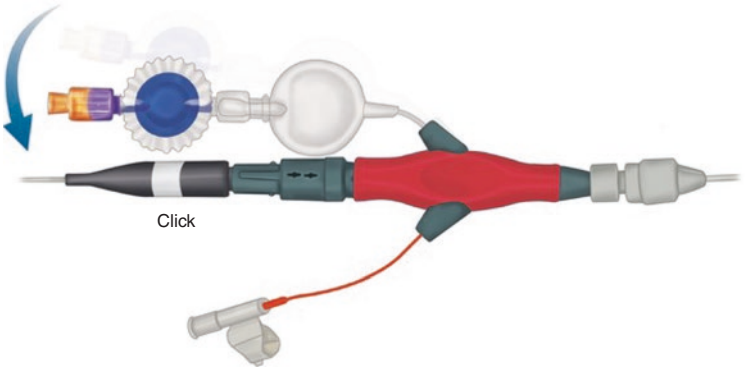


Fig. 27.26 Connect cable to console

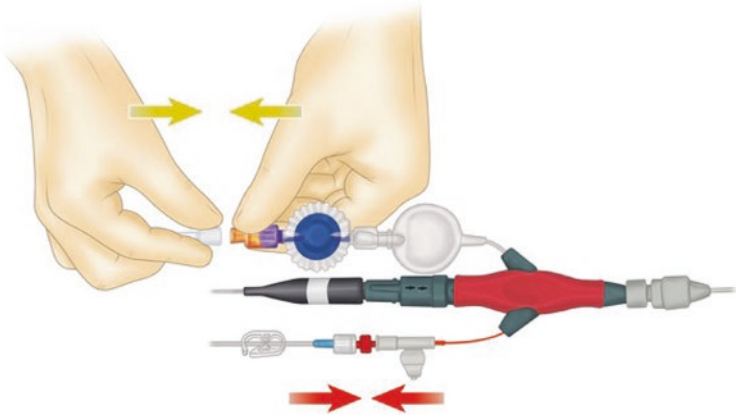


Fig. 27.27 Connect Impella to purge system

- Auto de-air
  - Connect the red port of the purge system to the red port of the Impella catheter and the yellow port of the system to the yellow port of the Impella catheter (Fig. 27.27). Ensure connections are tight.
  - Squeeze the white flush valve for 10 s until the controller beeps and fluid exits the Impella catheter (Fig. 27.28). The screen will show "Catheter is ready to insert." Select "default" for purge fluid values.

Implantation

- Insert a 7 F introducer/catheter and remove the introducer. Administer heparin to achieve an ACT of 250–300 s or bivalirudin to achieve an ACT of >300 s.
- After successive dilations with 8 Fr, 10 Fr, and 12 Fr dilators (Fig. 27.29), upgrade to a 13 Fr (Impella 2.5) or 14 Fr (Impella CP) peel away catheter/dilator by supporting the shaft of the introducer (Fig. 27.30).

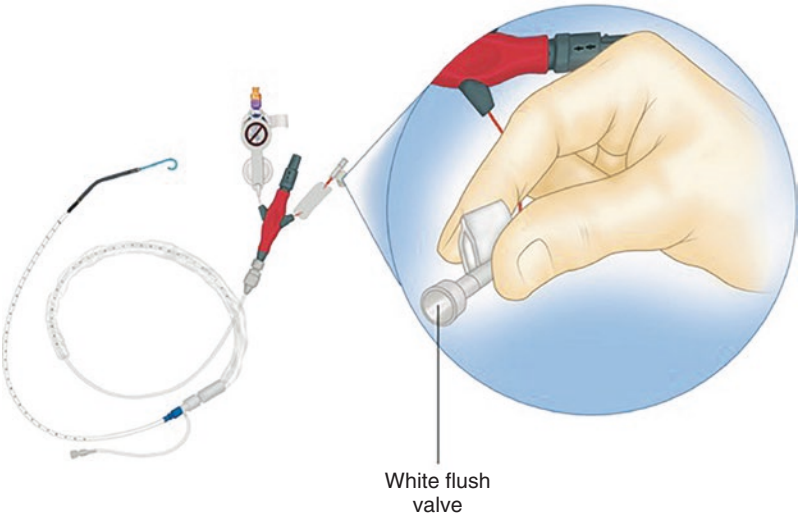


Fig. 27.28 Squeeze white valve for 10 s until controller beeps and fluid exits the Impella

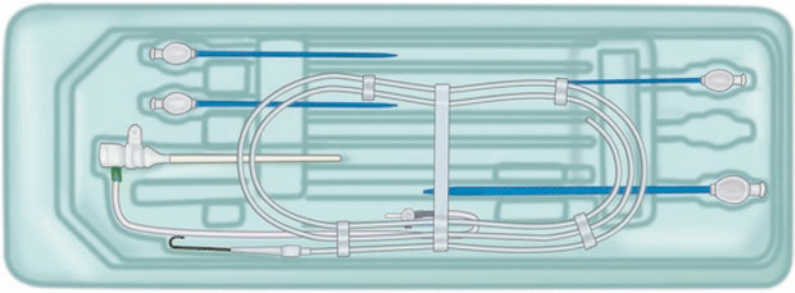
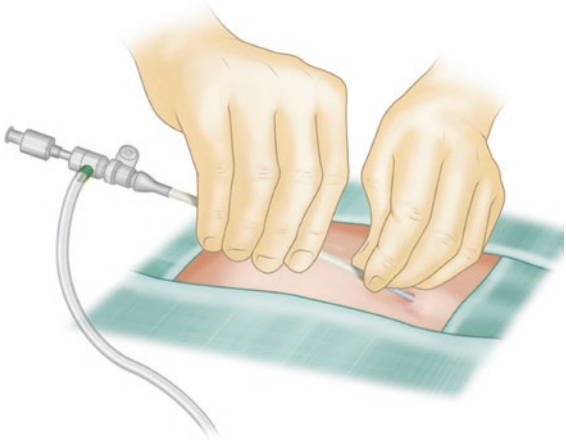


Fig. 27.29 Successively dilate to 12 Fr

Fig. 27.30 Upgrade to a 13Fr catheter/dilator



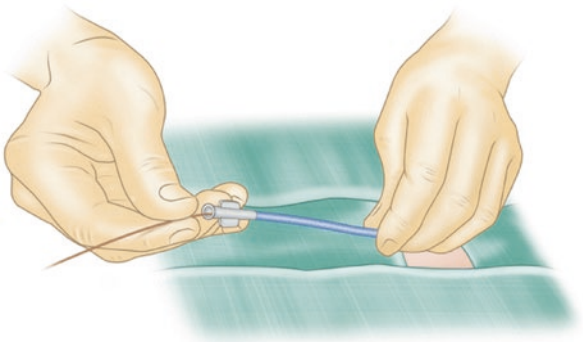


Fig. 27.31 Insert 0.035″ guidewire into LV

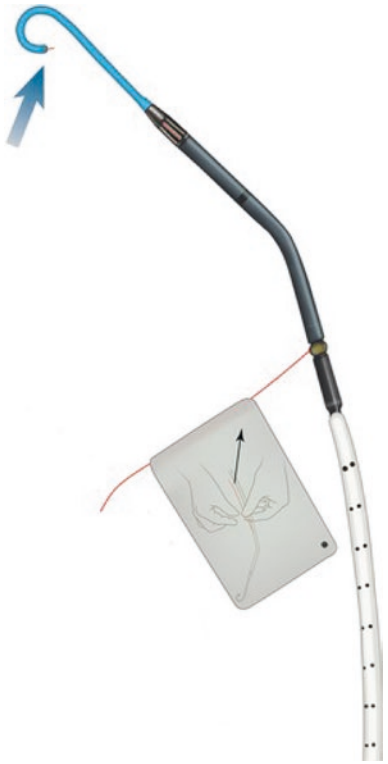


Fig. 27.32 EasyGuide Red lumen

- Remove the 13 or 14 Fr dilator and insert a 6 Fr diagnostic catheter (Judkins Right or Multipurpose with no side holes) over a 0.035″ guidewire into the left ventricle (Fig. 27.31).
- Exchange the 0.035 wire for a 0.018 guidewire and advance it until the floppy end and 3–4 cm of the stiffer part are visible in the left ventricle. Remove the 6 Fr diagnostic catheter.
- Backload the blue pigtail section on a 0.018 guidewire using preassembled EasyGuide Red lumen until it exits near the label (Fig. 27.32).

- Remove EasyGuide by gently pulling the label while holding the Impella® catheter (Fig. 27.33).
- Keep the wire parallel to the cannula and advance the catheter in small increments to avoid bending the cannula (Fig. 27.34).
- Advance the catheter into the middle of LV, without coiling the guidewire, under fluoroscopy to 4 cm below the AV annulus free from mitral valve chordae. Ensure that the Impella is positioned across the aortic valve with the pigtail and inlet portion in the left ventricle and the outlet and motor portion in the aorta. Align catheter against the lesser curvature of the aorta (Fig. 27.35).

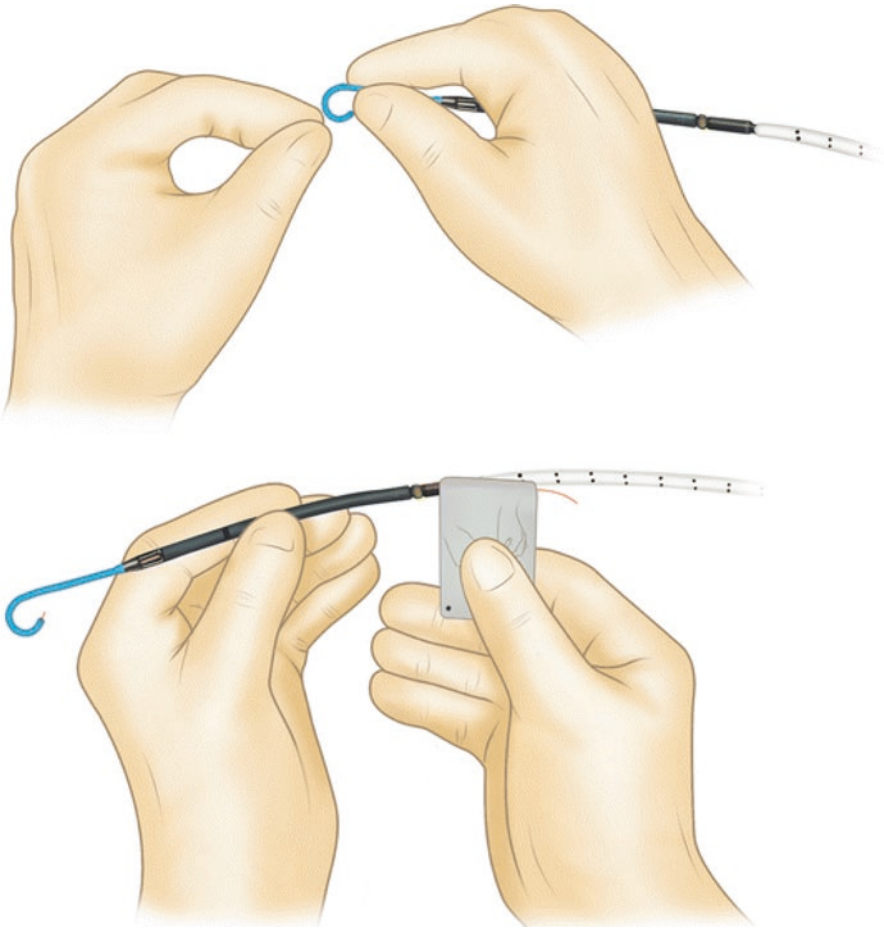


Fig. 27.33 Remove EasyGuide by gently pulling the label while holding the Impella

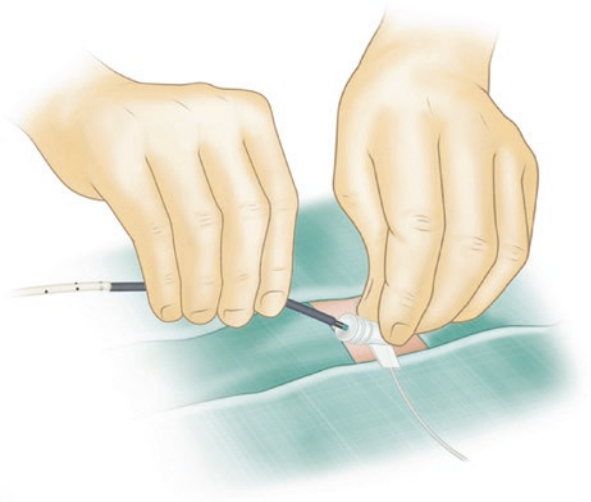


Fig. 27.34 Advance the catheter in small increments to avoid bending the cannula

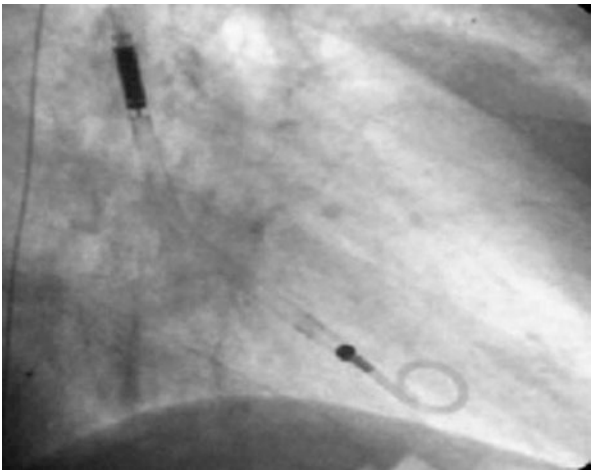


Fig. 27.35 Advance the catheter into the middle of the LV

- Remove any excess slack. Confirm that an aortic waveform is displayed on the Impella Console (Fig. 27.36).
- At the end of the procedure, the Impella catheter can be safely removed from the LV.

Troubleshooting

- If the Impella® catheter advances too far into the LV and the controller displays a ventricular waveform (Fig. 27.37), pull the catheter back until an aortic waveform is present. As soon as the aortic waveform appears, pull the catheter back

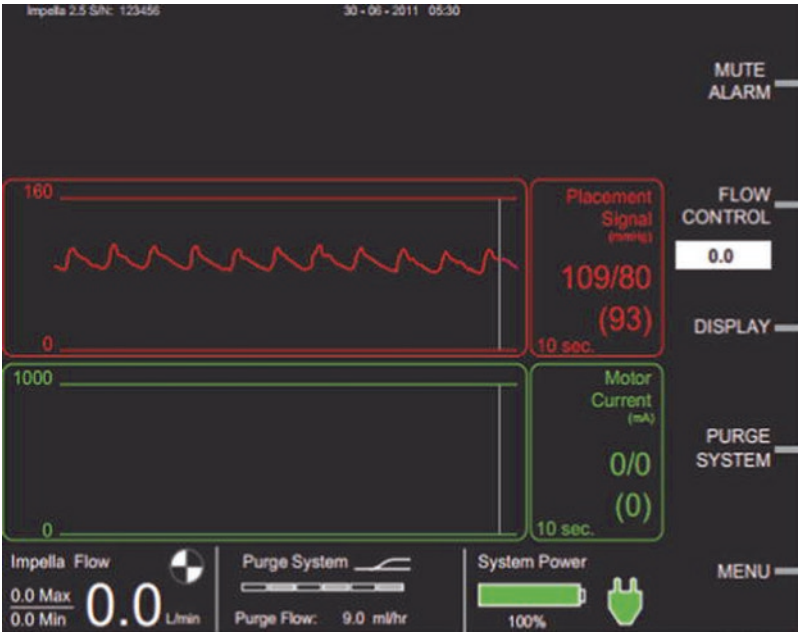


Fig. 27.36 An Aortic waveform is displayed on the Impella Console

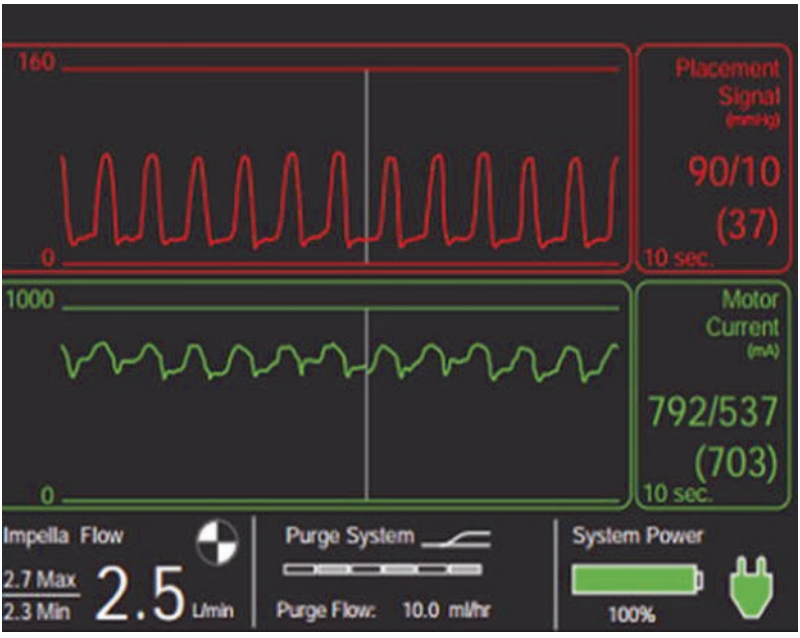


Fig. 27.37 If the Impella advances too far into the LV, the controller will display a ventricular waveform

an additional 4 cm (the distance between adjacent markings on the catheter is 1 cm).

Potential Complications

- Access site complications.
- Bleeding complications.
- Displacement of the pump back into the aorta can also occur, but the addition of the pigtail catheter tip minimizes the displacement potential (see troubleshooting above).
- Hemolysis, as measured by free hemoglobin, resulting in higher rates of blood transfusion [12].

Extracorporeal Membrane Oxygenation (ECMO)

Extracorporeal membrane oxygenation (ECMO) can provide oxygenation, carbon dioxide removal, and perfusion support for days to weeks in patients needing cardiopulmonary support. The ECMO circuit requires vascular access, a blood pump, and an oxygenator. Vascular access may be veno-venous or veno-arterial depending on the nature of physiologic support. Veno-venous (VV) ECMO is used for respiratory support and provides no hemodynamic support. Veno-arterial (VA) ECMO is placed in patients who need cardiopulmonary support and provides both respiratory and hemodynamic support. It requires both arterial and venous cannulation, and blood is drained from the right atrium and returned to peripheral circulation using femoral or axillary arteries. Blood can be returned into central circulation using aortic cannulation [13–17].

Indications

- Cardiogenic shock.
- Bridge to transplant.
- Support for high-risk percutaneous coronary intervention.
- Severe cardiomyopathy.
- Respiratory failure.

Contraindications

- Severe aortic regurgitation.
- Malignancy.
- Prolonged CPR without adequate tissue perfusion.
- Unwitnessed cardiac arrest.

Complications

- Access site bleeding.
- Vascular injury.
- Central vein or intracardiac thrombosis.
- Cerebral hypoxia.
- Device thrombosis.

Cannula Size and Access Site Considerations

- Fem-Fem ECMO requires selection of one long cannula (50–55 cm) capable of reaching the right atrium and another 25 cm cannula capable of reaching the central circulation.
- A multistage cannula (multiple access points) vs a single-stage cannula (single access point) could be used. The single-stage cannula is preferred for the return limb of the circuit and the multistage cannula is preferred for the drainage line as it provides maximal emptying.
- When selecting cannula size, one method is to calculate the patient's full cardiac output based on body surface area and then select cannulas, which provide this flow using the information provided by the manufacturer.
- The other method is to estimate the largest French size cannula capable of being passed through the vessel by measuring the vessel diameter (D) and calculating circumference:
  - Circumference: $\pi D = 3.1 \times D$ = maximum size of a cannula that could be used. If $\geq 2/3$ of the vessel is occluded by the cannula, then venous stasis can lead to deep vein thrombosis, lower limb venous congestion, and ischemic injury [16].

Steps of Implantation

- V-V ECMO: Single access (Avalon catheter (13–31 Fr) is a dual lumen catheter that matches the body's flow by draining venous blood from superior and inferior vena cava and infusing blood in the right atrium (Figs. 27.38 and 27.39).
  - Obtain venous access and advance guidewires.
  - Administer heparin anticoagulation.
  - Place the dual-line catheter through venous access.
  - Back bleed the lines to eliminate air or clot.
  - Connect these lines "wet to wet" to a primed ECMO circuit.
  - Start flow through the ECMO circuit.
- V-V ECMO: dual access
  - Obtain dual venous access (femoral and right internal jugular veins) (Fig. 27.40).
  - The remaining steps are the same as V-V ECMO (as above).

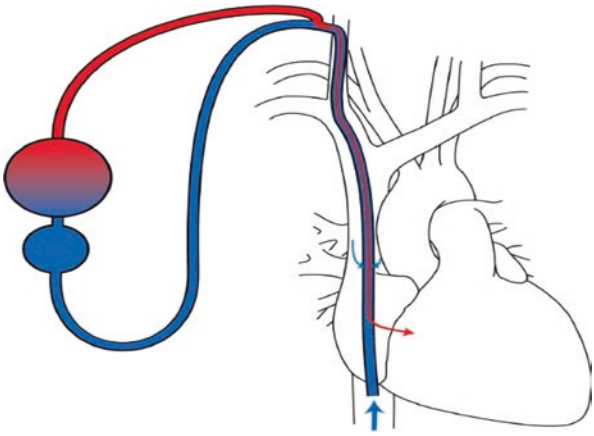


Fig. 27.38 V-V ECMO: single cannula approach [13]

Fig. 27.39 Avalon (Getinge, Gothenburg Sweden) catheter Bi-caval dual lumen catheter [17]

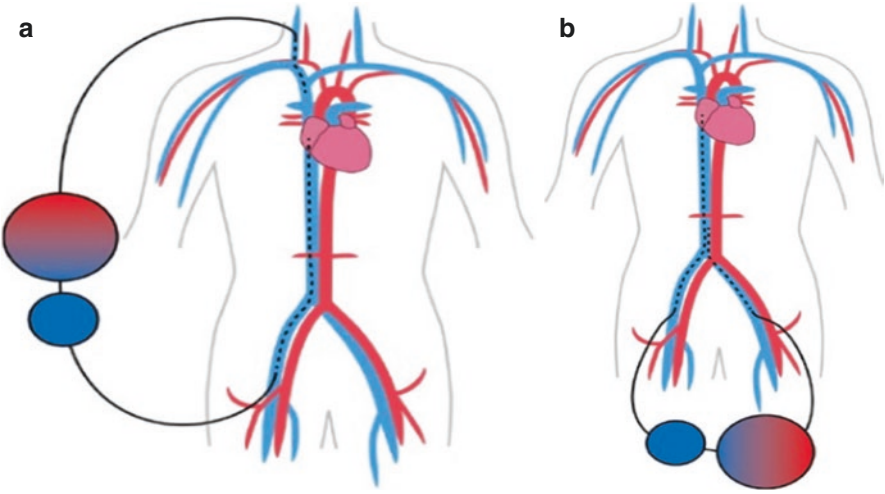


Fig. 27.40 V-V ECMO: two cannulation approach (**A**) femoral vein (for drainage) and right internal jugular for perfusion, (**B**) both femoral veins are used for drainage and perfusion [13]

- V-A ECMO:
  - Obtain femoral venous access and place a long cannula into the right atrium.
  - Obtain arterial access in the femoral, axillary, or internal jugular artery (Fig. 27.41).
  - The remaining steps are the same as V-A ECMO (as above).

RV Impella

The Impella RP (Abiomed Inc., Danvers, MA, USA) is an axial flow pump with a 22 F electric motor and outflow cannula, mounted on a thin and flexible 11 F catheter. Impella RP is designed to assist the right ventricle for up to 14 days in patients who develop acute right heart failure and can be used concurrently with other LVADs. Impella RP could provide flow up to 4 L/min at 33,000 rpm from the inferior vena cava to the pulmonary artery [18, 19].

Recently, the FDA issued a notification to healthcare providers that RV Impella post-approval study showed higher mortality than premarket clinical study, but the patients in the post-approval study were more likely to be in cardiogenic shock for > 48 hours, experienced a cardiac arrest, or suffered an ischemic neurologic event before getting Impella RP system [20].

Fig. 27.42 depicts the Impella RP placement and Fig. 27.43 depicts the Impella RP catheter.

Contraindications

- Mural thrombus in the right atrium.

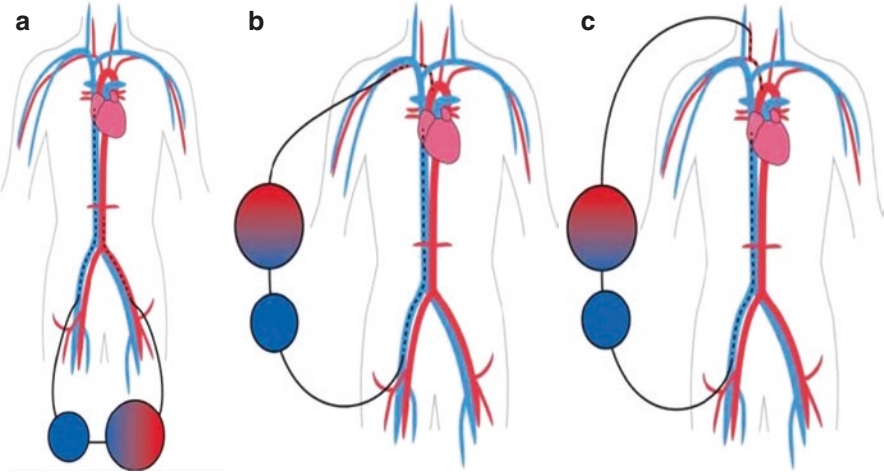


Fig. 27.41 Peripheral V-A ECMO cannulation approach: femoral vein (for drainage), (**A**) femoral, (**B**) axillary, (**C**) carotid, artery are used for perfusion [13]

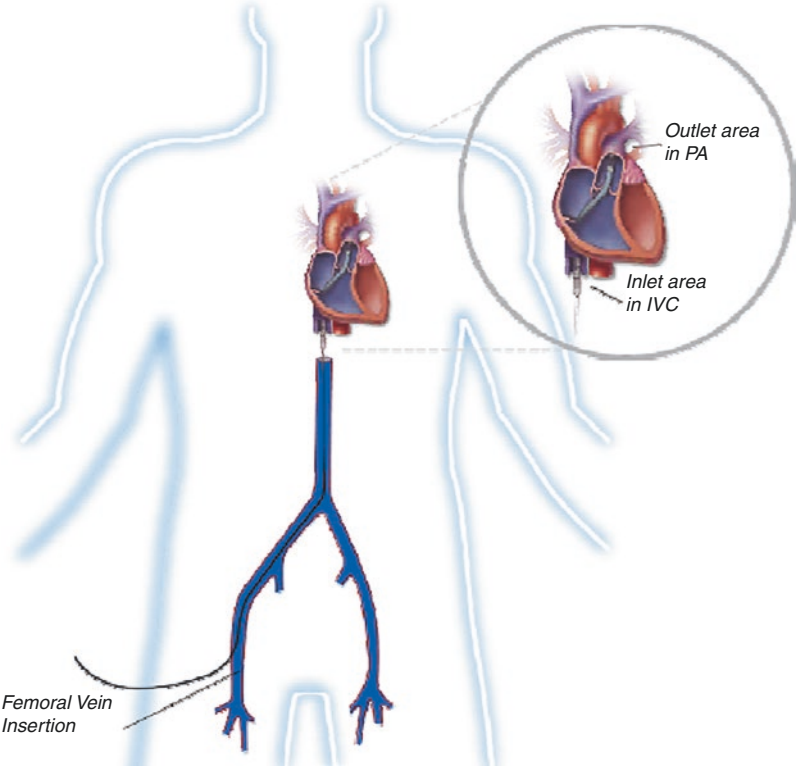


Fig. 27.42 RP Impella placement [20]

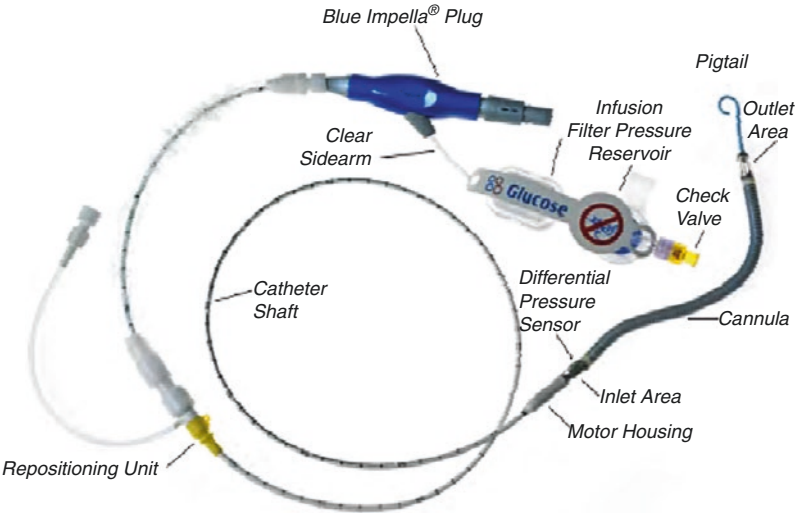


Fig. 27.43 Impella RP catheter [20]

- Mechanical valves, severe valvular stenosis or regurgitation.
- Presence of vena cava filter.
- Disorder of pulmonary artery that would preclude placement of Impella.

Steps of Implantation

- Obtain access in the femoral vein.
- Insert a 5–8 Fr introducer over the 0.035-inch guidewire to pre-dilate the vessel.
- Remove the 5–8 Fr introducer over the 0.035-inch guidewire. Insert the 8 Fr, 12 Fr, 16 Fr, and 20 Fr dilators sequentially, as needed. Remove the 20 Fr dilator and insert the 23 Fr introducer with a dilator. While inserting the 23 Fr introducer, hold the shaft of the introducer to advance it into the vein.
- Administer heparin. When ACT is at least 250 seconds, remove the 23 Fr dilator.
- Insert a 5–6 Fr diagnostic catheter or a flow-directed balloon-tipped catheter into the 23 Fr introducer and advance it over a guidewire into the left (preferred) or right pulmonary artery.
- Remove the 0.035-inch diagnostic guidewire, leaving the diagnostic or balloon-tipped catheter in the pulmonary artery. Form a curve or bend on the 0.025 inch Platinum Plus or similar stiff, 260 cm placement guidewire and then insert it.
- Advance the placement guidewire into the pulmonary artery, avoiding deep insertion into the most distal pulmonary artery.
- Remove the diagnostic or balloon-tipped catheter.
- Wet the cannula with sterile water and backload the catheter onto the placement guidewire. Advance the guidewire into the Impella RP Catheter and stabilize the cannula between the fingers. This prevents pinching of the outlet area. The guidewire must exit the inlet area on the inner radius of the cannula and align with the straight black line on the catheter. The guidewire must exit the inlet area on the inner radius of the cannula and align with the straight black line on the catheter. Advance the catheter through the hemostatic valve into the femoral vein and along the placement guidewire using a fixed-wire technique.
- Follow the catheter under fluoroscopy, and rotate the catheter as it enters the right ventricle to direct the cannula tip upward and across the pulmonary valve. Position the outlet area of the cannula approximately 4 cm past the pulmonary valve annulus.
- Remove the placement guidewire.
- Confirm position with fluoroscopy [18].

Complications

- Hemorrhage, valvular or vessel injury, perforation, arrhythmia.
- Arrhythmia.
- Insertion site infection.

TandemHeart

Tandem Heart (Cardiac Assist, Inc.), a percutaneous ventricular assist device is a left atrium to a femoral artery bypass system that provides hemodynamic support. The device reduces preload, augments cardiac output up to 4–5 L/min, and ultimately decreases myocardial work and oxygen demand. The device can be used as a bridge to transplant or surgical left ventricular assist device, left ventricular support in cardiogenic shock, and to maintain hemodynamic support in high-risk percutaneous coronary interventions (Fig. 27.44).

The system comprises of the following:

- 21 Fr venous "transseptal" inflow cannula with 14 side holes and an end hold.
  - Extracts oxygenated blood from the left atrium and delivers it into a continuous flow centrifugal blood pump.
  - The placement of the inflow cannula into the left atrium requires transseptal puncture under TEE guidance.

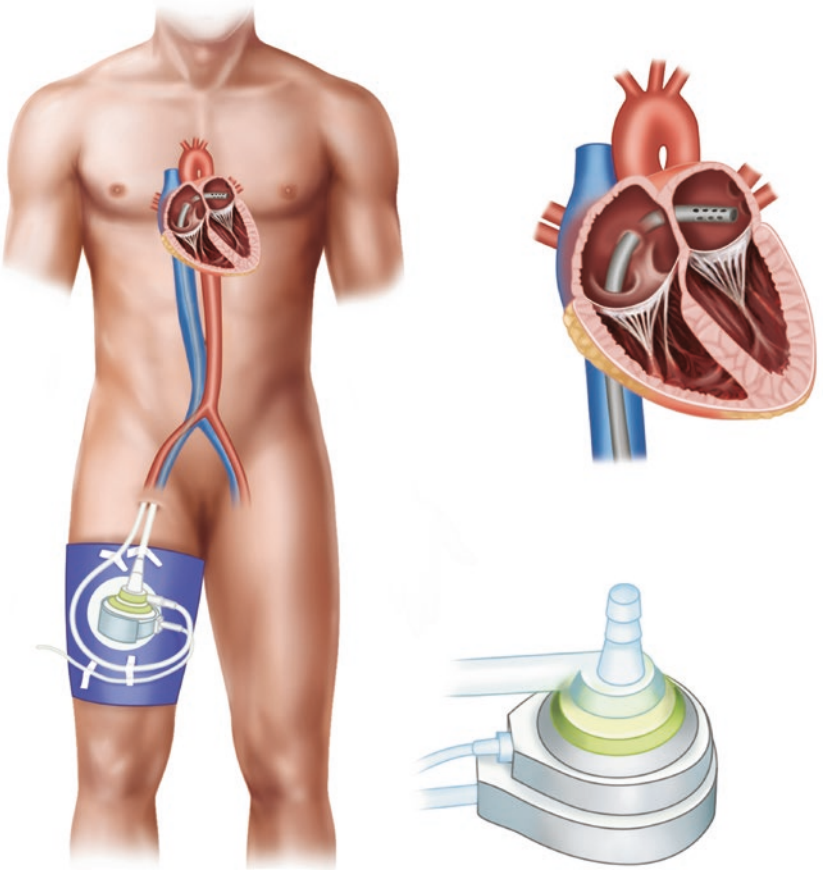


Fig. 27.44 Tandem heart device and centrifugal pump [23]

- Continuous flow centrifugal blood pump.
  - The pump is connected to a 15–17 F arterial infusion cannula.
- 15–17 Fr arterial infusion cannula.
  - Typically placed in the femoral artery and are required to pump blood from the LA to the right femoral artery.
  - Alternatively, two 12 Fr arterial perfusion catheters to pump blood into the right and left femoral arteries can be used instead of a single 15–17 Fr catheter in the right femoral artery.
- The Tandem heart pump comprises of a:
- Unique lubrication system, which feeds a nominal 10 cc/h of heparinized saline to cool the motor. The device requires systemic anticoagulation to prevent thrombosis and embolism [21–23].
- Pressure transducer that monitors the infusion pressure and identifies any disruption in the infusion line.
- In-line air bubble detector that monitors for the presence of air in the infusion line.

Contraindications

- Ventricular septal defect.
- Aortic insufficiency.
- Severe PVD.

Steps of Implantation

- Iliofemoral angiography to rule out severe PVD.
- Femoral artery access and preclosure with the Perclose™ device (see VCD Devices); upsize to a 15–17 Fr arterial sheath.
- Transseptal puncture via a femoral vein under fluoroscopic guidance using the Brockenbrough needle and a Mullins sheath is performed and its position in the LA is confirmed.
- Unfractionated heparin to achieve an ACT >400 s.
- Exchange Mullins sheath for the 21 Fr TH transseptal cannula with the 14 Fr obturator over the 0.038 in. J-tip 260 cm Amplatz Super Stiff guidewire and confirm its position (ensure all side holes of the TH are in the LA) by injecting dye and assessing the blood oxygen saturation level.
- The obturator and the wire are then removed and clamps applied for temporary homeostasis. Suture the peripheral end of the cannula to the skin of the patient's thigh and clamp it.
- Place the 15 Fr arterial perfusion cannula of the TH device with the distal end of the cannula lying above the aortic bifurcation. Suture the peripheral end to the patient's thigh and clamp it.
- De-air the extracorporeal system and attach the TH cannula to the inflow port of the centrifugal blood pump and femoral arterial cannula to the outflow conduit of the TH pump in the standard wet-to-wet fashion with Tygon tubing. Connect the power supply to the microprocessor-based controller.

- Connect the pump to the TH controller and adjust the speed to provide a cardiac output of 2.5–3.0 L/min.

Complications

- Puncture/rupture of the aortic root, coronary sinus, or posterior free wall of the right atrium.
- Thromboembolism—Unfractionated heparin to maintain an activated clotting time of 400 s during insertion and 250–300 s during support is mandatory.
- Hypothermia.
- Access site complications.

Protek Duo

Protek duo is a dual lumen cannula system that contains two lumens within a 29 or 31 F system. One lumen connects to an inflow cannula, with multiple holes, with access to the right atrium, whereas, the other lumen connects to an outflow cannula with access to the main pulmonary artery. The inflow cannula drains blood from the right atrium into a centrifugal pump, which delivers blood into the pulmonary artery bypassing the right ventricle. Hence, single venous access via an internal jugular approach is required for device placement (Fig. 27.45). Protek duo, in addition to providing right ventricular support, provides oxygenated blood directly into the pulmonary artery [24–26].

Contraindications

- Valvular stenosis (Table 27.4).
- Bleeding diathesis.

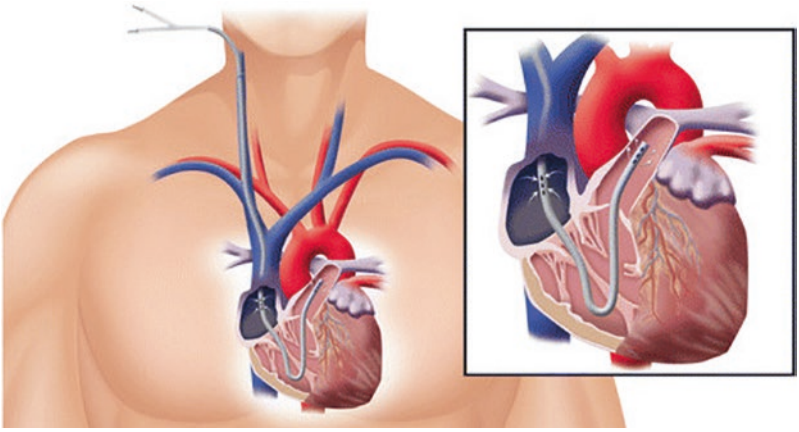


Fig. 27.45 Protek Duo dual lumen cannula. Published with permission from Kazui T et al [26]

Table 27.4 Summarizes an overview of the above hemodynamic support devices [27]

	IABP	Impella 2.5	Impella CP	RV Impella	V-A ECMO	V-V ECMO	Tandem Heart	Protek Duo
Vessel access	Arterial	Arterial	Arterial	Venous	Venous and arterial	Venous	Venous and Arterial	Venous
Fr Size	7–8	13	14	23	14 Arterial 21 Venous	21 Venous 21 Venous	15 Arterial 21 Venous	29
Support chamber	LV	LV	LV	RV	LV/RV	none	LV	RV
Oxygenation	No	No	No	No	Yes	Yes	No	Yes
Flow rate	< 0.5	< 2.5	<4	4	<7	<7	<5	<5
Preload	No change	Decreased	Decreases	No change	Decreases	Decreases	Decreases	No change
Afterload	Decreases	Decreases	Decreases	Decreases in RV	Increases	No change	Increases	Decreases in RV
Maximum implant duration	14 days	7–10 days	7–10 days	7–10 days	4 weeks	4 weeks	2–3 weeks	2–3 weeks
Coronary perfusion	Increases	Increases	Increases	No change	No change	No change	No change	No change
Affected by arrhythmias	Yes	No	No	No	No	No	No	No

Complications

- Bleeding and vascular injury.
- Right ventricular injury.
- Valvular injury.
- Thromboembolism.

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28 Aortic Valve Interventions: Balloon Aortic Valvuloplasty and Transcatheter Aortic Valve Replacement

Parasuram Krishnamoorthy, Nagendra Boopathy Senguttuvan, Samin K. Sharma, and Annapoorna Kini

Introduction

This chapter explains steps of aortic valvuloplasty and transcatheter aortic valve replacement.

Balloon Aortic Valvuloplasty (BAV) (Fig. 28.1).

Balloon Aortic Valvuloplasty (BAV)

Balloon aortic valvuloplasty (BAV) is restricted only to few clinical indications as mentioned in Table 28.1 from the 2014 ACC/AHA guideline for the management of patients with valvular heart disease [1]. A recent study of 3691 patients from the National Readmission Database showed that about 40% of BAV patients undergo subsequent TAVR mostly within 90 days. In-hospital outcomes of TAVR in these patients were comparable with propensity-score matched patients who underwent TAVR without prior BAV. Figure 28.2 shows the central illustration of the study [2].

Indications

- See Table 28.1 for indications.

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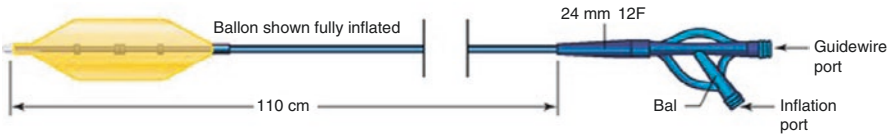


Fig. 28.1 BAV balloon

Table 28.1 BAV in adults >21 years of age with severe aortic stenosis

Indication	Class
Bridge to aortic valve replacement in hemodynamically unstable patients	IIb
Palliation in patients with serious comorbid conditions who are deemed high risk for aortic valve replacement	IIb
Prior to urgent noncardiac surgery	IIb
Before TAVR as a bridge to the procedure	IIb
Alternative to AVR	III

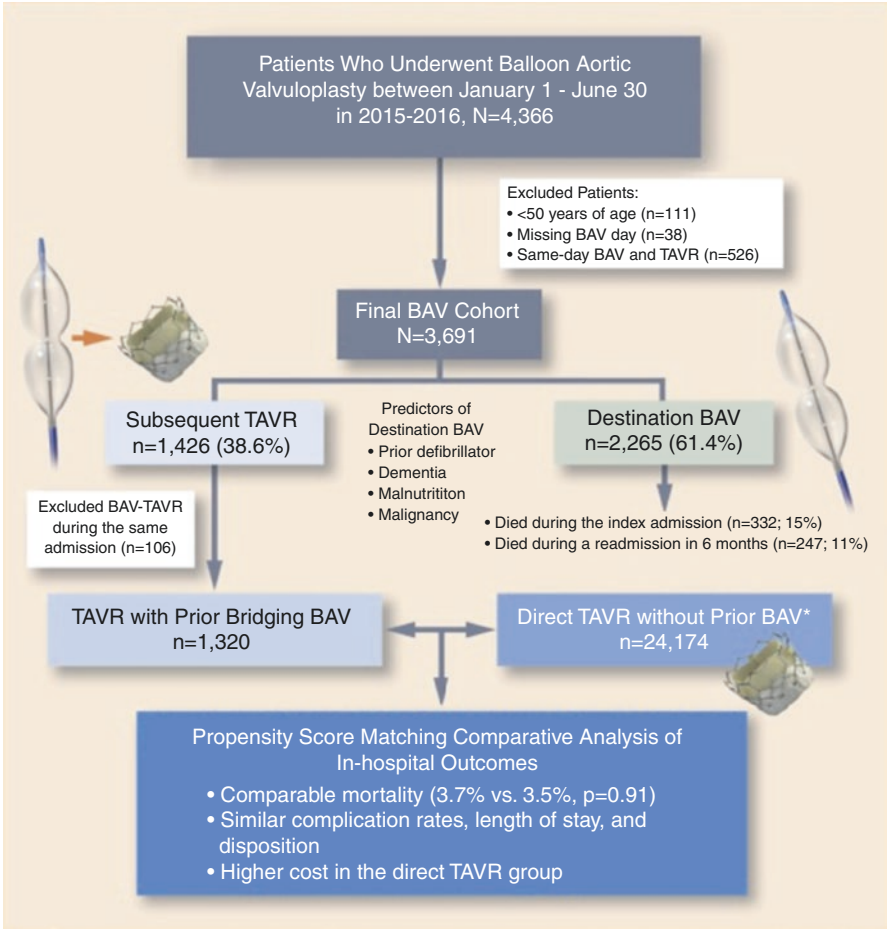


Fig. 28.2 Central illustration of study flow chart: Kawsara, A. et al [2]

Contraindications

- Absolute: Severe aortic regurgitation (AI).
- Relative: Moderate AI.

Equipment

- Sheaths:
  - Definite BAV: Single arterial access
    Micropuncture access kit: 6-, 7.5-, 8-, 11-/12-/13-French (Fr) sheaths (depending on the balloon size).
  - For possible BAV: Single arterial access
    Micropuncture access kit: 6- and 7.5-Fr sheaths.
- Catheters:
  - Swan-Ganz catheter.
  - 5-Fr Amplatz right 2 (AR2).
  - 6-Fr Judkins left 4 (JL4).
- Wires:
  - 0.035″ standard J wire.
  - 0.038″ straight-tip stiff Terumo® wire.
  - 0.035″ Amplatz Super Stiff™ wire (Fig. 28.3).
- Balloon:
  - Z-MED II™ balloon (18–23 mm).
  - True™ balloon (20–22).
- Closure device:
  - Perclose × 2 for definite BAV (need only 1 for possible BAV).
- Other equipment:

Fig. 28.3 Straight-tip Amplatz Super Stiff™ wire

- Contrast 50 cc, diluted 1:3.
- 3 manifolds.
- Heparin 3000 units prior to crossing the valve and consider additional heparin if difficulty in crossing the valve.
- Atropine or neosynephrine if needed to maintain an adequate heart rate and blood pressure.
- Temporary venous pacemaker (TVP).

Vascular Access

- Definite BAV:
  - Single arterial access: 6-Fr sheath in the common femoral artery (use micropuncture to ensure that entry site is in the CFA and the groin can be sutured with a Perclose device).
  - Place two suture-based closure devices (Perclose) in orthogonal positions (referred to as Preclose). After Preclose, upgrade to a 10 Fr sheath.
  - Venous access: Femoral vein with 7.5 Fr sheath for hemodynamic assessment using a Swan-Ganz catheter.
- Possible BAV:
  - Single arterial access: 6-Fr sheath in the common femoral artery (use micropuncture to ensure that entry site is in the CFA and the groin can be sutured with a Perclose device). Place one suture-based closure device (Perclose) in the same direction as the needle puncture (referred to as Preclose). After Preclose, upgrade to an 8 Fr sheath (see Chap. 13).
  - Venous access: Femoral vein (7.5 Fr sheath) for Swan-Ganz hemodynamic assessment.
- Angiographic views
  - Aortogram: LAO 30°.
  - Crossing the valve: LAO 30°.
  - Inflation of balloon: RAO 30°.

Crossing of the Aortic Valve

- *Catheter manipulation*
  - Advance Amplatz right 2 (AR2) or Amplatz left 1 (AL1) catheter to the aortic root. Amplatz left 2 (AL2) can be used for very large aortic root, AR1 and JR4 for a small root, and occasionally, an MP catheter may be required.
  - Once the catheter is positioned in the aorta, compare the pressure in the aorta to the femoral side-arm pressure (both transducers need to be zeroed and flushed again). Measure the difference Ao-FA gradient between the central aortic pressure and common femoral arterial pressure Ao-FA. If the difference is more than 10 mmHg, a double-lumen pigtail should be used.

  - In 30° LAO view, perform a root aortogram and store this image as a reference picture on the second monitor. Pull the catheter back with a slow but firm clockwise rotation to direct the catheter tip to the center of the valve plane.
  - Cross the aortic valve with a 0.038″ straight-tip hydrophilic guidewire (Terumo®).
- *Wire manipulation*
  - In the same fluoroscopic view as the reference image, move the guide wire with firm gentle movements in and out of the catheter tip with the right hand, while the left hand torques the catheter to keep in plane the valve orifice.
  - Once the wire crosses the aortic valve, it is advanced to the middle of the LV, and the fluoroscopic view is changed to RAO to see the wire tip.
  - The catheter is then advanced over the guidewire and positioned in mid-ventricular position in RAO 30° view.
  - Connect the catheter to the manifold system and check the left ventricle-femoral artery gradient (LV-FA). If original femoral pressures were discrepant by more than 10 mmHg with the aortic pressure, then double-lumen pigtail (Langston) catheter is placed with exchange length J wire into the ventricle. If the central aortic pressure was greater than the FA pressure, subtract the Ao-FA value from the gradient measured across the valve to get the true gradient across the valve. If the central aortic pressure was lesser than the FA pressure, add the Ao-FA value from the gradient measured across the valve to get the true gradient across the valve.
  - Use dobutamine for low-gradient, low-flow aortic stenosis (AS) if left ventricular ejection fraction (LVEF) is <50% as per protocol.
  - If valvuloplasty is appropriate, then advance Confida Wire™ wire (Fig. 28.4) in LV, through the AR2/Langston catheter.

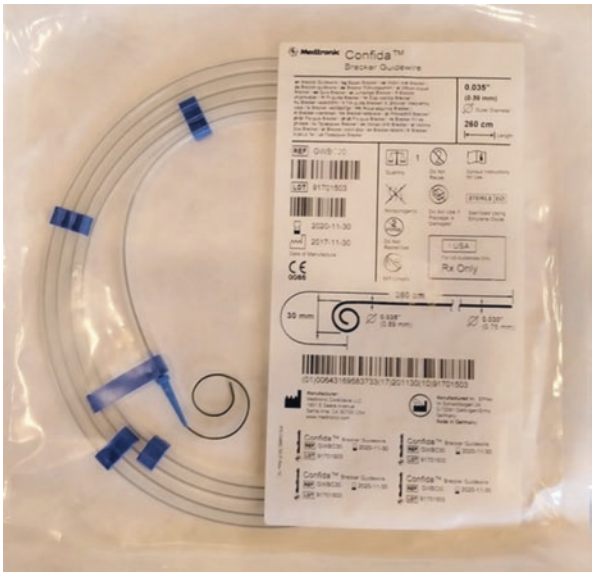


Fig. 28.4 Confida™ wire

Balloon Management

- *Balloon sizing*
  - Around 1:0.9 to annulus size.
  - NuMED Z-MED II™ balloon catheter: 20 mm (11 Fr sheath), 22 mm (12 Fr sheath), 23 mm (13 Fr sheath), and 25 mm (14 Fr sheath).
  - Use the smaller 20 mm balloon if the valve is densely calcified or the aortic annulus is small (<19 mm by echocardiography).
  - In general, we begin with a 22-mm Z-MED II™ balloon. In most of our cases, we do not use a larger balloon, though a 25-mm balloon can be used if the aortic annulus diameter is larger than 24 mm.
  - True™ balloon: 20 mm (11-Fr sheath) and 22 mm (12-Fr sheath).
  - Pediatric Atlas balloon 20 mm (9-Fr sheath) and Tyshak 22-mm balloon (8-Fr sheath).
- *Balloon Preparation*
  - Flush the balloon through the flush port. Attach the 3-way stopcock to the balloon inflation extension of the catheter. Attach a 60-cc syringe filled with a 40 cc of diluted contrast (1:3) to the straight port of the stopcock. De-air the balloon by pulling negative with a 60-cc syringe. Repeat three to four times to ensure the balloon does not have air (Fig. 28.1) and remove air from the syringe as well. Attach another 10 cc balloon filled with contrast/saline (diluted 1:3) to the other port.

Temporary Pacemaker Use

- Position a balloon-tipped temporary pacemaker in the right ventricular apex/posterior wall through the femoral vein after removing the Swan-Ganz catheter and deflate the temporary wire tip balloon to secure the position. The pulse generator has a capability of up to 220 beats/min.
- Capture is verified prior to balloon inflation (test at least 10–20 beats/min faster than the intrinsic rhythm). Use the pulse generator to pace at 180 beats/min (or above) to decrease systolic blood pressure to 50 mmHg. If BP does not decrease, or a 2:1 conduction block is seen, a slower rate may be considered (160/min) (see Fig. 28.5).

Balloon Inflation

- While pacing, simultaneously exert forward pressure on the balloon catheter (loaded on the stiff wire). Inflate the balloon via 60-cc syringe and a 10-cc syringe if needed to ensure complete expansion. Look for loss of aortic pressure waveform with balloon inflation and disappearance of the waist at valve orifice (see Fig. 28.6). In case the BAV is done as a part of a transcatheter valve

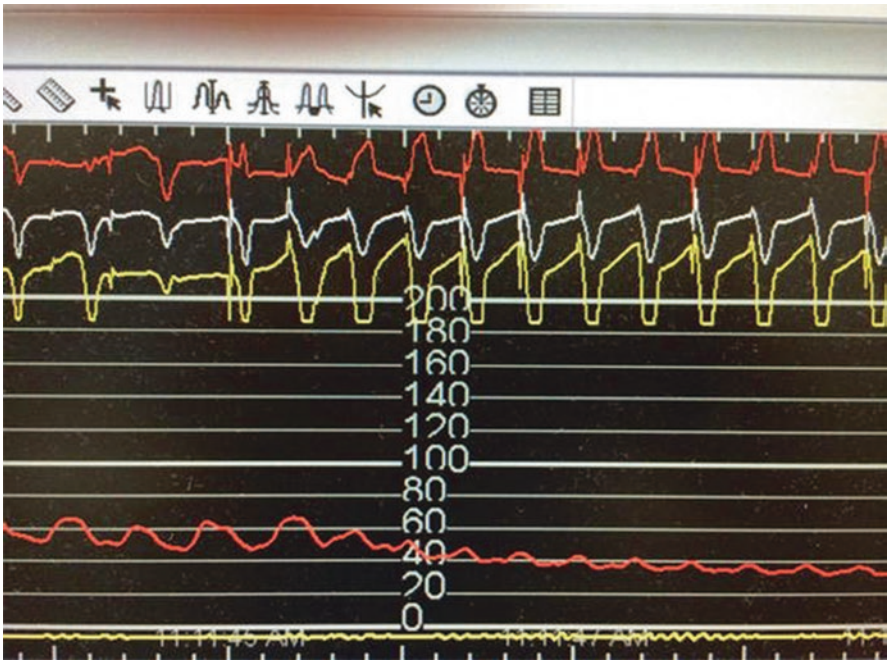
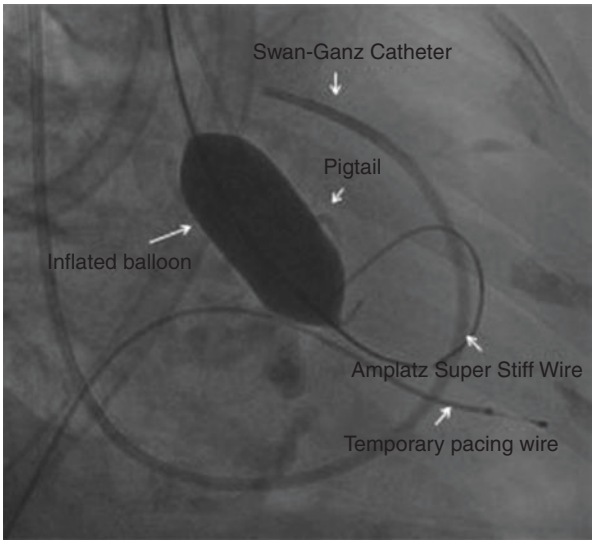


Fig. 28.5 Rapid pacing at 180 beats/min with drop in systolic pressure to 50 mmHg

Fig. 28.6 Valvuloplasty



replacement, a 20-cc aortogram may be performed (20-cc volume with a flow rate of 20 cc/s at 700 psi) to look for adequacy of balloon size.

Balloon Deflation

- Immediately after balloon inflation and complete expansion, deflate the balloon by applying negative pressure on the 60-cc syringe, and while maintaining negative pressure pull the rapidly deflating balloon into the ascending aorta and wait for the restoration of the aortic waveform (for CABG cases pull the balloon in the descending aorta to avoid graft compromise).
- If hypotension or bradycardia persists, then administer phenylephrine and atropine as needed and be vigilant of other causes of hypotension (tamponade, aortic dissection, rupture retroperitoneal bleed).
- Pull the balloon out with negative pressure on the 60-cc syringe usually through the arterial sheath.
- If the balloon is stuck in the sheath, then take the balloon and sheath out while maintaining the wire in LV under fluoroscopy and replace it with a new sheath over the wire.

Post-procedure Assessment

- Recheck the transvalvular pressure gradient with AR2 catheter in LV and side port of femoral arterial sheath (peak to peak).
  - If the gradient is reduced by 2/3 of the initial gradient or the valve area doubles, then the procedure is considered successful.
  - If not, then repeat the procedure with a larger balloon.
- Pull AR2 catheter in the aorta and perform aortogram in LAO 30° projection to evaluate the aortic regurgitation post BAV.
- Remove the temporary pacemaker (TPM), and reintroduce the PA catheter to measure PA, PCWP, and CO.

Complications

- Differential causes of hypotension (including access-related hemorrhage, tamponade, LV rupture, and aortic dissection).
- Acute aortic regurgitation.

Sheath Removal

- Arterial access is closed with 2 preclose stitches (see Chap. 13).

Post-procedure Management and Monitoring

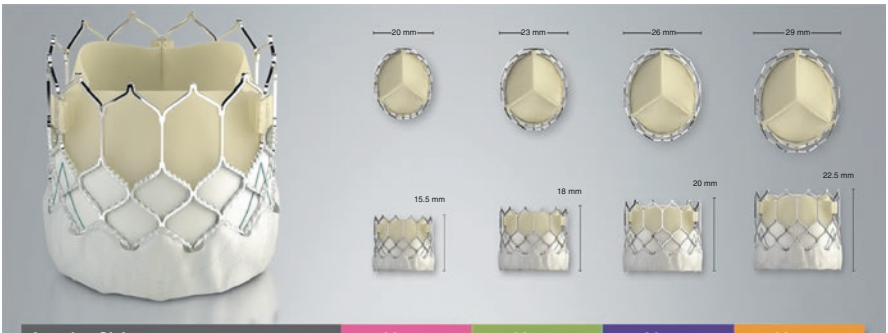
- CCU care.

Transcatheter Aortic Valve Replacement (TAVR)

Transcatheter Aortic Valve Replacement (TAVR) has revolutionized the treatment of patients with symptomatic aortic stenosis (AS) with recent FDA approval of TAVR even for low risk patients. There are currently three different platforms approved by FDA—balloon expandable (Edwards Sapien), self-expandable (Medtronic Evolut PRO CoreValve), and mechanically expandable (Lotus) valves.

Balloon Expandable Valve: SAPIEN Family of Valves

The SAPIEN family of valves are the only FDA approved balloon expandable valve platform available in the USA. Edwards SAPIEN-3 Ultra and SAPIEN-3 belong to the latest generation of valves within the SAPIEN family. These valves are available in multiple sizes (Fig. 28.7) and come color coded in a commander delivery system (Fig. 28.8).



Annulus Sizing		20 mm	23 mm	26 mm	29 mm
Native Valve Annulus Size (CT)	Area	273 - 345 mm^2	338 - 430 mm^2	430 - 546 mm^2	540 - 683 mm^2
	Area Derived Diameter	18.6 - 21 mm	20.7 - 23.4 mm	23.4 - 26.4 mm	26.2 - 29.5 mm
Native Valve Annulus Size TEE		16 - 19 mm	18 - 22 mm	21 - 25 mm	24 - 28 mm

Fig. 28.7 Edward SAPIEN-3 valve (© Edwards Lifesciences LLC, Irvine, CA. All rights reserved. Used with the permission of Edwards Lifesciences LLC)

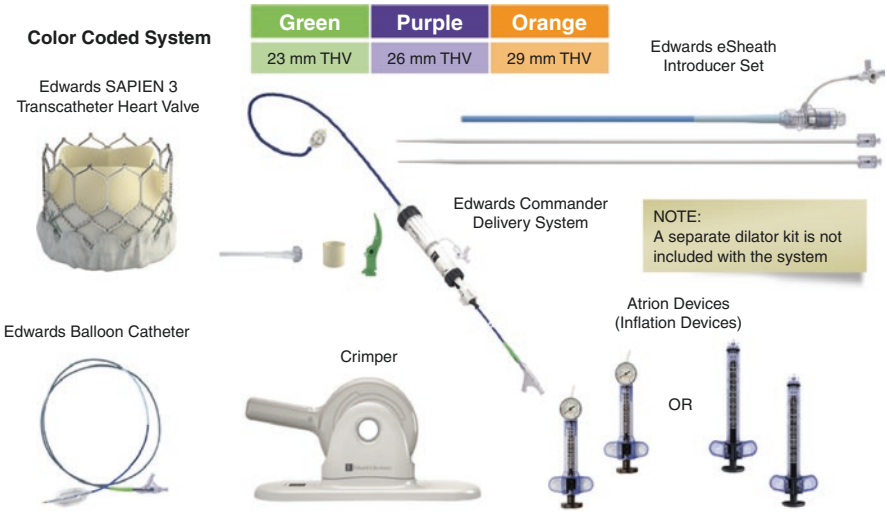


Fig. 28.8 Color Coded Edwards Commander delivery system

Sizes Available

- SAPIEN-3 Ultra valve is available in 20, 23, and 26 mm in diameter.
- SAPIEN-3 valve is available in 20, 23, 26, and 29 mm in diameter.
- Advantage of SAPIEN-3 Ultra compared with SAPIEN-3
  - Proven frame and leaflet design
    Ultra-low delivery profile with high radial strength facilitating future coronary access.
    Bovine pericardial leaflets.
  - Features a taller, textured PET outer skirt
    Approximately 40% increased outer skirt height resulting in less paravalvular leak (PVL).
    In TAVR patients, moderate-severe PVL, but not mild PVL is associated with increased mortality.

Table Setup

- The key components of table setup on the back table are shown (Fig. 28.9).

Sheath Introducer Set Preparation

- Unpack Edwards eSheath Introducer set and visually inspect for damage. Set loader to the side for later use. Gently flush dilator through guidewire lumen with

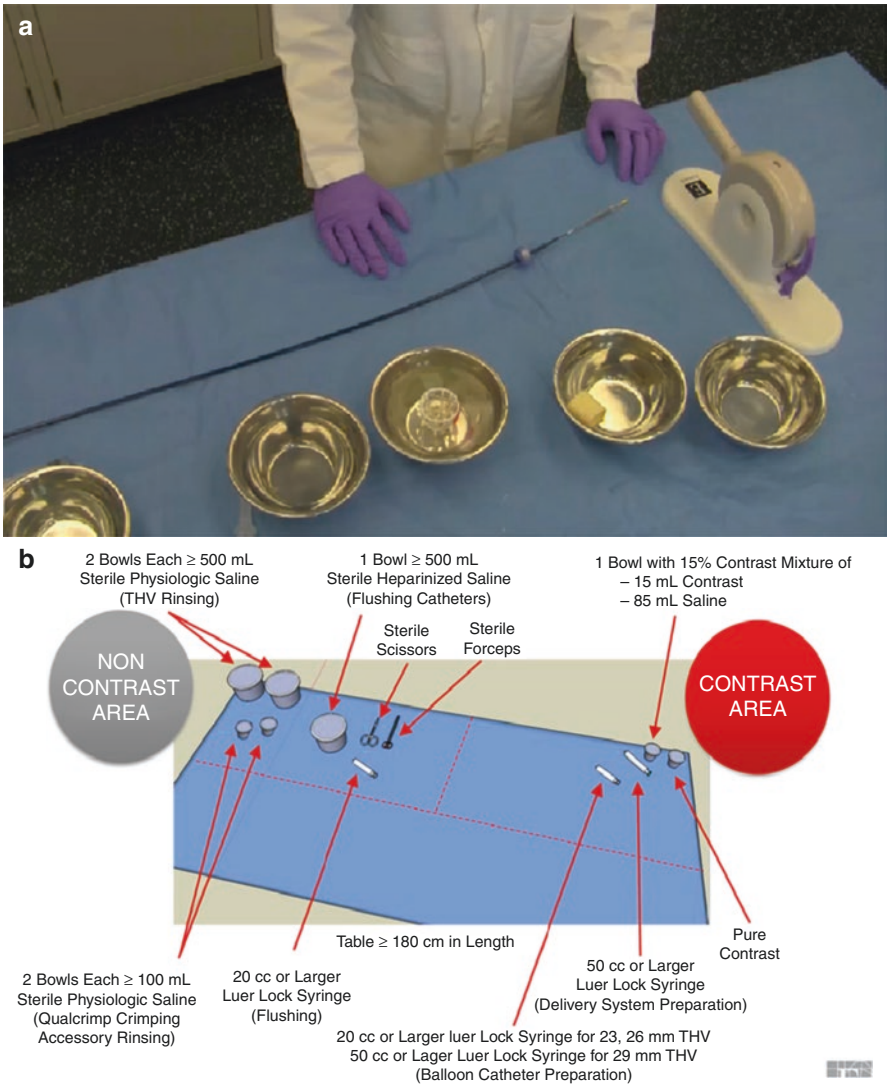


Fig. 28.9 (**a**) The valve preparation table. (**b**) Back table for valve assembly

heparinized saline. Wet entire length of the sheath and dilators with heparinized saline (Fig. 28.10a). Gently flush sheath through flushport with heparinized saline until filled and close stopcock to sheath (Fig. 28.10b).

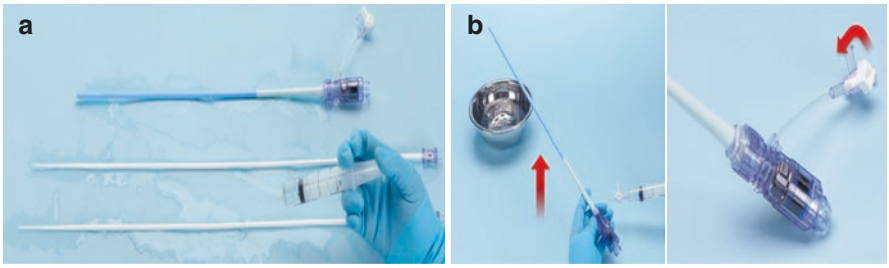


Fig. 28.10 (**a**) Wet the entire length of the sheath and dilators. (**b**) Flush dilators and sideport of sheath

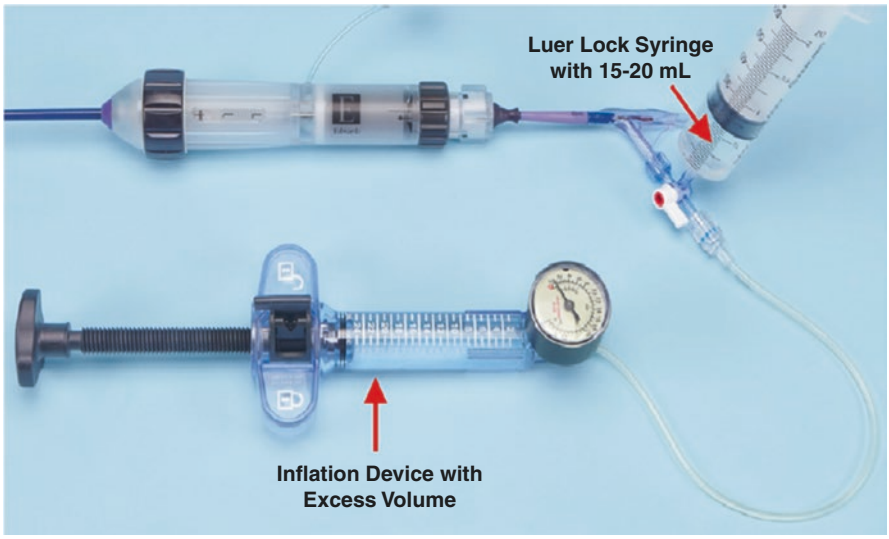


Fig. 28.11 Balloon catheter preparation

Balloon Catheter Preparation

- Attach high pressure 3-way stopcock to balloon inflation port. Prepare a Luer lock syringe with 10 ml diluted contrast and attach to 3-way stopcock. Fill inflation device with excess volume relative to indicated balloon catheter inflation volume, lock, and attach to 3-way stopcock. Close stopcock to balloon catheter and de-air inflation device (Fig. 28.11).
- Close stopcock to inflation device and de-air balloon catheter with Luer lock syringe. Close stopcock to balloon catheter and rotate inflation device knob clockwise to remove any remaining air bubbles and contrast medium from inflation device to Luer lock syringe to achieve specified volume. Ensure inflation device is locked, open 3-way stopcock to inflation device, and remove Luer syringe (Fig. 28.12).

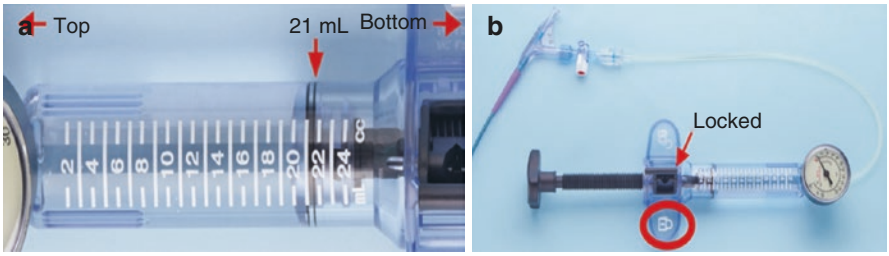


Fig. 28.12 (**a**) Removing air bubbles from the inflation device (**b**) Correct Position of the inflation device and 3-way stopcock attached to balloon catheter

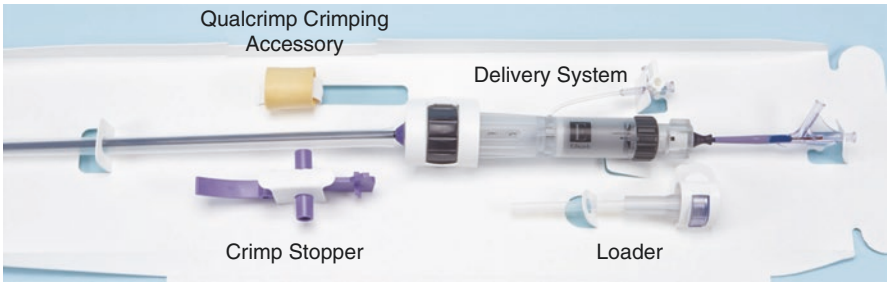


Fig. 28.13 Components of delivery system before unpacking

Delivery System Preparation

- Components of the delivery system are shown in Fig. 28.13. Visually inspect the following components for any damage.
- Crimper has to be prepared by ensuring crimper aperture is in open position and attach crimp stopper to the base of crimper and click into place (Fig. 28.14).
- Gently flush the delivery system through flushport with heparinized saline. Remove the distal balloon cover over tapered tip. Remove stylet and set aside. Gently flush the guidewire lumen with heparinized saline and insert stylet back into guidewire lumen (Fig. 28.15).
- Place loader cap on delivery system. Place delivery system in default position (end of strain relief is aligned between the two white markers on the balloon shaft). Make sure the Flex Tip is covered by the proximal balloon cover. Unscrew loader cap from loader tube and flush loader cap and seal. Gently place loader cap over proximal cover and onto the Flex Catheter with inside of cap facing towards the Tapered Tip. Place delivery system back to Packaged Position (fully advance the Balloon Catheter in the Flex Catheter) (Fig. 28.16a–c).
- Remove proximal balloon cover (Fig. 28.16d).
- Attach high pressure 3-way stopcock to balloon inflation port. Prepare 50 cc or larger Luer lock syringe with 15–20 mL diluted contrast solution and attach to

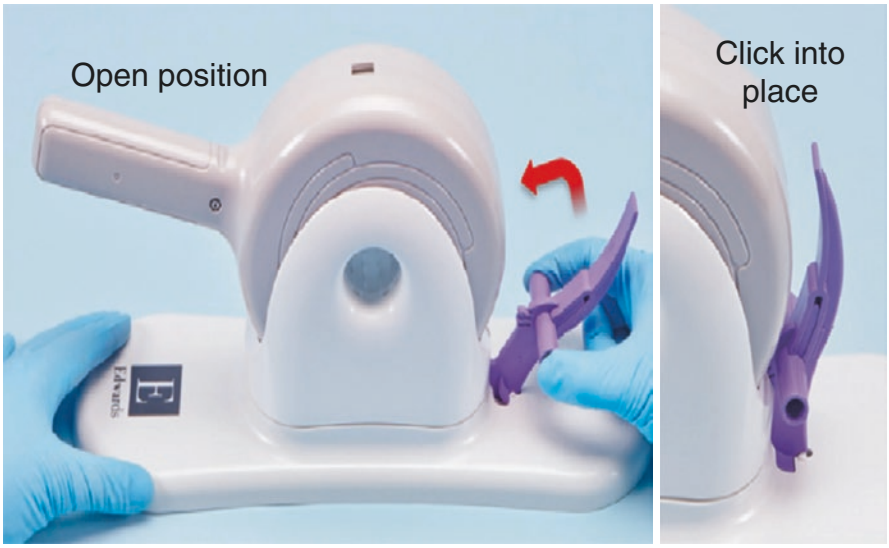


Fig. 28.14 Preparation of crimper

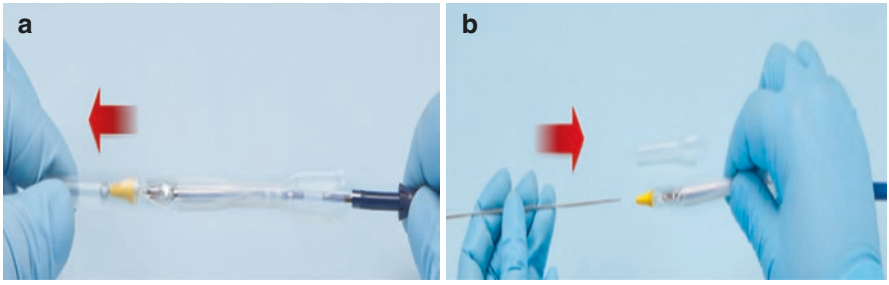


Fig. 28.15 (**a**) Remove the stylet before flushing. (**b**) Insert stylet after flushing

3-way stopcock. Fill inflation device with excess volume relative to the indicated inflation volume, lock, and attach 3-way stopcock (Fig. 28.17).

- Close stopcock to delivery system and de-air inflation device. Close stopcock to inflation device and de-air delivery system with Luer lock syringe ensuring no fluid is left in balloon. Air bubbles might be observed in the delivery system when pulling vacuum with the syringe. Check that you have de-aired the delivery system only when at neutral pressure and tilt the balloon tip down to remove air bubbles from balloon (Fig. 28.18).
- Close stopcock to delivery system and rotate inflation device knob clockwise to remove any remaining air bubbles and contrast medium from inflation device to Luer lock syringe to achieve appropriate volume required to deploy THV. Ensure inflation device is locked, remove Luer syringe, and open stopcock to inflation device (Fig. 28.19).
- Mounting and Crimping THV on Delivery System: Place THV into Qualcrimp Crimping Accessory aligning the edge of the Qualcrimp crimping Accessory with the outflow of the THV (Fig. 28.20).

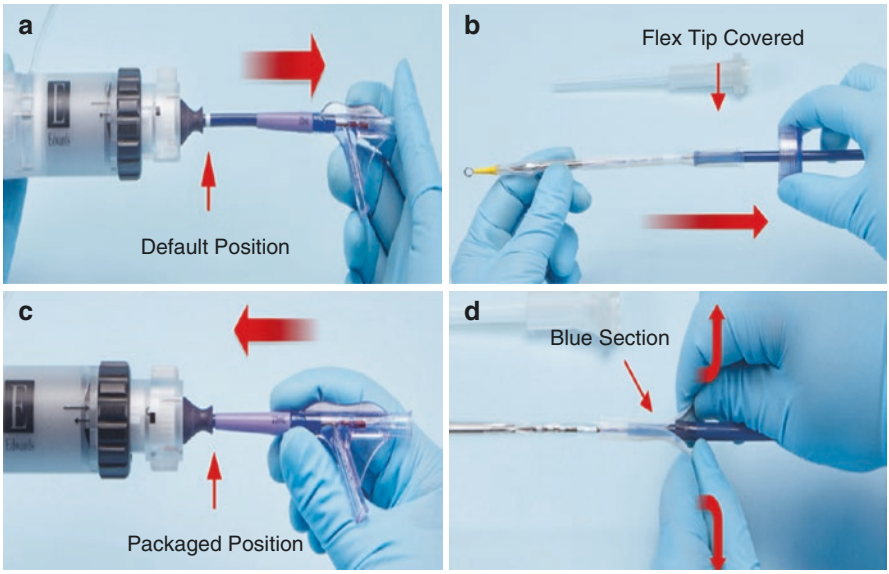


Fig. 28.16 (**a**) Default position of the delivery system. (**b**) Flex tip is covered by the proximal balloon cover. (**c**) Place delivery system in packaged position. (**d**) Proximal balloon cover is removed

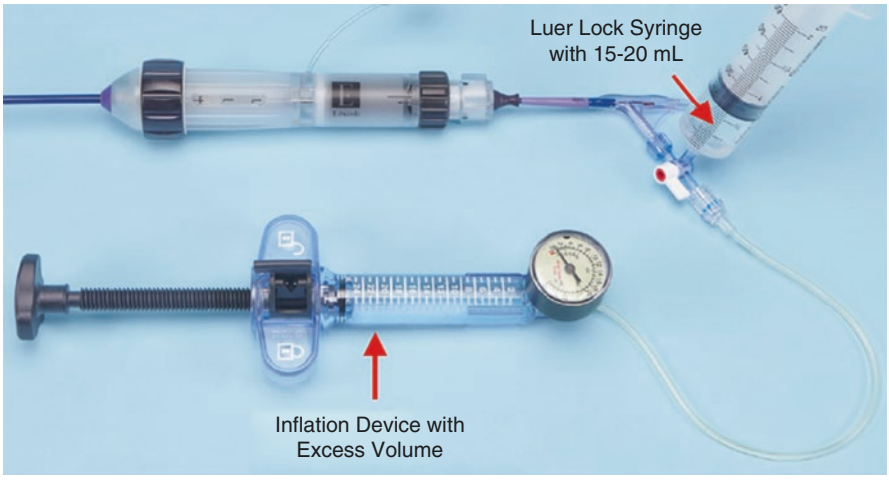


Fig. 28.17 De-airing delivery system

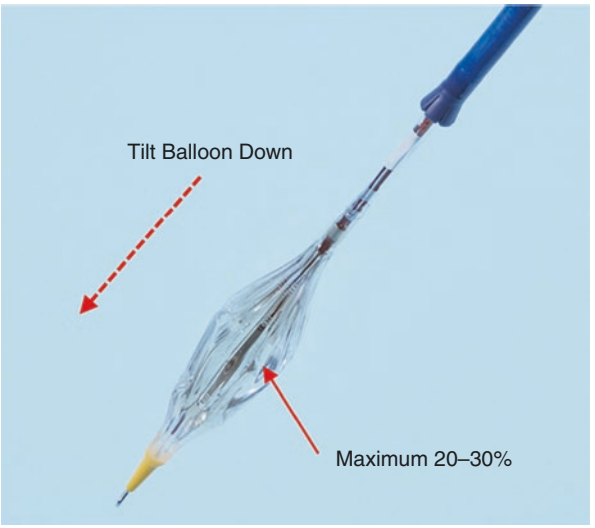
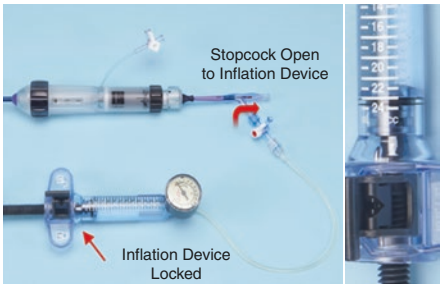


Fig. 28.18 Tilt balloon down during de-inflation

a



b

Nomianl Balloon Diameter	Nominal Inflation Volume	Rated Burst Pressure	Luer Lock Syringe
23 mm	17 mL	7 atm	50 cc or Larger
26 mm	23 mL	7 atm	50 cc or Larger
29 mm	33 mL	7 atm	50 cc or Larger

Fig. 28.19 (**a**) Correct position of the inflation device. (**b**) Nominal inflation volume and rated burst pressure

- Full Crimp is performed until it reaches final stop and hold for 5 seconds. Repeat this Crimp step two more times, for a total of 3 crimps each for 5 seconds (Fig. 28.21).
- Pull balloon shaft to the default position and engage the balloon lock (Fig. 28.22).
- Advance THV into the loader until yellow portion of the tapered tip is exposed, but loader tip is still within the tapered tip section. THV is within the straight section of the loader (Fig. 28.23).
- Screw loader cap to loader tube. Gently flush flex catheter through flushport until loader is completely filled and close stopcock to delivery system (Fig. 28.24).

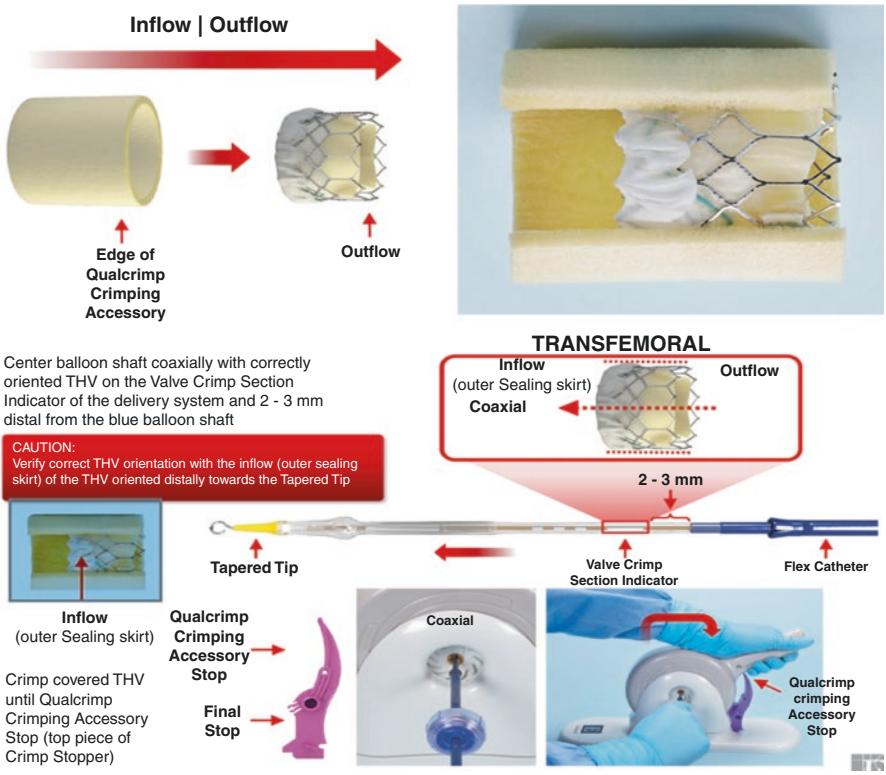


Fig. 28.20 Mounting and crimping THV on delivery system

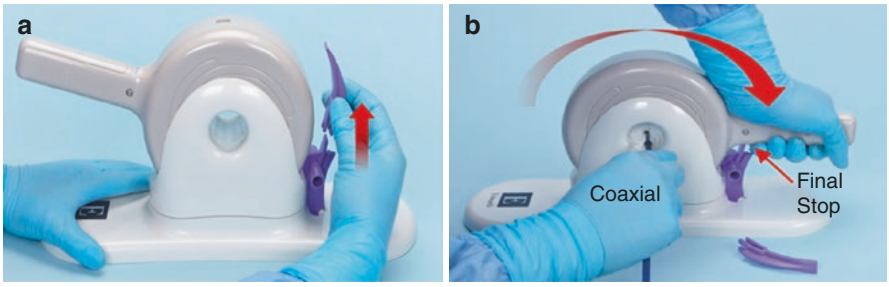


Fig. 28.21 (**a**) Remove crimp stopper. (**b**) Process of crimping

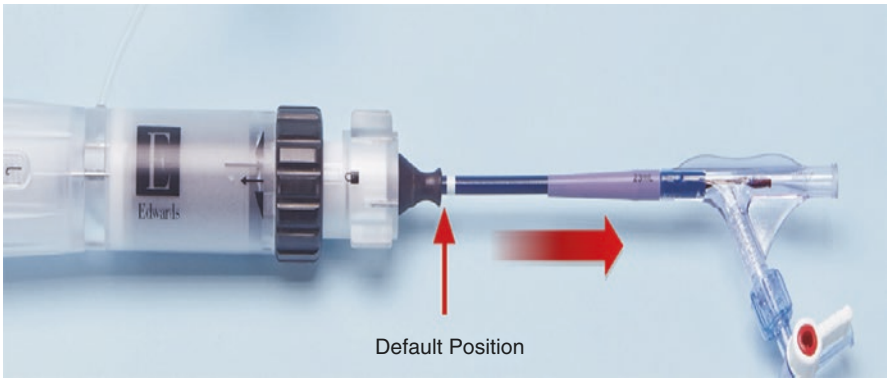
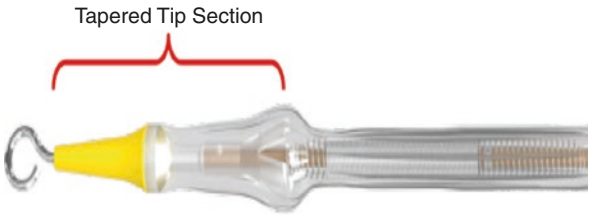


Fig. 28.22 Balloon shaft back to default position

Fig. 28.23 Advance THV into the loader



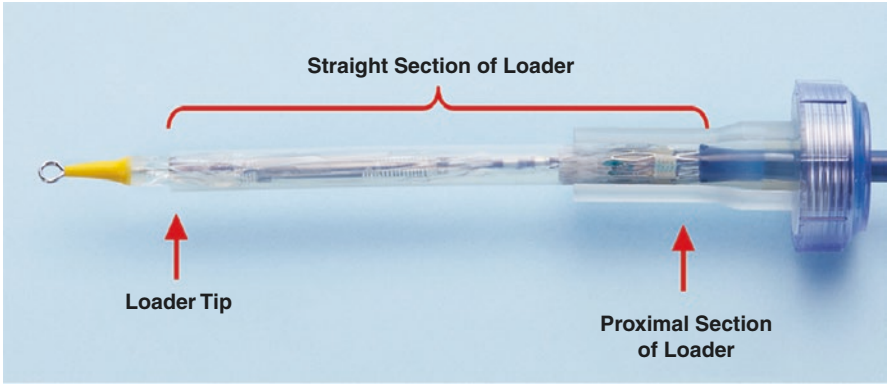


Fig. 28.24 Inserting loader cap to loader tube

Access Preparation for Transfemoral Approach

- The femoral arterial access is serially dilated to accommodate sheaths, or dynamic expansion mechanism is used which allows for transient sheath expansion during transcatheter valve delivery. Preclosure of the arteriotomy site using Perclose Proglide (typically with two sutures) is performed to load the sutures at the margin of the arteriotomy. The sutures are then clamped with a hemostat and covered by drapes to be used for closure at the conclusion of the procedure. A

THV Size	23 mm	26 mm	29 mm
Sheath ID	14F	14F	16F

Fig. 28.25 Sheath ID sizes for different THV size

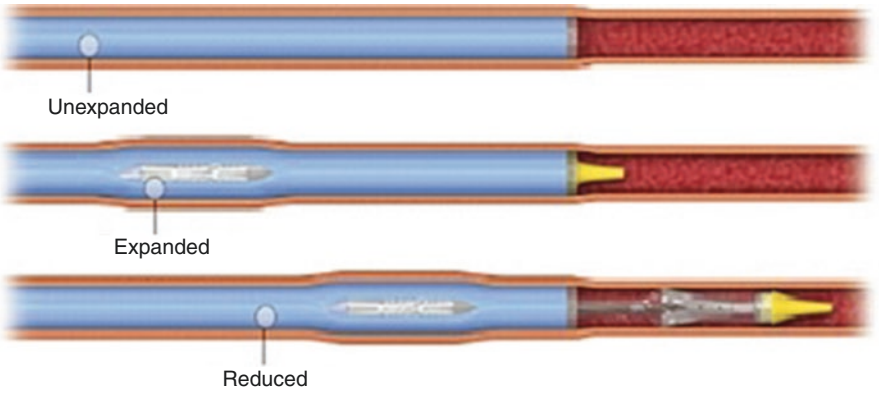


Fig. 28.26 Different scenarios of sheath expansion

14-French sheath (for 20-mm, 23-mm, 26-mm Edwards SAPIEN-3, and Ultra valve) and a 16-French sheath (for 29 mm SAPIEN-3 valve) are used (Fig. 28.25).

- The Edwards eSheath should be inspected prior to use and discarded if damaged. Weight based heparin (100 U/kg) should be administered prior to sheath insertion with a goal activated clotting time (ACT) of >250. The sheath should be hydrated prior to insertion without wiping off the hydrophilic coating. The sheath should be inserted with the Edwards logo facing upward over a stiff wire (Supracore or Extra Stiff Amplatz wire) that is held with tension to provide a firm rail for placement. Insert the sheath slowly with a continuous motion allows gradual dilation of the vessel. Ensure visualization of the sheath tip under fluoroscopy and advance past the aortic bifurcation. Once inserted, suture the sheath in place, and intermittently flush per standard practice (Fig. 28.26).

Implantation Angle

- In the contralateral femoral artery, a diagnostic pigtail catheter is advanced into the right coronary cusp for root aortography. The implantation angle is the fluoroscopic angle where the bases of all three cusps are aligned in one line. Annular plane is an imaginary plane that touches the lowest points of all 3 cusps (Fig. 28.27).
- Figure 28.28 shows an aortogram: A 5 Fr angled pigtail is placed in the right coronary cusp, and aortogram is performed identifying a projection with all three cusps visible (a careful review of CT scan reconstructions can help identify the best fluoroscopic view to get the appropriate annular planes).

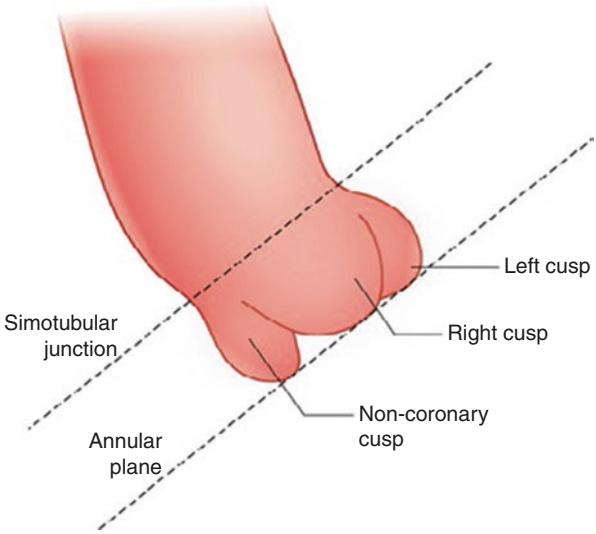


Fig. 28.27 Annular plane is an imaginary line joining the lower part of 3 cusps

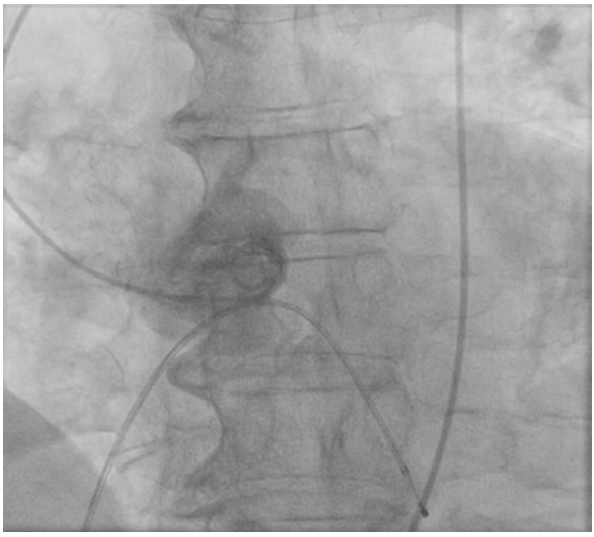


Fig. 28.28 Angiographic view of annular plane

Crossing the Valve and Wire Positioning

- Refer to the BAV section for crossing the valve. Consider different wires such as extra small Safari wire depending on the LV size and risk factors for LV perforation depending on the patient's clinical scenario such as chemotherapy or on chronic immunosuppression or radiation therapy (Fig. 28.29).

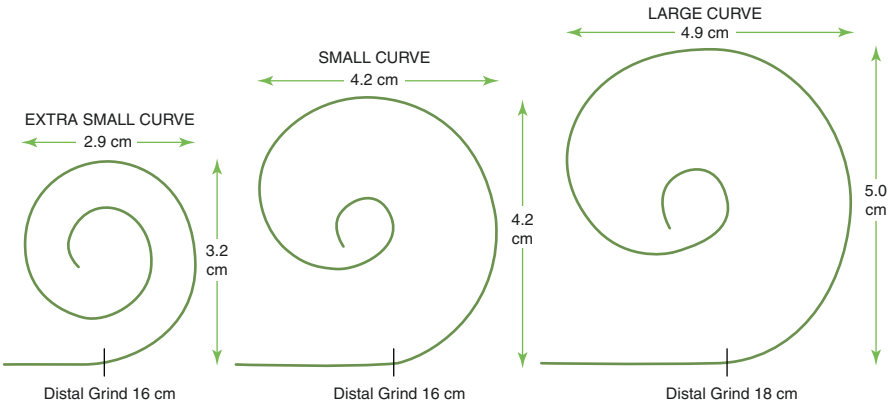


Fig. 28.29 Different size and shapes of Safari wire

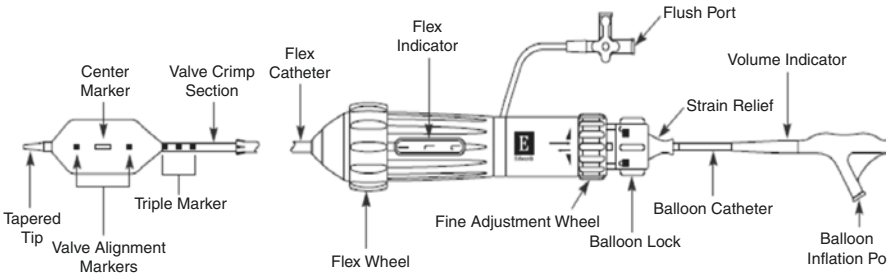


Fig. 28.30 Commander delivery system for Edward SAPIEN valve (© Edwards Lifesciences LLC, Irvine, CA. All rights reserved. Used with the permission of Edwards Lifesciences LLC)

Advancement of the Delivery System

- Different components of the Commander delivery system is shown (Fig. 28.30).
- The delivery system should be inserted into the eSheath with the Edwards logo facing upwards, the flush port pointing away, and holding the loader cap along with the THV. Once the delivery system is inserted into the sheath, the wire positioning should be confirmed in the left ventricle and the sheath tip distal to the aortic bifurcation.

Align the Valve

- The balloon-mounted valve is then advanced to the descending aorta. To align the valve, the flex catheter should be retracted at the Y-connector until part of the warning marker is visible (but not past the warning marker) and the balloon lock secured. In general, this technique involves fixation of the delivery system with your left hand and pulling back the balloon catheter with your right hand. The fine adjustment knob should be slowly rotated to center the THV between the valve alignment markers with no gaps or overlap (Fig. 28.31).

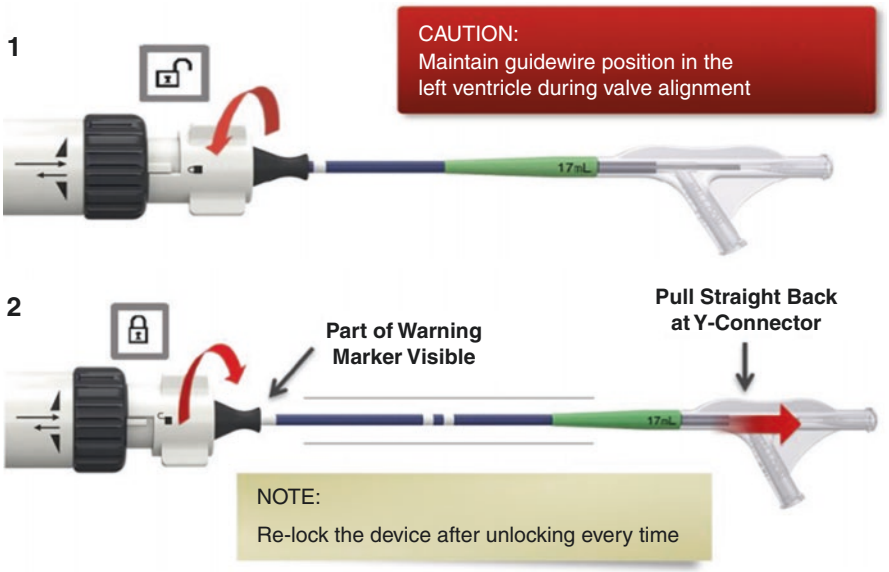


Fig. 28.31 Aligning the valve

Crossing the Aortic Valve with Edwards SAPIEN Delivery System

- The delivery system is then advanced to the aortic valve position using angiographic and echocardiographic guidance. For ease of prosthesis arch transit, the delivery platform is equipped with the Commander system which should be mostly fully flexed upon entering the distal aortic arch.
- Before crossing the aortic arch, the delivery catheter is flexed, a left anterior oblique projection is obtained, and the catheter is advanced with traction maintained on the wire. When the nose cone is past the aortic valve, return to the prior fluoroscopic view for implantation of the valve. It is important that the wire position in the ventricle is maintained at all times even when difficulty is encountered advancing the catheter system through the stenotic native valve (as this valve cannot be retracted in the sheath and it is very unlikely that a new wire will cross the valve through the valve delivery system).
- Once the bioprosthesis crosses the native valve, the outer "flex" catheter is withdrawn so as not to interfere with balloon inflation (there is a fluoroscopically visible marker).

Edwards SAPIEN Delivery System

- For the transfemoral approach, the recommended positioning of the prosthesis is 80–20%, which means 80% of the prosthesis should be on the ventricular side of the aortic annulus with 20% of the prosthesis on the aortic side of the annulus (Fig. 28.32).

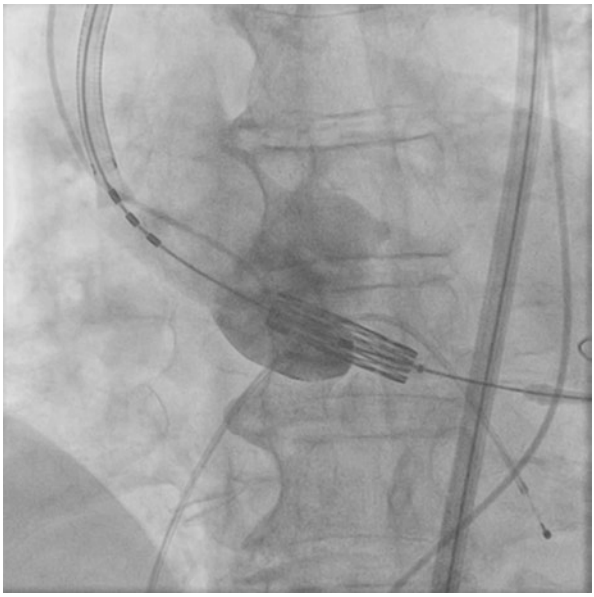


Fig. 28.32 Positioning of valve

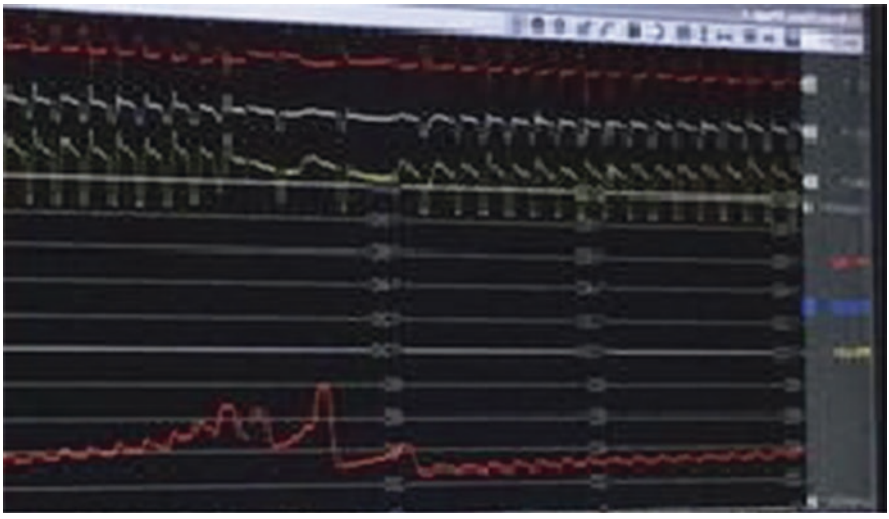


Fig. 28.33 After confirmation of the valve position on echocardiogram and angiogram, pacing is started on command of the operator, and blood pressure is depressed to systolic 50 mm of Hg

- The valve plane is confirmed with the pigtail placed on the noncoronary native cusp and injections through this catheter assist in valve placement. The correct orientation of the prosthesis should be confirmed both visually prior to placement into the introducer sheath and angiographically prior to deployment (during a test v-pace run) (Fig. 28.33).

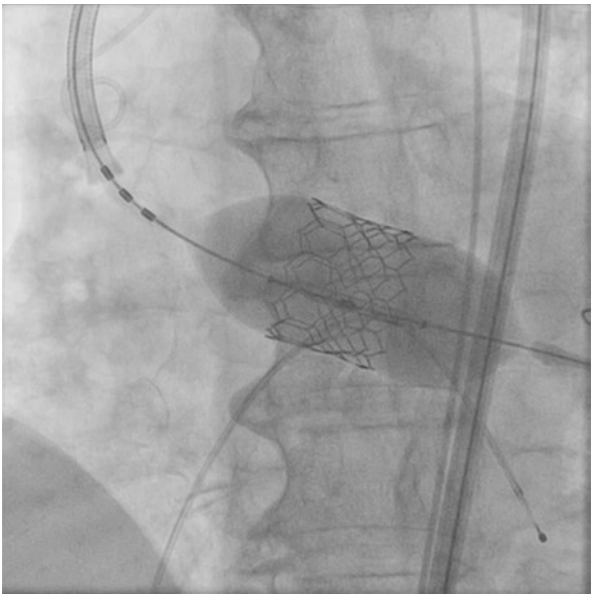


Fig. 28.34 Edwards SAPIEN-3 Ultra valve being deployed

- The primary operator defines the roles of the assistants (the "inflator," the "contrast injector," and the "pacer"). Next, transvalvular flow is severely depressed by rapid ventricular pacing (Fig. 28.33).
- The primary operator gives simple, clear instructions for pacing, and when blood pressure drops to around 50 mm of Hg, the valve is then balloon expanded in position with rapid but steady inflation mode. The primary operator calls out 1–1000, 2–1000, 3–1000, 4–1000, 5–1000, 6–1000 to ensure proper expansion of the valve. The rapid pacing can be stopped by the implanting physician by giving clear instructions to "stop pacing." The balloon is deflated and pulled in the ascending aorta. Be well prepared to manage complications expediently. If there is moderate to severe aortic insufficiency from a paravalvular leak after the deployment, post-dilation may need to be performed by adding 1 cc of the dye to increase the capacity of the balloon to a larger size (Fig. 28.34).

TAVR in Low Risk Patients

- Outcomes after transcatheter aortic valve replacement (TAVR) were superior or at least as good as those following surgical aortic valve replacement (SAVR) among patients with severe aortic stenosis at low surgical risk, according to results of the PARTNER 3 [3] and EVOLUT [4] trials. In the PARTNER 3 trial, 1000 patients were randomized to either TAVR with a third-generation balloon expandable valve or standard SAVR with a bioprosthetic valve. Primary endpoint was a composite of death from any cause, stroke, or rehospitalization at one year after the procedure. At one year, the primary endpoint occurred in 8.5 percent of

the TAVR group compared with 15.1 percent of the surgery group, meeting the requirements for both noninferiority ($p < 0.001$) and superiority of TAVR vs. surgery ($p < 0.001$). The Kaplan-Meier analysis of the primary endpoint components with TAVR vs. surgery found mortality rates of 1.0 percent vs. 2.5 percent, stroke rates of 1.2 percent vs. 3.1 percent, and rehospitalization rates of 7.3 percent vs. 11.0 percent, respectively. The length of hospital stay was reduced from seven to three days with TAVR.

- In the EVOLUT low risk trial, 1468 patients were randomized to TAVR with a self-expanding bioprosthesis compared with surgical replacement. The primary endpoint was the composite of death from any cause or disabling stroke at 24 months. The as-treated cohort included 1403 patients. At 24 months, death or disabling stroke occurred in 5.3 percent of the TAVR group compared with 6.7 percent of the surgery group, meeting the prespecified criteria for noninferiority. The mortality rate from any cause was 4.5 percent in both groups. The rate of disabling stroke was 1.1 percent with TAVR vs. 3.5 percent with SAVR. At 30 days, TAVR was statistically superior to surgery for the secondary combined endpoint of all-cause mortality or disabling stroke (0.8 vs. 2.6 percent). Patients receiving TAVR had significantly better quality of life and hemodynamics at 30 days.

Cerebral Embolic Protection Device (EPD)

- Despite advances in device technology and procedural techniques, periprocedural stroke remains a severe complication of TAVR, affecting 2% of patients. The Sentinel device, developed by Claret Medical and acquired by Boston Scientific in 2018, is the only cerebral EPD in the United States cleared by the US Food and Drug Administration (FDA). It is a dual-filter system placed via intra-arterial access during TAVR that traps embolic debris within the right brachiocephalic and left common carotid arteries, protecting their vascular territories. The cerebral circulation of the left vertebral artery is not protected (Fig. 28.35).

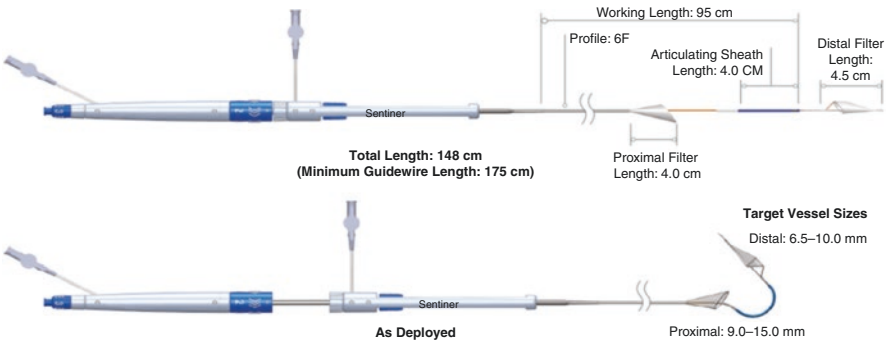


Fig. 28.35 Components of sentinel Cerebral Embolic Protection Device

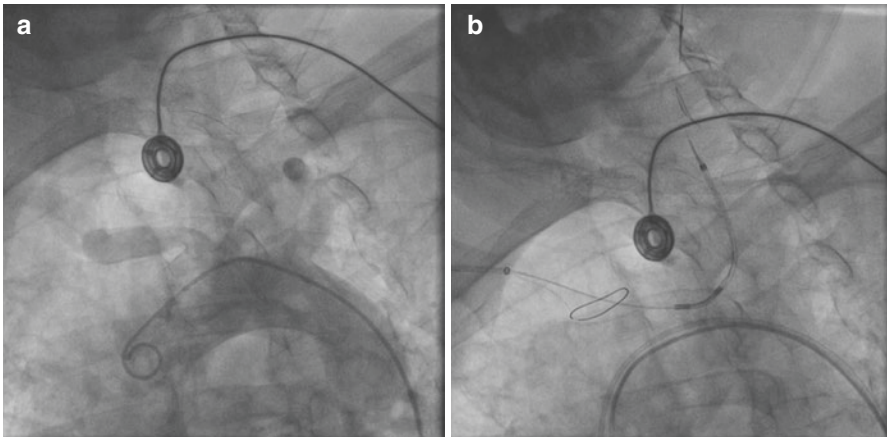


Fig. 28.36 (**a**) Arch angiogram. (**b**) Sentinel placed in situ

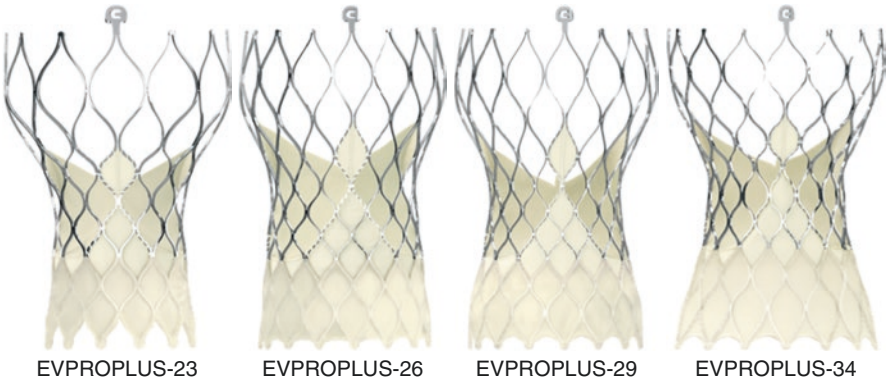


Fig. 28.37 Different size of Evolut PRO PLUS CoreValve

- Perform arch angiogram by placing the pigtail catheter in the aortic arch and at a projection of LAO 40 to assess the feasibility of aortic arch anatomy for Sentinel Cerebral Embolic Protection Device. Anatomy of the arch angiogram can also be assessed from CTA (Fig. 28.36).

Self-Expanding Valve: Evolut PRO PLUS CoreValve

Self-expanding include the Evolut PRO PLUS CoreValves.

Sizes Available

- Evolut PRO PLUS CoreValves come in 4 different sizes including 23, 26, 29, and 34 mm as shown in Fig. 28.37.

- Perimeter of the aortic annulus plays an important role while selecting the size of the valves along with other anatomical parameters such as Sinus of Valsalva diameter, Sinus of Valsalva height, and Left ventricular outflow tract perimeter as listed in Fig. 28.38.

Loading the Valve

- EVOLUT PRO PLUS Enveo loading system is color coded (Fig. 28.39).
  - Loading system for the 23 mm, 26 mm, and 29 mm valves is gray.
  - Loading system for the 34 mm valve is blue.

Evolut™ TAVR system
Valve size selection criteria per msct

	Valve size	Aortic annulus measurements		Sinus of valsalva diameter	Sinus of valsalva height
		Diameter	Perimeter		
Evolut™ PRO and Evolut™ R valves	23 mm	17†/18–20 mm	53.4†/56.5–62.8 mm	≥ 25 mm	≥ 15 mm
	26 mm	20–23 mm	62.8–72.3 mm	≥ 27 mm	≥ 15 mm
	29 mm	23–26 mm	72.3–81.7 mm	≥ 29 mm	≥ 15 mm
Evolut™ R valves	34 mm	26–30 mm	81.7–94.2 mm	≥ 31 mm	≥ 16 mm

Fig. 28.38 Valve size selection criteria

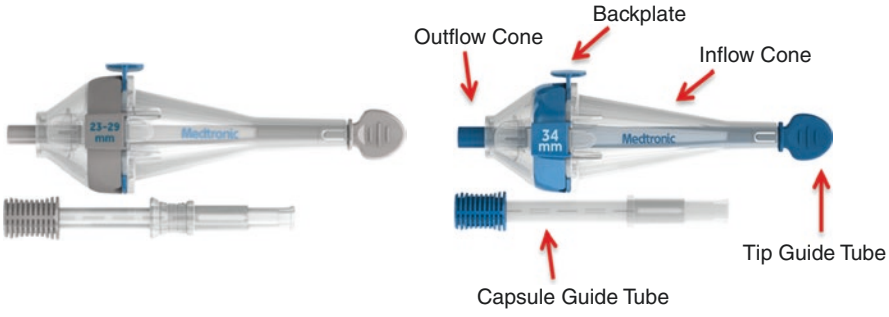


Fig. 28.39 Components of loading system for different valve sizes

Loading Steps: Configure Loading Tray

- Remove the blue locking tabs and integrated baths and rotate the distal half of the delivery system tray counterclockwise 180° (Fig. 28.40).
- Clip the tray tab on the distal tray to the tray tab holder on the proximal tray.
- Fill the integrated loading bath with cold, sterile saline (0° C to 8° C [32° F to 46° F]).

Rinse the Valve

- Immerse bioprosthesis in the first rinsing bowl filled with sterile saline at ambient temperature (15°C to 25°C [59°F to 77°F]) and gently agitate it by hand for 15 seconds as shown in Fig. 28.41.
- Repeat the 15 second rinse in the second rinsing bowl and leave the bioprosthesis submerged in the third bowl until it is ready to be loaded.

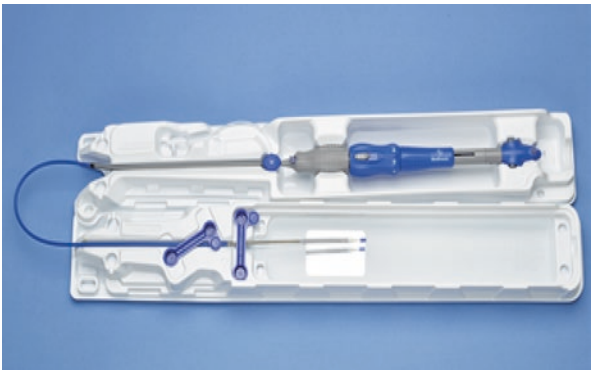


Fig. 28.40 Loading tray

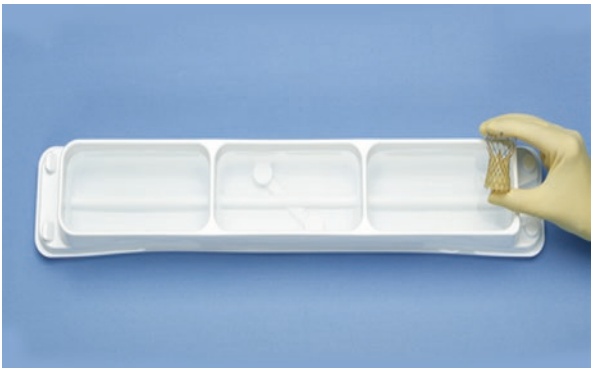


Fig. 28.41 Rinsing the valve

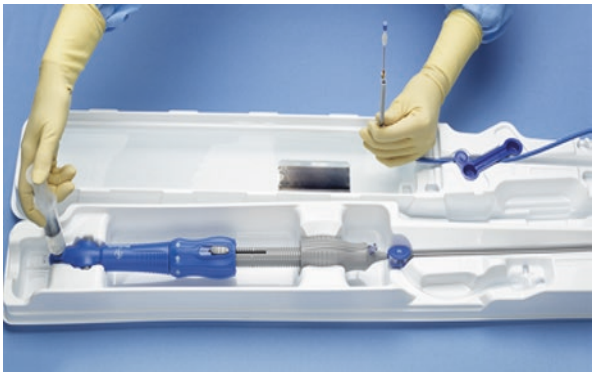


Fig. 28.42 Preparing flash capsule

Flush Capsule

- Raise the capsule vertically and completely open the capsule using the deployment knob.
- Slowly flush the capsule and continue to flush while lowering the capsule until it is completely submerged in bath (Fig. 28.42). *Leave the syringe in place until loading is completed.*
- Attach a locking tab to hold the capsule submerged in the cold saline bath for remainder of loading.

Misloads

- A misload is an improperly loaded valve that can result in damage to the valve, delivery system, or both, and can negatively affect deployment and performance of the valve if not recognized (Fig. 28.43).
- If a misload is identified before removing the backplate and crimping the inflow, deploy and reload the valve in the current delivery system.
- If a misload is identified after removing the backplate and crimping the inflow, discard and replace the valve, delivery system, and loading system.
- Assess for misloading Angiographically
  - Outflow crowns should be parallel to the distal end of the paddle attachment as shown in the figure below (Fig. 28.44)
- Shadow or outline present in the outflow indicating a bent outflow strut as seen in the figure below. It is important to note that crown overlap in the inflow region may be observed during the fluoro load inspection and this is not a misload (Fig. 28.45).

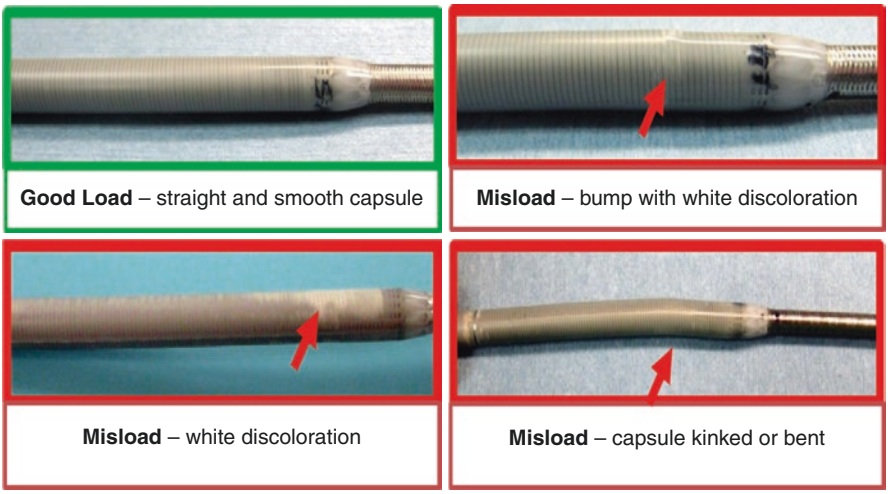


Fig. 28.43 Different types of misloads and indicators of misloads

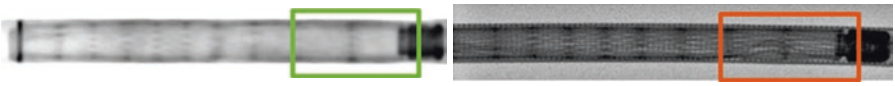
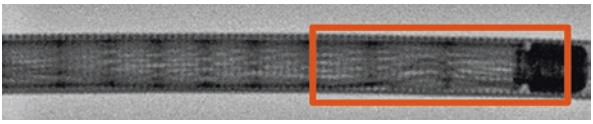


Fig. 28.44 (Green) Correct angiographic load (Red) Outflow crown not parallel to the distal end of paddle

Fig. 28.45 Shadow in the outflow



Procedural Steps

- Insertion of Delivery System with THV
  - Position the pigtail in NCC.
  - Take it in flush @ 3'O clock position.
  - Look @ Hat market-should be on the outer curve of descending aorta in LAO view (i.e., "Hat" on right side of the screen). If not, rotate the delivery catheter counterclockwise to spin the "Hat" from inner to outer curve.
  - Take it across the arch under fluoro in a continuous motion to avoid getting hung up at the arch where it can be calcified.
  - Make sure there is no gap between the nose cone and capsule while navigating through the aortic arch, especially in tortuous and calcified vessels.
  - Ensure "Hat" marker is on the greater curvature (left side of the fluoro screen) if in the LAO/Classic deployment view, or center front facing you in the 2-cusp view.

  - Cross the AV with Evolut device in two cusp view.
  - Remove parallax of the valve—going more CAU should work; usually not necessary in 2-cusp view.
  - Give puff and see whether we are good (just below the NCC); 1–2 mm below NCC is the key.

Deployment Steps

- Start pacing at 120 bpm to reduce systolic motion or PVC.
- Initial 2–3 rotations fast-later slow till we get annular contact.
- Check position by small injection; First operator adjusts position after annular contact; move fast (as pressure is dropped and if we go slower here, valve may pop out).
- Rumble-continue three rotations past till you see pressure wave return.
- Reduce pacing to 70–80 bpm.
- Remove parallax of the valve (LAO/caudal).
- Check with angiography again. If appropriate positioning is confirmed (3–5 mm of the valve below the annular plane), continue to final release. If not, start rapid pacing up to 150 beats per minute. Reposition the valve by recapturing, fully or partially.
- Recapture can be done not more than 3 times with a single THV device.

Final Release

- Pull pigtail out of the aortic root (not a must since it can also be removed via J wire carefully if trapped).
- Pull back stiff wire to remove tension in the system.
- Pace again at 150 bpm to avoid pop-out.
- Release slowly and first person gives slight forward push on the valve to avoid pop-out if implanting high or not much Ca on leaflets.
- Make sure paddle is out of c tab and other tab; can rotate delivery catheter to help free up or push the delivery catheter forward to release tab from paddle.

Removal of the Delivery System

- Pull stiff wire with soft portion inside nose cone—make sure nose cone is away from Evolut frame.
- Remove nose cone carefully to inside the valve, then advance the stiff wire into LV as the delivery catheter is removed; careful not to have nose cone interact with the crowns or tab at the inner curve of the ascending aorta.
- After the delivery system is in the descending aorta, push grey to blue to remarry nose cone to the capsule.
- Then the delivery system is taken out of the body.
- The procedure is finished with aortogram to assess for aortic regurgitation.

Troubleshooting Techniques

- If valve is too deep after full deployment, it may be snared to a higher position.
- If leak persists with appropriate depth of the valve, consider post-dilation. If leak still persistent, a second valve can be placed in the appropriate position (consider left main height while placing the second valve).
- If the valve is deployed above the annulus after it is fully released, then it is critical to maintain wire position in the LV and implant a second valve in an appropriate (lower position) at the annulus level.
- If there is severe paravalvular leak without a deep (low) position of the valve, post-dilation with a balloon size 1:1 to the maximum annular diameter should be performed.

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29 Guide Catheter Selection in Patients with Transcatheter Aortic Valve Replacement

Sunny Goel, Annapoorna Kini, and Samin K. Sharma

Introduction

There is a high prevalence of coronary artery disease in patients undergoing transcatheter aortic valve replacement (TAVR). With indications expanding toward treating younger patients with lower-risk profiles, more and more patients post TAVR will inevitably require coronary angiography and percutaneous coronary intervention. This chapter provides a practical guide on accessing coronary arteries in patients with TAVR valves.

Coronary Artery Disease and TAVR

The prevalence of coronary artery disease (CAD) in patients with severe aortic stenosis (AS) undergoing TAVR ranges from 40 to 75% [1]. Despite the high incidence of CAD in these patients, there is no clear consensus on the timing of revascularization. Current guidelines favor performing percutaneous coronary intervention (PCI) prior to TAVR in presence of coronary artery stenosis >70% in proximal coronary segments but it lacks scientific evidence. Given the progressive nature of CAD, a significant portion of these patients will eventually require coronary angiography and/or PCI post TAVR.

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Challenges in Accessing Coronaries After TAVR

The TAVR valve frame and the displaced native aortic valve leaflets can partially obstruct the coronary ostia after implantation, rendering angiogram and/or PCI after TAVR technically challenging. Unlike for surgical aortic valve replacement, where the prosthesis is sutured under direct vision to match the prosthesis to the native aortic valve commissures, during TAVR the valve orientation is random, and, therefore, a commissural post can end up directly in front of the coronary ostium. Simply engaging the coronary ostia through the frame of the TAVR device can be difficult, if not impossible especially if the commissural tab is overlying the coronary ostium [1].

Practical Considerations

If timing, renal function, and clinical presentation permit, contrast-enhanced cardiac computed tomography can be very useful to identify the relationship of the TAVR valve frame, the displaced native aortic valve leaflets, and the coronary ostia to plan the PCI [2]. If PCI is deferred before TAVR but is believed to be likely required in the future, a shorter balloon-expandable TAVR device may be preferable to a self-expanding device to facilitate subsequent access to the coronaries.

Geometric Interaction Between Self-Expanding and Balloon-Expandable Valves and Coronary Ostia

Self-Expandable Valve

- The CoreValve self-expanding valve is composed of two parts: the self-expanding nitinol support frame with diamond cell configuration and the trileaflet porcine pericardial tissue valve. The frame has three levels: The inflow exerts a high radial expansive force that secures the frame across the annulus; the concave central portion allows the frame to avoid contact with the coronary ostia and the outflow is the largest part of the frame and rests in the ascending aorta. There are several important considerations regarding coronary reaccess. First, it is important to consider the depth of implantation, particularly in patients with low coronary ostia. Due to their design, these self-expanding valves extend beyond the coronary ostia, but the narrow waist inappropriately large sinuses ensures that the risk of acute coronary obstruction is low. To optimize future coronary reaccess, implantation depth is critical, especially if the ostia is <10 mm, as shown in figure below (Fig. 29.1). Because the skirt height of the Evolut-PRO is 13 mm, we need to implant at least 4 mm below the annular plane to ensure the skirt is not overlaying the coronary artery. Because the Evolut-R and Evolut-PRO are recapturable when partially deployed, it is possible to position the valve with such precision. In this optimal position, it is feasible to engage the coronary artery in a coaxial manner, assuming the native aortic valve leaflets will not

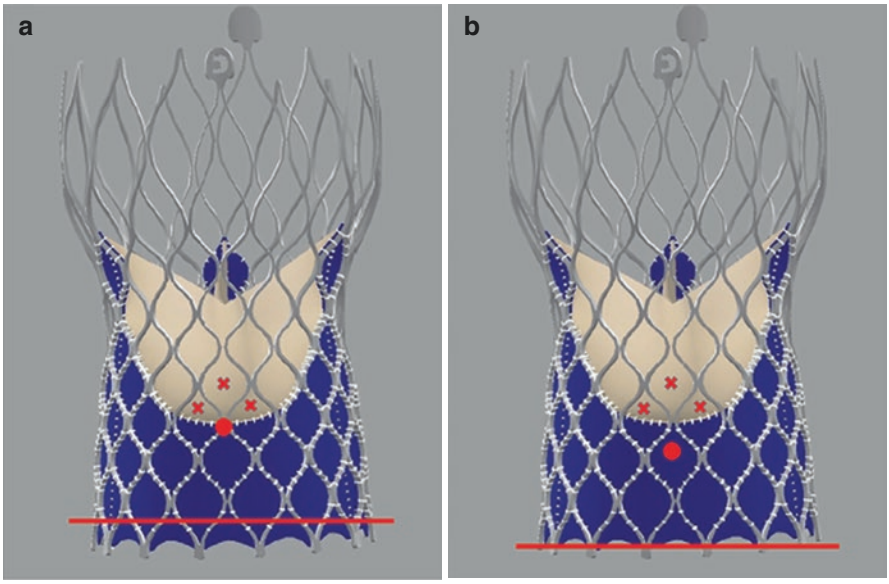

Fig. 29.1 Self-expanding valve and coronary access depending on level of implantation across the annulus. Used with the permission from Yudi et al. [1]

interfere with the path to the coronary ostium (Fig. 29.1a). If the valve is deployed high, as seen in Fig. 29.1b, coronary obstruction would not occur due to the narrow waist of the valve and sufficient sinus of Valsalva width. However, selective coronary angiography would be difficult in this scenario and would have to occur from a diamond above the ostium, given that the supra-annular valve and its covered segment (e.g., sealing skirt) would be above the level of the ostium. A straighter catheter with a short tip, such as a Judkins right (JR) 4, could be used in this scenario, even for left main artery engagement [1].

- With the repositionable Evolut, self-expanding valve it is not possible to determine its final position until after the valve has been released. Theoretically, a commissure can end up positioned directly in front of the coronary ostium. In this scenario, coaxial engagement of the coronary ostia would be challenging, if not impossible. Figure below (Fig. 29.2) shows a theoretical scenario where a repositionable Evolut-PRO self-expanding valve is positioned 4 mm below the annular plane, consistent with the recommended implantation depth of 3–5 mm, and the commissure lines up with the coronary ostia. The three red dots depict coronary ostia heights of approximately 10, 14, and 18 mm above the annular plane, respectively. The cross (X) depicts the closest diamonds that can be used to access the coronaries. It is important to note the width of the sinus of Valsalva determines the space between the valve frame and the coronary ostia; the wider the sinus the more room there is to manipulate a catheter toward the coronary ostia, particularly in the scenario shown in figure below (Fig. 29.2). A narrow sinus would require a very acute angle for the catheter to be pointing toward the

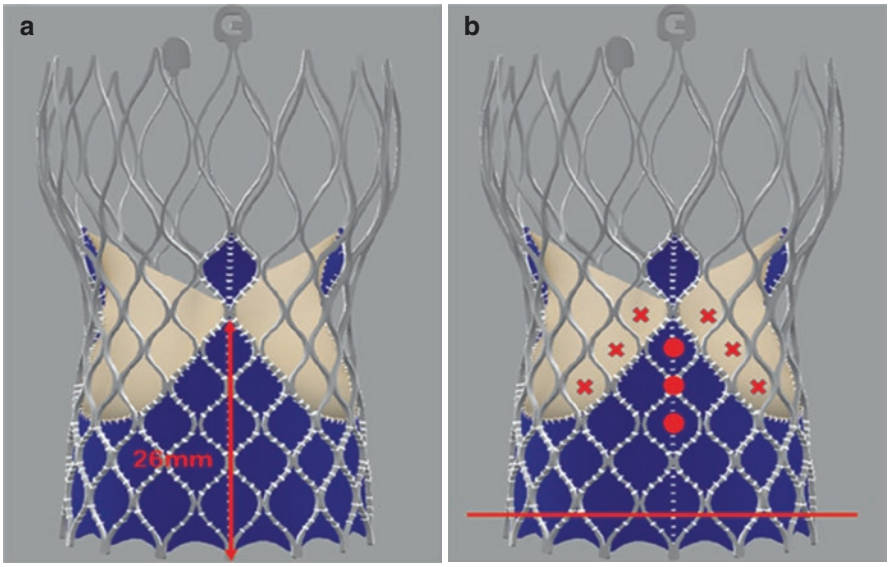

Fig. 29.2 Self-expanding valve and coronary access if sstia lines up with commissural post. Used with the permission from Yudi et al. [1]

ostia for a nonselective coronary angiogram. If selective engagement is required, a coronary wire would have to be manipulated into the coronary artery, and the guide, or a guide extension catheter, would then have to be railed into the ostium. This represents the most difficult scenario: a valve commissure overlying a low coronary ostium in a patient with a narrow sinus of Valsalva. Of course, this description has not accounted for the native aortic leaflet height and severity of calcification facing the left and right sinuses. A tall and bulky leaflet may extend beyond the 13- or 14-mm sealing skirt of the repositionable Evolut-PRO self-expanding valve and would likely further add to the challenge of coronary reaccess based on these scenarios.

- The repositionable Evolut-R self-expanding valve has a concave central portion ("waist") that measures 20–24 mm, depending on the valve size. This is narrower than native aortic root dimensions; therefore, it is not surprising that smaller catheters, such as a JL3.5 or JL3, have frequently been used to engage the LCA. On the contrary, engagement of the RCA can usually be managed with a JR4 catheter. If the sinus width is large, there is a larger distance from the valve frame to the ostium, and thus a longer catheter tip would be required. In this circumstance, JR4.5, JR5, Amplatz right (AR) 2 catheters would be more suitable.

Balloon-Expandable Valve

- The Sapien 3 valve has 12 open cells on its frame, three of which contain the commissures and leaflet attachment, along with a 3-mm pledget in the middle of the upper row of cells. Thus, the commissure may end up directly in front of a

coronary ostium after valve deployment. Another important consideration more pertinent to the balloon-expandable valves is the sinotubular junction (STJ) diameter and height. Because it does not have a narrowed waist like the self-expanding valve, the balloon-expandable valve frame, especially Sapien 3, can extend beyond the STJ, making future coronary access from above the valve more challenging or impossible.

- In figure (Fig. 29.3) below is shown a 29-mm balloon-expandable Sapien 3 valve with the commissural post over the coronary ostia at heights of approximately 10, 14, and 18 mm, respectively, with the valve deployed optimally at 80% aortic position. In this position, only low coronary arteries (<10 mm) would cause any concern, as the skirt on the frame would be around this level. If the valve is deployed in a more aortic position, with the inflow near the level of the annulus, this would complicate engagement further, as the skirt is well above the ostia. Of course, this model has not considered the native aortic valve leaflets. A tall and bulky leaflet may create an even higher barrier for coronary access, given the bulky calcium would be in the way. Coronary angiography and PCI may be undertaken in two ways. First, if the catheter is placed just above the skirt through the upper row of cells, only a nonselective angiogram is likely to be achieved, and PCI would require a coronary wire to engage the artery with a railing technique to engage the catheter. Second, if the STJ is high enough above the valve, a catheter can be used to engage the coronary artery from above the Sapien 3 valve. Coaxial engagement from this position would depend on the sinus of Valsalva width. If the sinuses are effaced, there will be a relative lack of room to manipulate the catheter, and engagement of the artery would be more difficult. In cases where the commissural tab faces the coronary ostium and the top of the Sapien 3 valve frame is in contact with the STJ, it may be necessary to engage from the open cell on either side of the commissural tab to reaccess the coronary artery [1].

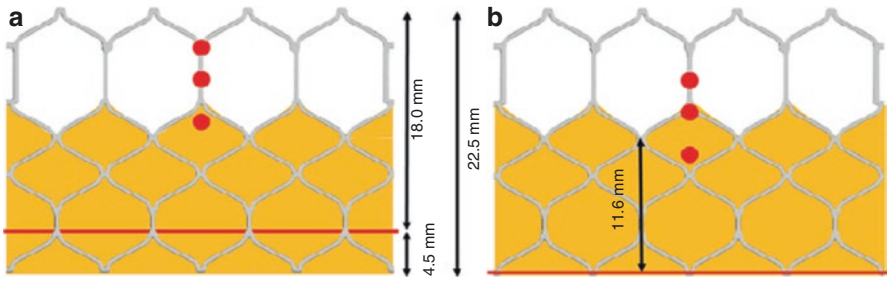

Fig. 29.3 Balloon-expandable valve and coronary ostia based on depth of implant. Used with the permission from Yudi et al. [1]

Systematic Approach to Selecting a Diagnostic Coronary Catheter in Patients with a TAVR

See Fig. 29.4 for algorithmic approach for selecting a diagnostic coronary catheter in patients with a Medtronic CoreValve. Figures 29.5 and 29.6 show the diagnostic angiogram in a patient with CoreValve.

- See Fig. 29.7 for algorithmic approach for selecting a diagnostic coronary catheter in patients with an Edwards Sapien Valve.

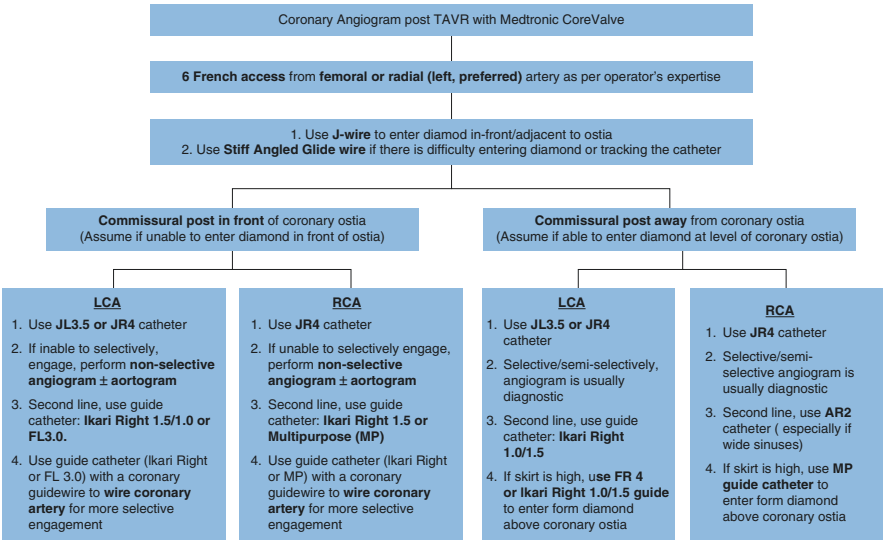

Fig. 29.4 Step-by-step guide for choosing diagnostic coronary catheter in patients with CoreValve. Used with the permission from Yudi et al. [1]

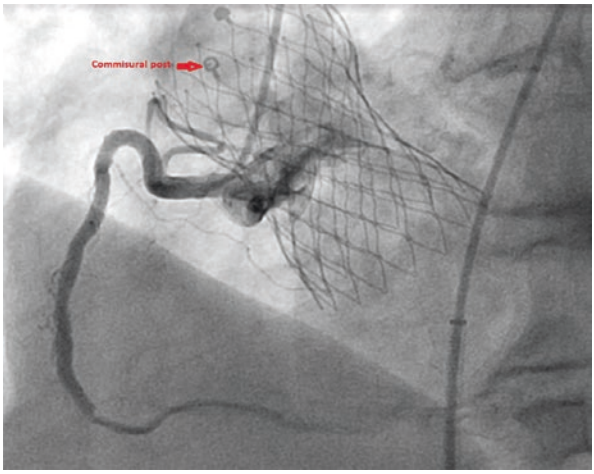

Fig. 29.5 Diagnostic angiogram of RCA using JR 4 catheter via Rt femoral access in a patient with CoreValve and commissural post away from coronary ostia

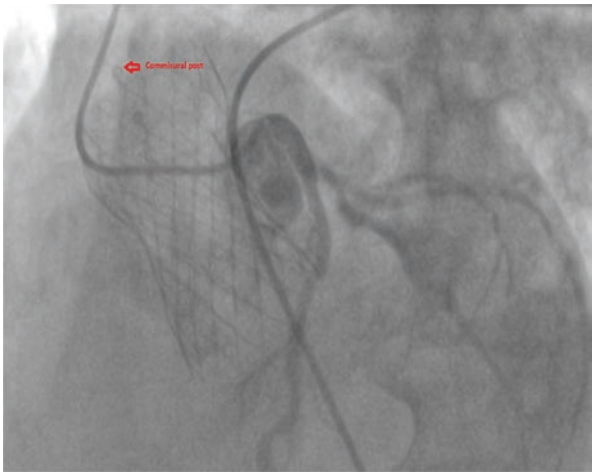

Fig. 29.6 Diagnostic angiogram of LCA using JL 4 catheter via Rt femoral access in a patient with CoreValve and commissural post away from coronary ostia

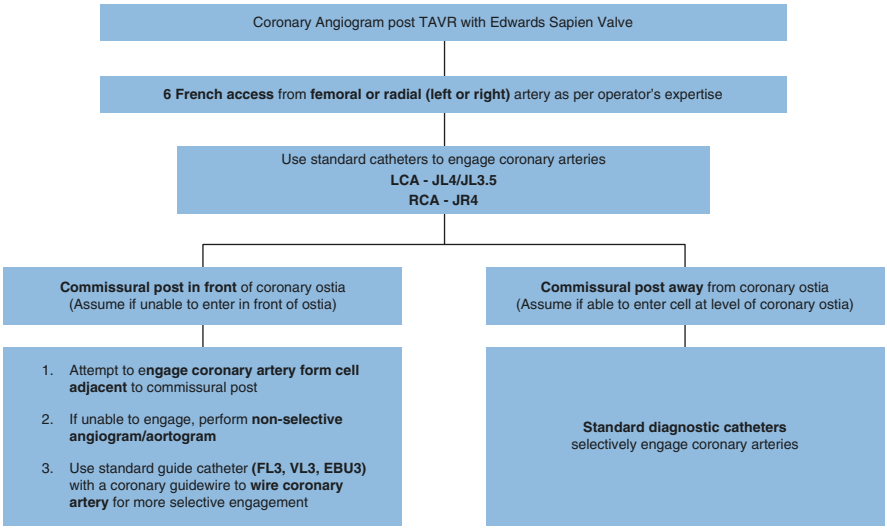

Fig. 29.7 Step-by-step guide for choosing diagnostic coronary catheter in patients with Edwards Sapien Valve. Used with the permission from Yudi et al. [1]

Systematic Approach to Selecting a Guiding Catheter for PCI in Patients with a TAVR

- See Fig. 29.8 for algorithmic approach for selecting a guiding catheter for PCI in patients with a Medtronic CoreValve. Figures 29.9 and 29.10 shows guide engagement using a guide extension catheter in a patient with a CoreValve.
- See Fig. 29.11 for algorithmic approach for selecting a guiding catheter for PCI in patients with an Edwards Sapien Valve.

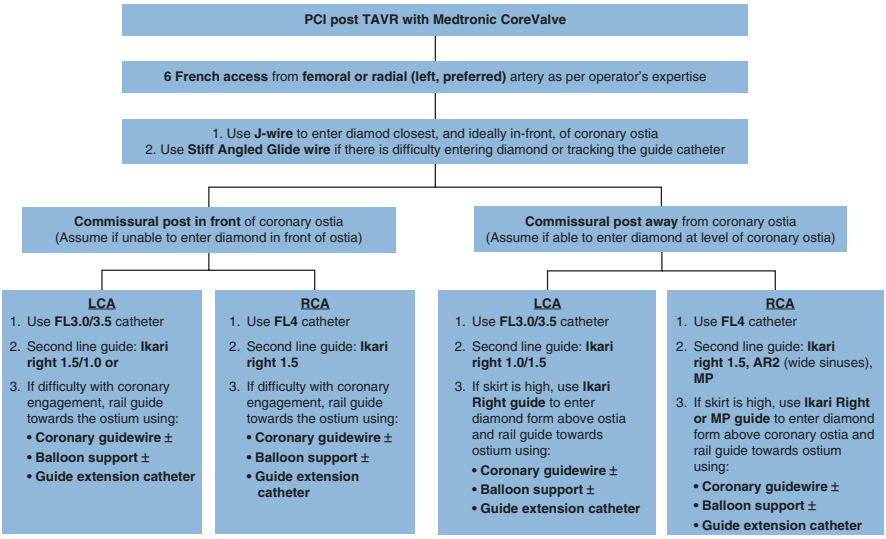

Fig. 29.8 Step-by-step guide for choosing guiding catheter for PCI in patients with Core Valve. Used with the permission from Yudi et al. [1]

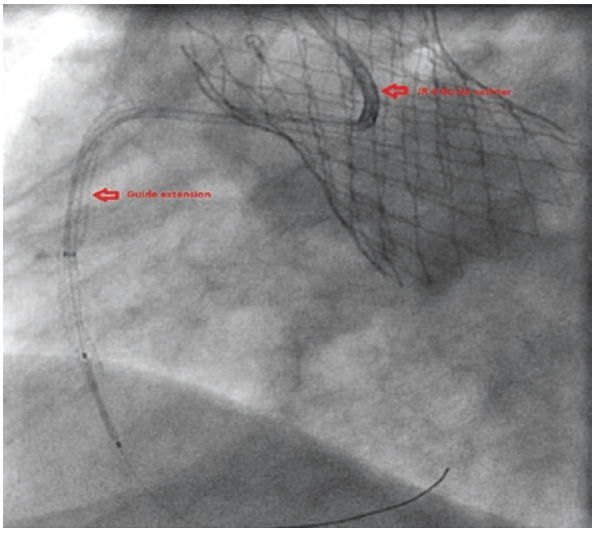

Fig. 29.9 PCI of RCA using JR 4 guide catheter and guide extension via Rt femoral access in a patient with CoreValve and commissural post away from coronary ostia

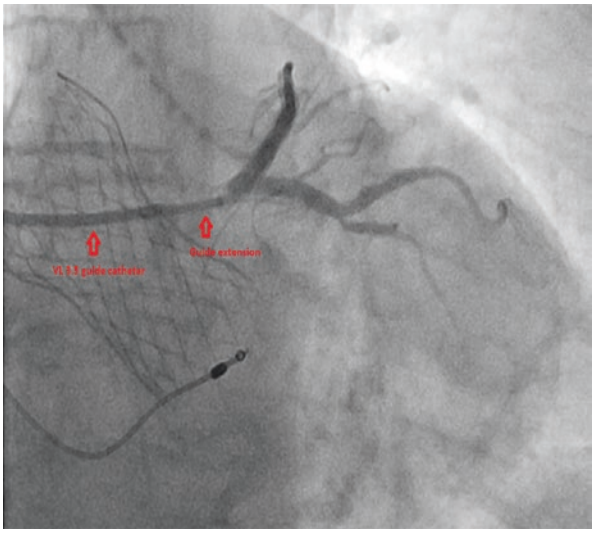

Fig. 29.10 PCI of LCA using VL 3.5 guide catheter and guide extension via Rt radial access in a patient with CoreValve and commissural post away from coronary ostia

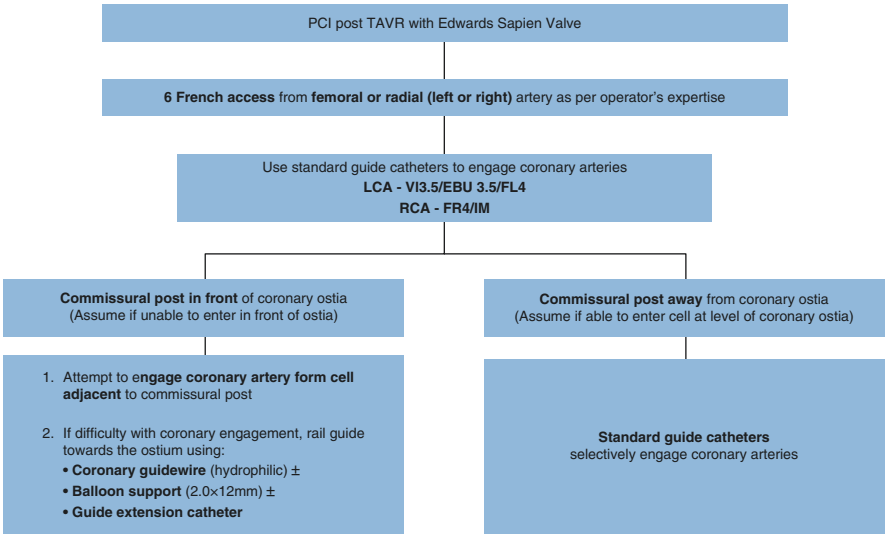

Fig. 29.11 Step-by-step guide for choosing guiding catheter for PCI in patients with Edwards Sapien Valve. Used with the permission from Yudi et al. [1]

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Percutaneous Transvenous Mitral Commissurotomy

30

Nagendra Boopathy Senguttuvan, Gurpreet S. Johal, Samin K. Sharma, and Annapoorna Kini

Introduction

Mitral stenosis (MS) is a progressively disabling disease for which until 1984 open surgery was the only cure. Introduction of percutaneous transvenous mitral commissurotomy (PTMC) by Inoue in 1984 for the treatment of selected MS patients has transformed the treatment of MS. Other names for PTMC include percutaneous balloon mitral valvotomy (PBMV) or valvuloplasty. The decision of PTMC is based on the clinical presentation and imaging study results.

Percutaneous Transvenous Mitral Commissurotomy (PTMC)

Indications

- AHA/ACC guidelines for indications of PTMC is listed in Table 30.1 [1]. A successful result can be predicted by a Wilkins score of ≤8, which is calculated by assigning a point of 0–4 for each of the following four criteria for a maximum score of 16 [2].
  - Leaflet mobility.
  - Leaflet thickening.
  - Leaflet calcification.
  - Sub-valvular thickening.

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Table 30.1 Indications for percutaneous transvenous mitral commissurotomy (PTMC) as per guidelines

Favorable valve morphology in the absence of contraindications	
Symptomatic patients with severe MS (MVA < 1.5 cm^2)[a]	Ia
Asymptomatic patients with very severe MS (MVA ≤ 1.0 cm^2)[a]	IIa
Asymptomatic patients with severe MS (MVA ≤ 1.5 cm^2) who have a new onset of AF	IIb
Symptomatic patients with MVA > 1.5 cm^2 if there is evidence of hemodynamically significant MS during exercise [PASP > 60 mmHg, PCWP > 25 mmHg, mean valve gradient >15 mmHg]	IIb
Considered for severely symptomatic patients with severe MS (MVA ≤ 1.5 cm^2) who have suboptimal valve anatomy and are not candidates for surgery or at high risk for surgery	IIb

[a]Favorable valve morphology (Wilkins score <8) in the absence of contraindications

Contraindications to Percutaneous Transvenous Mitral Commissurotomy (PTMC)

- Persistent left atrial or left atrial appendage thrombus.
- More than moderate mitral regurgitation.
- Massive or bicommissural calcification.
- Severe concomitant aortic valve disease.
- Severe organic tricuspid stenosis or severe functional regurgitation with an enlarged annulus.
- Severe concomitant coronary artery disease requiring bypass surgery.

Preparation

- Labs: CBC, BMP, and coagulation profile.
- Transesophageal echocardiography (TEE): To exclude LAA/LA thrombus and to calculate the Wilkins score.

Equipment

- Sheaths
  - For definite BMV—8 Fr sheath for right venous access, 7.5 Fr sheath for left venous access, 5 Fr sheath for left arterial access, and a 14 Fr Mullins sheath.
  - For possible BMV—7.5 Fr sheath for left venous access and a 5 Fr sheath for left arterial access.
- Catheters
  - Swan-Ganz catheter, 5-Fr pigtail catheter, and Brockenbrough needle.
- Wires
  - 0.035″ standard J-wire, 0.032″ straight wire, and 0.025″ Swan wire.
- Balloon
  - Inoue balloon kit (balloon size is calculated in mm = [Height of patient in cm/10] + 10).

  - 0.025″ curled guidewire/Protrack wire.
  - 14-Fr dilator.
  - Preshaped stylet (LV wire).
- Contrast
  - 50 cc (1:3 dilution).
- Manifolds
  - Three manifolds.
- Heparin
  - 5000 IU.

Vascular Access for Definite BMV

- 8 Fr right FV, 7.5 Fr left femoral vein, and 5 Fr left femoral artery.
- Ultrasound/fluoroscopy-guided right femoral venous access is obtained using micro-access on the right side. RFV access was obtained. Single preclose was performed with one ProGlide device with or without a figure-of-eight suture or a mattress suture.

Vascular Access for Possible BMV

- 7.5 Fr left femoral vein and 5 Fr left femoral artery.

Procedural Steps

- Assess coronary anatomy for patients >40 years of age.
- Perform a complete right heart catheterization first through the left femoral vein.
- Leave the Swan-Ganz catheter in PA for hemodynamic monitoring during the procedure.
- Advance 5-Fr pigtail catheter over J-wire to LV via left FA access and wedge the Swan-Ganz catheter. Record the left ventricular end-diastolic pressure (LVEDP) and pulmonary capillary wedge pressure (PCWP) simultaneously for the transmitral gradient and calculate the mitral valve area (MVA).
- Medrad LV gram (30 cc volume at 20 cc/s at 450 PSI in LAO 45 and RAO 30 positions to evaluate for mitral regurgitation (MR)).
- Pull pigtail into ascending aorta and perform an aortogram in LAO 30 to assess for coexisting AI.
- Place the pigtail in the aorta in the noncoronary sinus.

Transseptal Puncture Using Fluoroscopy

- In AP position, advance 0.032″ wire to SVC via right FV and change to 8-Fr Mullins sheath.
- Remove 0.032″ wire and Mullins sheath dilator and de-air the system.
- Advance Brockenbrough needle (with the third transducer attached) through Mullins sheath and keep the tip of the needle 2 cm below the tip of the Mullins sheath.
- Use the AP view to confirm the correct needle position and pull down the sheath and needle as one unit. Position the Mullins sheath in RA against the interatrial septum (IAS) in the AP position.
- Maintain needle tip direction indicator (at the needle hub) at the 5'o clock position.
- In the AP position, while keeping the needle inside the sheath, position the needle tip by clockwise rotation to the optimal IAS puncture site (clockwise rotation moves the needle tip toward the posterior edge of LA, anticlockwise rotation toward aorta).
- At the midpoint of "end on mitral annulus" (in AP) and the midpoint of anterior and posterior halves of IAS (in LAO), penetrate IAS. Note LA pressure tracing and aspirate bright-oxygenated blood to check for oxygen saturation and/or inject contrast through the needle to confirm location.
- Advance Mullins sheath and dilator together over the needle while holding the needle firmly; counter-clock the entire system so it faces anteriorly during advancement.
- *See* Figs. 30.1 and 30.2 *landmarks of descent for transseptal puncture.*

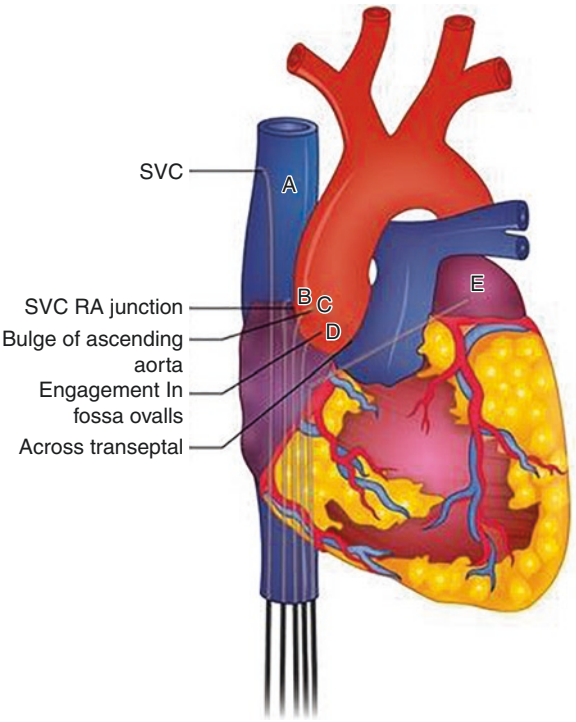

Fig. 30.1 Landmarks of descent for transseptal puncture AP view. (*a*) SVC. (*b*) SVC-RA junction. (*c*) Bulge of ascending aorta. (*d*) Engagement in fossa ovalis. (*e*) Across transseptal puncture

- Withdraw the needle and aspirate blood to check for oxygen saturation.
- Give 5000 IU of heparin once IA is crossed (or 100 U/kg).
- Measure the transmitral gradient using a LV pigtail catheter and Mullins sheath.
- Figures 30.3a–b depicts proper orientation for septal puncture.

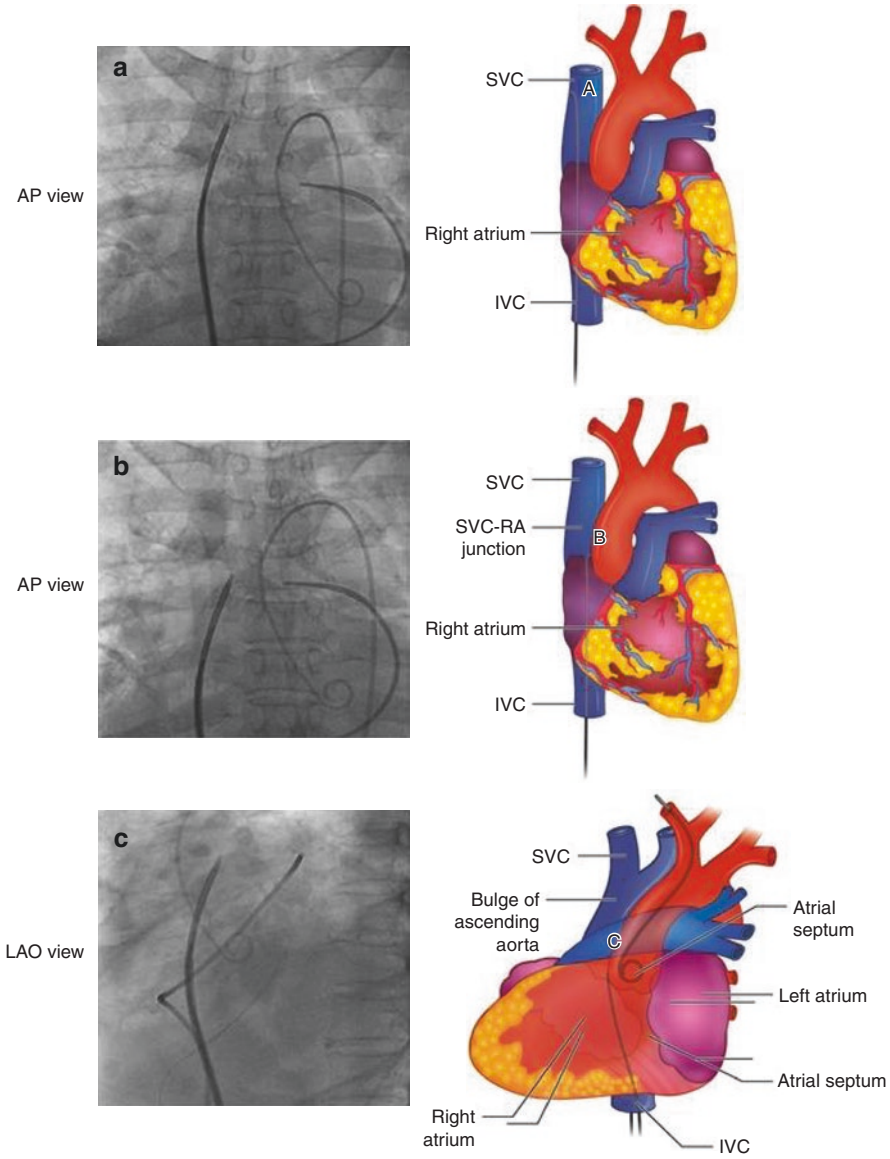

Fig. 30.2 Landmarks of descent for transseptal puncture with fluoroscopy pictures. (**a**) AP view. (**b**) AP view. (**c**) LAO view. (**d**) LAO view. (**e**) LAO view

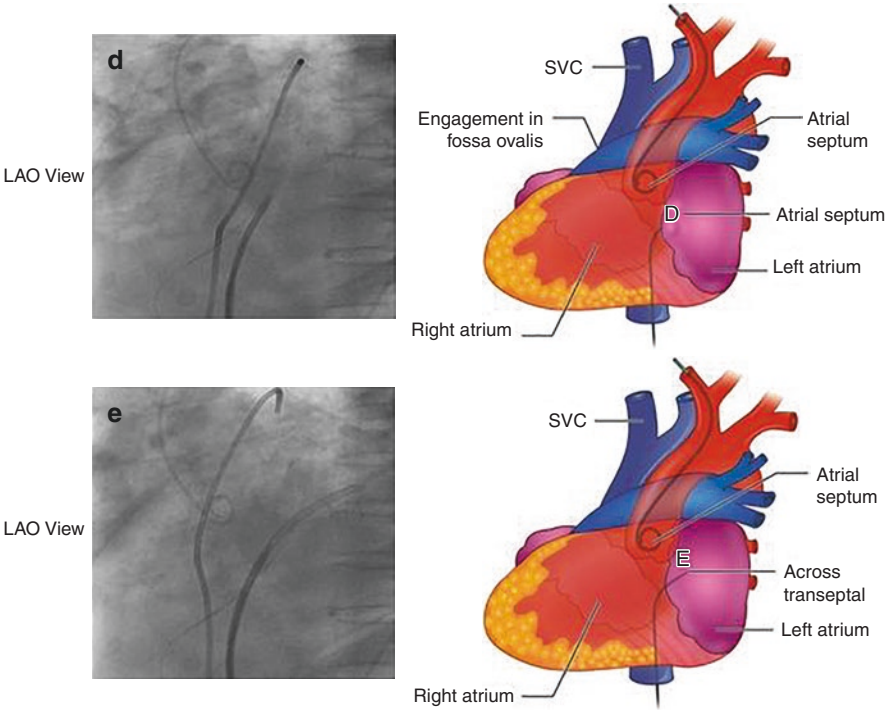

Fig. 30.2 (continued)

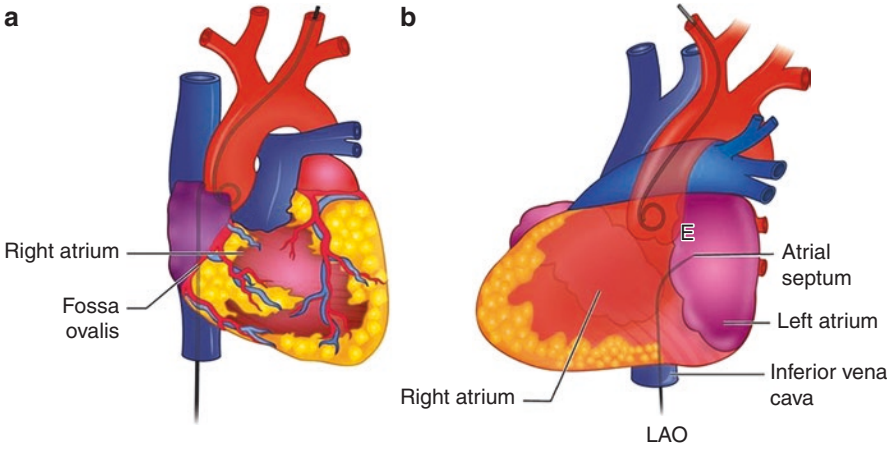

Fig. 30.3 Proper orientation for septal puncture. (**a**) AP view. (**b**) LAO view

Transseptal Puncture Using TEE

- Appropriate position of the patient for TEE.
- Assess severity of Mitral disease/AV disease/TV disease and PA pressure along with LV ejection fraction.
- Rule out LAA/LA thrombus.
- IAS anatomy:
  - Bicaval—Look for thickness/aneurysm/Eustachian valve/PFO.
  - Bicaval X plane; Bicaval shows the superior and inferior segment of IAS, while simultaneous X plane shows aortic short axis revealing the anterior and posterior segment of IAS (Fig. 30.4).
- An appropriate site of septal puncture for PTMC is in the posterior and inferior segment. A LA thrill can be felt with the septal puncture needle. Septal puncture sheath-like SL-1/SL2 or Mullins sheath with Brockenbrough needle or Baylis radiofrequency ablation system can be used for septal puncture. After a septal puncture, the needle alone is introduced first. Later hemodynamics pressure tracing changes to LA pattern from RA. Aspiration of blood from LA confirms bright red, oxygenated blood. Then the dilator is introduced carefully with a counter-clock turn to avoid injury to the posterior LA surface or roof of LA. Then sheath is introduced into LA partially. The later sheath is introduced over the dilator without pushing the dilator further into LA. The position of the sheath

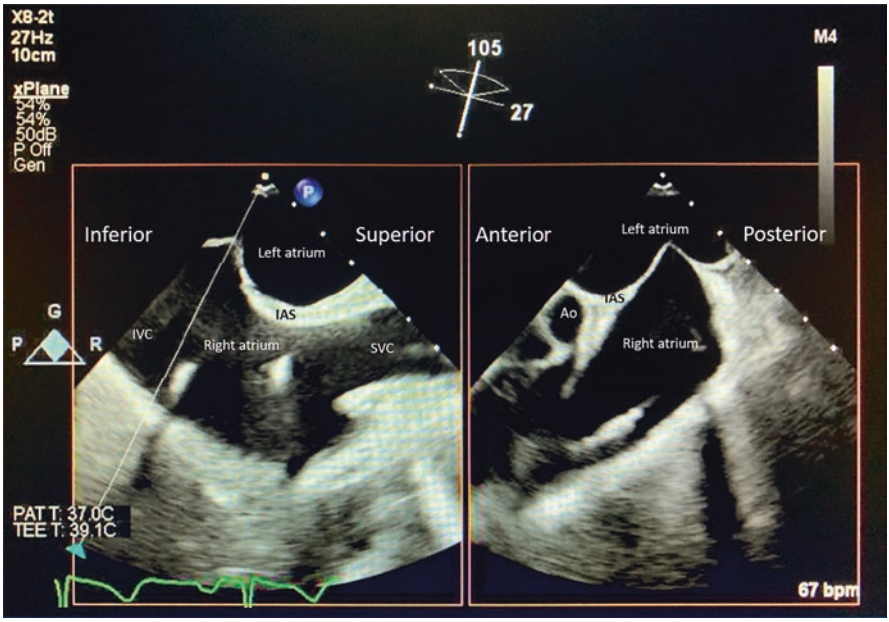

Fig. 30.4 Bicaval view with X plane to aortic short axis showing the position of the needle in the appropriate site

inside LA is confirmed with TEE. Later needle and dilator are withdrawn. LA sheath is flushed with normal saline.

Balloon Preparation

- Open the vent port (short port) and flush the balloon with undiluted contrast.
- Attach the contrast-filled marked syringe to the inflation port (longer port).
- Inflate the balloon to correct size and measure with a measurement gauge. Inject dye through the vent port if the balloon is to be upsized.
- Insert the metal balloon stretching tube (Silver color tube) in the center lumen and lock it into place (metal-to-metal attachment—Silver to Gold attachment followed by gold to the plastic attachment).

Balloon Insertion and Crossing the Mitral Valve

- With the Mullins sheath in LA, now introduce 0.025″ curled guidewire into LA and allow it to coil with the top of the coil against the roof of LA.
- After removing the Mullins sheath, introduce a 14-Fr black color dilator over the guidewire into LA across the IAS puncture site. Also, dilate the groin to allow free passage of the balloon.
- The stretched balloon is now inserted along the 0.025″ curled guidewire to the top of the curve. At this point, the stretching tube is released (loosen metal-to-plastic connection) and withdrawn 2–3 cm, and advanced further into LA following the curve of the wire. Thereafter unfasten the metal-to-metal connection and remove the stretching tube completely at this point.
- Now remove the 0.025″ curled guidewire while maintaining the balloon position in LA.
- Insert the stylet through the center lumen and guide the balloon into LV in the RAO view (counterclockwise rotation of the stylet while gently withdrawing the balloon). Wait for the gentle bobbing movement of the balloon, once it is seen, then the stylet is pulled and the balloon is pushed into LV.
- Once the balloon is across the MV orifice, observe free movement toward the apex.
- Withdraw the balloon partially and push volume into the balloon to inflate the distal end.
- Maintain gentle traction while the inflated portion of the balloon is pulled against the mitral valve.
- Inflate the balloon fully once movement toward the LA stops (Fig. 30.5).
- Once the waist disappeared and the balloon is fully inflated to its predetermined size, immediately deflate the balloon and withdraw into LA. 3D TEE can be done in case of echocardiographic guidance to know the post-PTMC MV area (Fig. 30.6).

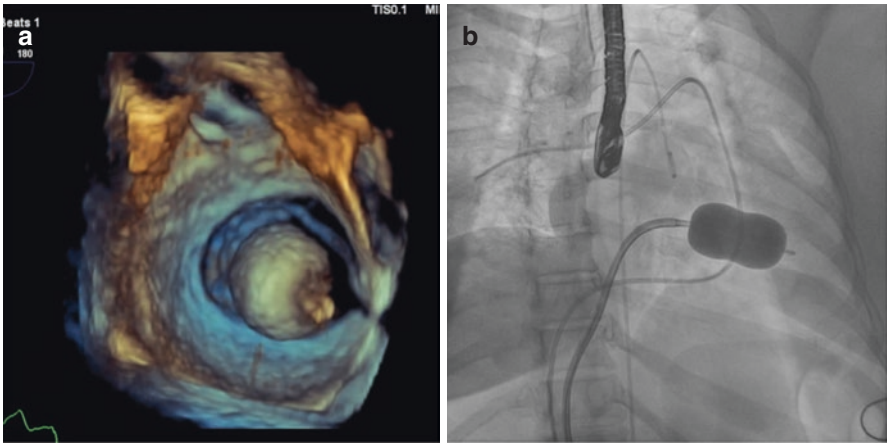

Fig. 30.5 Severe mitral stenosis with MV area of 1 sq.cm. (**a**) That increased to 2.2 sq.cm after PTMC. (**b**)

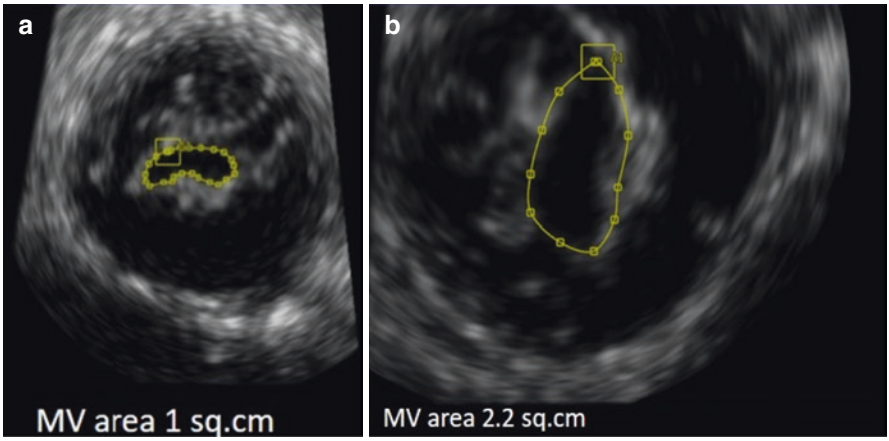

Fig. 30.6 Inoue balloon dilatation of the mitral valve with no waist

- Using the balloon's central lumen and LV pigtail catheter, measure the transmitral gradient.
- Perform Medrad biplane LV gram and compare with pre-procedure LV gram to assess for the worsening of MR. No need for LV gram when the procedure is TEE or ICE (intracardiac echocardiography) guided.
- Document cardiac output (CO), pulmonary artery (PA), and pulmonary capillary wedge (PCW) pressures. If there is no MR and residual mitral gradient >5, repeat the balloon dilation, after increasing the balloon size by 1 mm by injecting contrast via the vent port.
- Admit patient to coronary intensive care unit (CCU) or telemetry for observation and restart anticoagulation if indicated.

Complications and Their Management

- Severe MR, which occurs in 2–4% of patients.
- A large atrial septal defect (greater than 1.5:1 left-to-right shunt) occurs in fewer than 12% of patients with the double-balloon technique and fewer than 5% with the Inoue balloon technique. Smaller atrial septal defects may be detected by transesophageal echocardiography in larger numbers of patients.
- Perforation of the left ventricle (0.5–1.0%).
- Embolic events (0.5–3%).
- Myocardial infarction (0.3–0.5%).
- The mortality is 1–2%, but in carefully selected cases at experienced centers, it is <1%.

Post-Procedure Care and Follow-Up

- As needed based on the severity of MS and clinical status of the patient. Most patients can be discharged the next day.
- Coumadin if necessary for other reasons like atrial fibrillation, to be started or restarted the next day (at least 24 h after the procedure to reduce access site bleeding). If a patient is not on Coumadin, aspirin 81 mg per day to be started daily for the next day.

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Transcatheter Edge to Edge Mitral Valve Repair

31

Nagendra Boopathy Senguttuvan, Parasuram Krishnamoorthy, Gilbert H. L. Tang, and Annapoorna Kini

Introduction

An increase in the aging population has lead to an increase in the burden of heart failure and the prevalence of mitral regurgitation (MR). Surgical intervention for primary MR remains the gold standard of care. Minimally invasive mitral valve therapies for both primary and secondary mitral regurgitation are likely to grow in high-risk patients. A heart team approach is a well-established concept in managing such patients. Transcatheter edge-to-edge mitral repair using MitraClip (Abbott Structural Heart, Santa Clara, CA) is increasingly utilized in such patients [1].

MitraClip

Indications

- Primary Mitral Regurgitation [2]
  - In patients with moderate-severe to severe MR who have prohibitive surgical risk.
- Secondary Mitral Regurgitation [3]
  - In patients with moderate-severe to severe functional MR who are symptomatic (NYHA Class II–IVa) despite maximally tolerated guideline directed medical therapy (GDMT) (that includes ACEi/ARBs/ARNI, beta-blockers, spironolactone, ivabradine, and diuretics) evaluated by a heart failure specialist, cardiac resynchronization therapy (CRT) (in indicated patients) and

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coronary revascularization, can be considered as a potential candidate for MitraClip therapy, if anatomy is suitable [1].

TEE Views for MitraClip

- Optimal TEE imaging is mandatory for the MitraClip procedure. It helps in the evaluation of mitral valve anatomy, MR jet location and direction, and strategy on transseptal access and leaflet grasping. Various parameters that are assessed in pre-procedural TEE are mentioned below.
- Baseline:
  - Appropriate positioning of the patient for TEE imaging to obtain optimal image quality.
  - LV ejection fraction.
  - Rule out LAA/ LA thrombus.
  - Start from 0°; 4 and 5 chamber view with and without color:
  - Assess the severity of MR.
  - Origin of jet and direction of the jet.
  - Bi-Commissural view (bicom) with and without color: LV apex at 6 ‘o clock position with both papillary muscles on either side of MV.
  - Bi-Commissural view with Xplane to LVOT view to determine optimal grasping views
    Panning with the cursor from medial to lateral to assess A3P3, A2/P2, and A1P1 segments anatomy and jet location.
    Accessing for worst PISA for clip(s) implantation.
    Leaflet lengths in LVOT view at grasping target for Clip implantation and selection.
    Calcification in leaflet tip or annulus if any.
    Annular dimension along the anterior-posterior axis in LVOT view.
    Posterior LV wall motion towards PML.
- 3D en-face view of the mitral valve with and without color showing surgeons view of MV with aorta at the top of the screen; LAA at 9′o clock position and two mitral commissures at 2′o clock and 10′o clock positions. It shows the pathology of the mitral leaflet, the jet location, and the site of clip implantation.
- IAS anatomy:
  - Bicaval—Look for thickness/aneurysm/Eustachian valve/PFO.
  - Bicaval X plane; Bicaval shows the superior and inferior segment of the IAS while simultaneous X plane shows the aortic short axis or 4-chamber revealing the anterior and posterior segment of the IAS.
- Baseline gradient across MV/baseline pulmonary venous flow pattern in both right and left pulmonary veins/assess the severity of other valve pathologies/ assessing RV function/pulmonary artery hypertension/tricuspid regurgitation.
- Offline calculation: EROA, regurgitant volume/vena-contracta.
- A MitraClip information sheet that is shown in the annexure helps both the interventionists and the echocardiograph to grab all possible information during TTE/ TEE. TEE guidance and appropriate views during the procedure are discussed in the step-by-step section.

Step-by-Step MitraClip

Septal Puncture

1. Reduce tidal volume and increase respiratory rate. Maintain HR <80 bpm
2. Ultrasound/fluoroscopy guided right femoral venous access is obtained using microaccess. RFV access was obtained. Single preclose was performed with one ProGlide device along with a figure of eight suture or a mattress suture for external hemostasis.
3. 2000 U of IV heparin is given.
4. An 18 F sheath is placed with the support of a stiff wire. Serial dilatation of the groin can be done for difficulty in inserting a sheath.
5. Different transseptal sheaths (SL-1, SL-2, and Mullins sheath) and septal puncture systems (Brockenbrough needle, Baylis RFA system) are available. Selected sheath is introduced through the 18 F sheath into superior vena cava (SVC) with the support of 0.032 Teflon J wire. TranseptalAID developed by our team will help operators to learn more about transseptal puncture [4].
6. Fluoroscopy goes to RAO 20 from here onwards. After septal puncture in posterior and mid fossa (Fig. 31.1) with the puncture point to mitral leaflet coaptation distance being at least 4 cm at prolapse/flail segment level or annular level for restricted pathology, needle alone is introduced first. Later hemodynamics

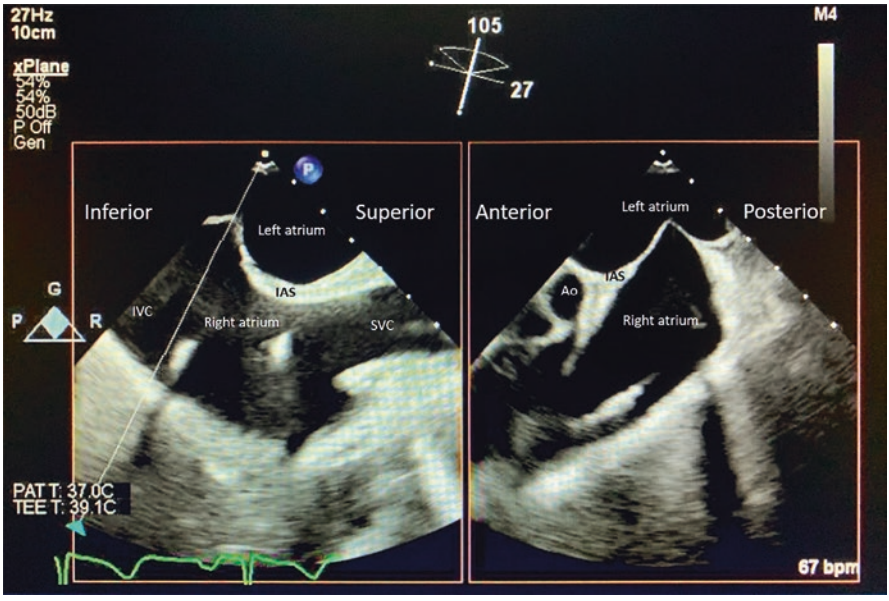

Fig. 31.1 Bicaval view with X plane to aortic short axis showing the needle position in the appropriate site

pressure tracing changes to LA pattern from RA. Then the dilator is introduced carefully with a counter clock turn to avoid injury to the posterior LA wall or roof of LA. Then sheath is introduced into LA partially. Later, sheath is introduced over the dilator without pushing the dilator further into LA. Position of sheath inside LA is confirmed with TEE. Later needle and dilator are withdrawn. LA sheath is flushed with normal saline.

7. Remainder of IV heparin is given to achieve target ACT >300 s.
8. A stiff 035 wire is introduced into LA (Confida) through the sheath. Then the transseptal sheath and 18F introducer sheaths are removed.

Insertion of Steerable Guide Catheter

9. Steerable guide catheter (SGC) is inserted in a negative deflection into the femoral vein with the support of Confida wire up to RA (Fig. 31.2).
10. Turn +/− knob on SGC back to neutral and enter into LA. If resistance, corkscrew in gently or turn SGC knob (−) negative to straighten SGC to give better 'pushability'. Use fluoroscopy and echocardiogram to help. In case the dilator or guide does not go, then balloon dilatation (usually 10–12 mm × 40 mm balloon) of the interatrial septum is necessary.
11. Once the SGC and dilator are parked in LA, ask the echocardiographer to show the guide. SGC is parked on the stabilizer without any gap between the proximal SGC and the stabilizer portion. A screw should be used to anchor the SGC on top of the stabilizer. A Ridged appearance in the echo arises from the dilator. With the wire still in the LA, the dilator alone comes out (check with TEE) gently into the guide.

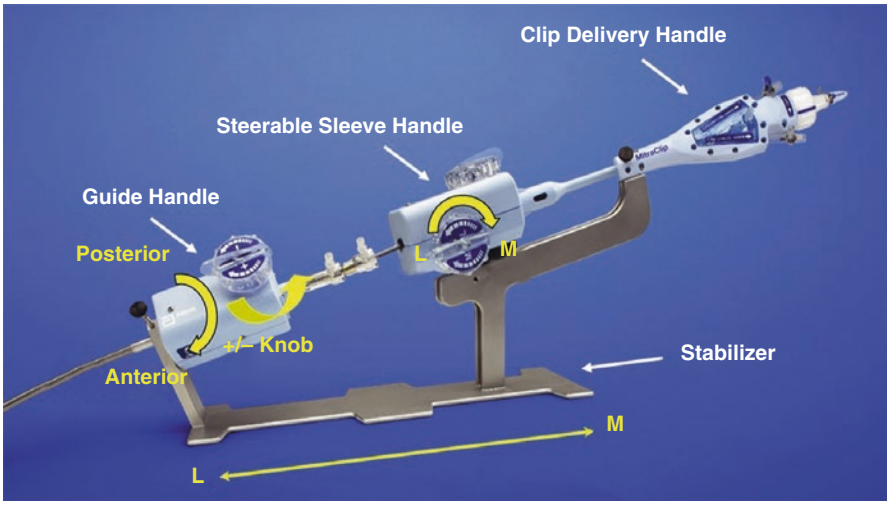

Fig. 31.2 MitraClip system showing steerable guide catheter and catheter delivery system

12. After ensuring at least 1 cm of the 'steerable' guide catheter is inside LA, wire is pulled back into the dilator. Later both dilator and wire come out with wire inside the dilator—with a gentle negative suction at the SGC port to avoid air entry. Once the wire and dilator are outside, the Y connector port of the SGC is closed with a finger while gentle aspiration is continued through the external port. Then the SGC external port is connected to the pressure line to monitor LA pressure.

Insertion and Steering of CDS

13. Clip delivery system (CDS) (steerable sleeve catheter and handle catheter with clip) is inserted into SGC with a 'waterfall': blue-to-blue alignment is maintained (Fig. 31.3) until it is locked with the SGC system. This is essential for 1:1 torque movement in the required direction with the movement of knobs. CDS is advanced through the SGC (Fig. 31.4) in specific, controlled hand movements. This will avoid sudden jerky movements of CDS inside the LA.
14. Once the Clip is inside LA, ask the echocardiographer to follow the clip. Usually, both can be seen in aortic valve short axis view at 45°–60°, mid esophageal TEE View. In case we lose space to straddle, the entire system with the stabilizer can be retracted together slightly backward using fluoroscopy and echocardiogram to enable straddling (Fig. 31.5). Once straddling is done, the clip needs to be steered to make it perpendicular to the MV annular plane. M (Medial) knob movement, posterior rotation of SGC (clockwise movement of SGC) usually gets the CDS perpendicular to the MV. In the case of an aorta hugger position, a + knob movement on SGC is required with gentle anterior torque with SGC and lateral movement of the stabilizer to advance the system. During steering, the clip handle may be exposed out of the sleeve guide catheter. Loosen the screen, and retract the clip handle gently backward to remove the slack. Also, keep an eye on the straddle to avoid under/over straddle. MPSS is the pneumonic that helps to remember the knobology movement (Medial, Posterior, Straddle, Sleeve).

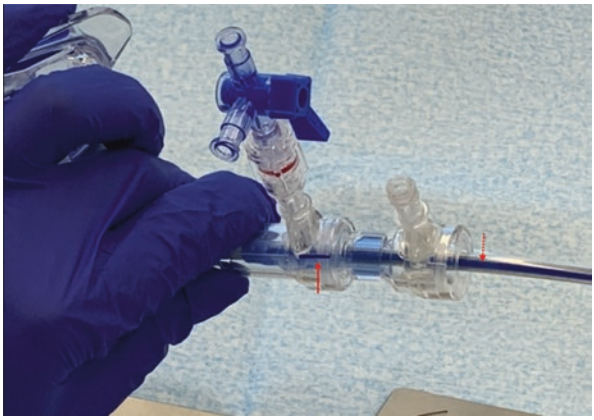

Fig. 31.3 Clip delivery system (CDS) should be inserted into steerable guide catheter with a "waterfall" showing *blue*-to-*blue* alignment between SGC (continuous *red arrow*) and CDS (broken *red arrow*)

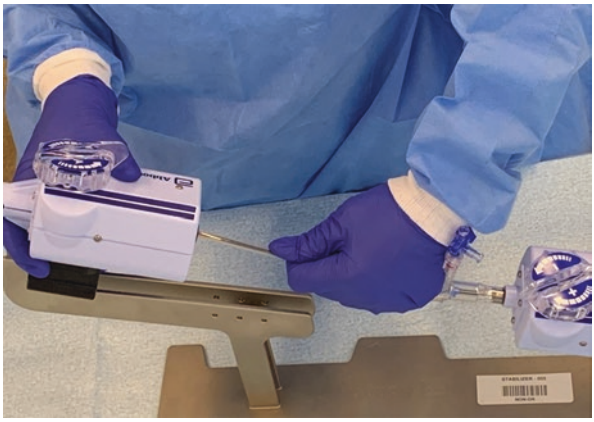

Fig. 31.4 Position of hands when advancing the catheter delivery system into the LA

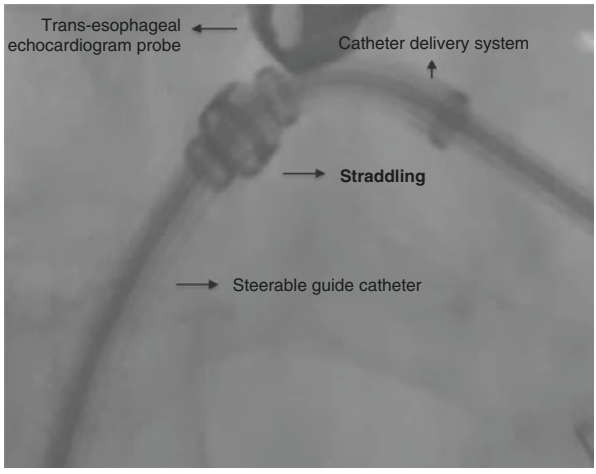

Fig. 31.5 Catheter delivery system straddled across steerable guide catheter

15. Now we need to keep the clip perpendicular to the line of coaptation (LOC) using fluoroscopy and TEE. Need to use Bicom Xplane to LVOT view, 3D en-face view to ensure the same. In Bicom, single clip arm is seen while in LVOT, both the closed clip arms are seen.
16. Before opening the clip arms to 60, we need to remove the tension in the system by doing a 'jiggle' motion of the clip handle. Jiggle movements should be very controlled in a clockwise or counterclockwise fashion to ensure that the clip is perpendicular to the LOC on TEE.

Optimize Clip Orientation

17. Gripper line up—until blue line slowly, just after blue line is fine.
18. Lock line up—until blue line slowly, just after blue line is fine.
19. Open clip arm—arm opener to 60°.

20. Jiggle movement of CDS handle catheter in a clockwise or counterclockwise direction (controlled fashion) to loosen up the tension in the system and on RAO view take the parallax out of the Clip arms while on 3D to confirm orientation.
21. Confirmation of clip arm position to MV plane: In fluoroscopy, CDS should be 90° angle to the SGC for optimal "M." On TEE Bicom view, the shaft of CDS should be perpendicular to MV annular plane. Watch out for Over "M" and the shaft turned >90° and slanted against the mitral annulus.

Ready to Enter into LV

22. Check trajectory by advancing the CDS slightly forward towards the MV annulus without crossing the valve. Should optimize trajectory typically with (+) knob to at least 60°–90° and anterior SGC torque to move the Clip more anterior. May need to remove some "M" to maintain a perpendicular trajectory of Clip to MV annulus in Bicom view.
23. When entering LV, left hand should hold the SGC while the right hand holds the clip handle (Fig. 31.6). We may need to close the Clip during the LV entry of the Clip near commissures or large flail to avoid getting the leaflet caught against the gripper or if it is a second clip. On fluoroscopy, check to make sure Clip has not rotated; may need to go opposite in rotation on CDS handle vs before (e.g., if clockwise before to orient Clip, may need to go counterclockwise to avoid Clip spinning as it enters LV). In addition, check on fluoro the CDS shaft remains straight instead of medial or later deflection. If so, may need to adjust the M knob to correct.

Grasping Leaflets in LV

24. Open clip arm to 150°.
25. Check Clip arm orientation on 3D on TEE by dropping the gain.

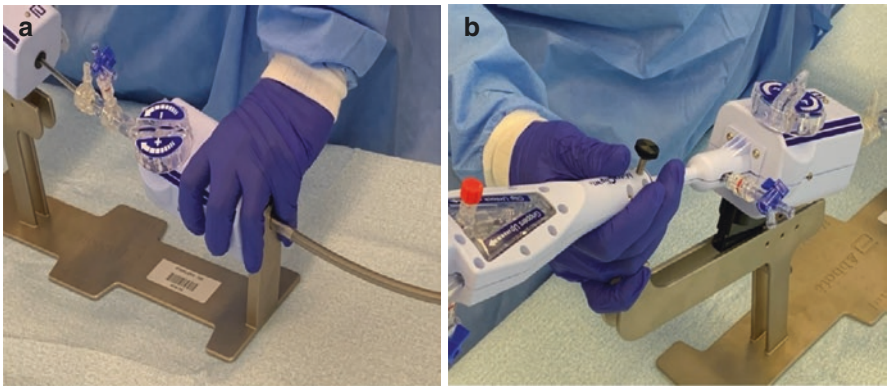

Fig. 31.6 Position of hands on steerable guide catheter (**a**) and catheter delivery system (**b**) during LV entry

26. Pull Clip fastener up under TEE gently. Try to grasp the anterior mitral leaflet first followed by the posterior mitral leaflet. Gentle and steady posterior torque of SGC is essential to grasp the posterior leaflet. Keep HR at 50–60 with tidal volume around 300–350 cc.
27. Continue to optimize grasping by slight SGC posterior/anterior torque, maintaining Clip orientation on fluoroscopy, some "M" or "+" knob adjustment if needed.
28. Ensure capture of both the leaflets over the clip arms in Bicom view X-planed to LVOT view. Grippers down in diastole slowly. Look for gripper bounce on both AML and PML. Let the echo person know! Slowly close the Clip arm to 60°; may need to advance CDS handle with slightly posterior SGC torque to reduce leaflet tension and maintain leaflet insertion especially in restricted pathology (secondary MR).
29. Ensure adequate leaflet capture before closing the clip further; avoid multiple closures to prevent leaflet damage.
30. Lock the Clip. Close the Clip under color on Bicom/LVOT views; may need to advance CDS handle to further reduce leaflet tension +/− slight posterior SGC torque especially in case of secondary MR and short posterior leaflet.

Assessment of Clip After Grasping

31. Check leaflet insertion by measuring residual leaflet length vs baseline, and immediately adjacent (lateral, medial) to the Clip.
32. Check MR reduction, MV gradient, PV flow pattern. Check LAP/V wave changes in hemodynamic tracings, On TEE use 4-chamber, BiCom X plane to LVOT, and 3D en-face with and without color.

Clip Deployment

33. Once the interventionist and echocardiographer are convinced about a good grasp with an appropriate reduction of MR and improvement in hemodynamics, Clip will be deployed.
34. Go to LAO 30°–45° to visualize both Clip arms; ensure arm opener is not able to open clips arms—by going to neutral and opening to 270° arc; Look for whether clips are opening are not: They should not open up. In case they open up, then open the lock arm again and close it; and redo the same to make sure that the clip arms do not open up.
35. Then turn the knob clockwise tight to close the clip tightly.
36. Drips on; Grippers cap off—gripper line out—floss in the same direction parallel to the Gripper lever; Floss both the lines.
37. Lock line cap out; unwind the two strands against the rubber cap; floss both the threads; remove lock line thread slowly.
38. Ensure arm opener is not able to open clips arms—by going to neutral and opening to 270° arc; Look for whether clips are opening are not: They should not open up; Then go back to neutral.

39. Remove the safety pin.
40. Open arm opener—Expose the grooves.
41. Go for at least eight rotations with the actuator knob; then pull the pin.
42. At the same time as the eight rotation-turn, unscrew the clip handle screw; retract the CDS handle backward slowly to free the needle from the Clip to deploy the Clip. Watch under fluoroscopy and TEE; if the needle is too posterior or anterior, try to center; this happens when a lot of posterior SGC guide torque is in place.
43. Gripper line thread comes out slowly under Fluoroscopy to make sure the pin is not pulled towards the valve and Clip to risk interaction.
44. Two kinds of Clip deployment:
    (a) Gripper line first: In case of a thin leaflet, small LA, not much slack remaining between the pin and the CDS; Gripper line is removed slowly first after informing the echocardiographer before removing the Pin; later rest of the steps happen.
    (b) IFU method: Here Gripper line is removed at the end.
45. Undo the "+" knob and gently remove the "M" knob as you retract the CDS back into the SGC under TEE and fluoroscopy guidance.
46. Once CDS inside SGC; remove the CDS and apply gentle negative suction at the SGC external port gently to avoid air entrapment. Look for the silver circle in the CDS as you remove the CDS. Once it is visible, the CDS is removed en-bloc with its introducer.

Assessment After First Clip

47. Assess the grasp by looking at the tissue grasped by MitraClip in 3D echo along with the severity of MR. Effect of MitraClip on LV inflow obstruction should be assessed by measuring the mean gradient across the MV. Also effect of MitraClip on pulmonary venous flow pattern that either should become blunted or is normalized after clip. In addition, LA pressure measured by SGC should drop-down. In general, MR should drop down at least 2 grades from baseline with an acceptable MV gradient (mean MV gradient <5 mmHg). If MR is more than 2+ with mean a MV gradient <5 mmHg, an additional clip can be contemplated if anatomically feasible. If it is decided to proceed with a second clip, steps 13–23 are repeated. An important step is to keep the clip closed while entering into LV from LA. Need to take the help of fluoroscopy to get the second clip parallel to the first clip. Often second clip is much faster than the first clip and it makes the first clip more stable. Repeat the same anatomical and physiological assessment to determine if a third clip is necessary.
48. If an additional Clip is not needed, then remove SGC, assess ASD size and shunt direction, remove the SGC with two hands slowly, then perform groin closure. Usually, the ASD does not need to be closed; ASD closure can be considered before removing SGC in case of a right to left or bidirectional shunt ASD, severe RV dysfunction, severe tricuspid regurgitation, or severe pulmonary hypertension.

Annexure: MitraClip TTE/TEE Information Sheet

Name:

Age/Sex:

MRN:

Qual. of Echo: Good/Fair/Poor

MR Pathology: Primary/Secondary/Mixed

LVEF:	LVEDD:	LAA: Thrombus/LA Thrombus
MVA:	MV Gradient:	EROA:
VC:	PV Flow Pattern:	Rt: Reversal/Systolic blunting
RVol:	RF:	Lt: Reversal /Systolic blunting

E-velocity: PAH: >70/<70 TR: Mild/Moderate/Severe

RV Function:Normal/Mild Dysfunction/Moderate Dysfunction/Severe Dysfunction

Doppler MR: Length of MR Spectrum:

Annulus Length in Short Axis:

Anatomy No of Jets:1/2/3

Origin of Jet: Central/Medial/Lateral/Anterior/Posterior

Direction of Jet: Central/Medial/ Lateral/Anterior/Posterior

Flail Length/Depth:

Co-Aptation Length/Depth:

Tethering: PML/AML/Both

Tethering: Asymmetric/Symmetric

Prolapse: AML/ PML-Scallop involved:

Pseudo prolapse: AML/PML-scallop involved:

IAS anatomy: Thin/Thick/LSH/Eustachian Valve/Chiari network

Septal puncture: Good/ fair/poor

COAPT: Satisfied/Not Satisfied COAPT TIER:1/2/3

EVEREST criteria Satisfied: Yes/No

IAS anatomy: Thin/Thick/LSH/Eustachian Valve/Chiari network

Septal puncture: Good/ fair/poor

Plan :

Site of clipping:

Mitraclip:NTR/ XTR

Possible no of clips:

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Alcohol Septal Ablation

32

Parasuram Krishnamoorthy, Gurpreet S. Johal, and Annapoorna Kini

Introduction

Alcohol septal ablation [ASA] is a percutaneous, minimally invasive procedure performed by interventional cardiologists in carefully selected hypertrophic cardiomyopathy patients who meet strict criteria and are severely symptomatic despite maximal medical therapy [1, 2]. Ablation induces localized myocardial infarction and thinning of the basal ventricular septum, thereby leading to a reduction in dynamic outflow obstruction.

Hypertrophic cardiomyopathy is the most common inherited cardiomyopathy characterized by severe left ventricular hypertrophy without a secondary cause. Prognosis is usually favorable but a small subset of patients are at risk for sudden cardiac death and severe congestive heart failure [1].

Alcohol Septal Ablation

There is no randomized control trial comparing ASA vs surgical myectomy (SM), although a recent meta-analysis has shown that ASA is associated with lower periprocedural mortality and stroke but higher rates of pacemaker implantation and reintervention [3]. A recent single-center study showed that ASA is a safe procedure with ongoing symptomatic improvement and excellent long-term survival and can be considered as a reasonable alternative to surgical myectomy in hypertrophic cardiomyopathy patients [4]. A recent reappraisal of ASA therapy for hypertrophic cardiomyopathy listed clinical criteria while selecting between ASA and myectomy in the decisional algorithm (Table 32.1) [5]. Central Illustration Fig. 32.1 shows a

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Table 32.1 How to choose between alcohol septal ablation and surgical myectomy

Alcohol septal ablation	Surgical myectomy
Adults and elderly	Children and adolescents
Septal bulge	Mitral valve intervention
Right bundle branch block	Left bundle branch box
Previous cardiac surgery	Low operative risk
Expert interventional team	Expert surgical team

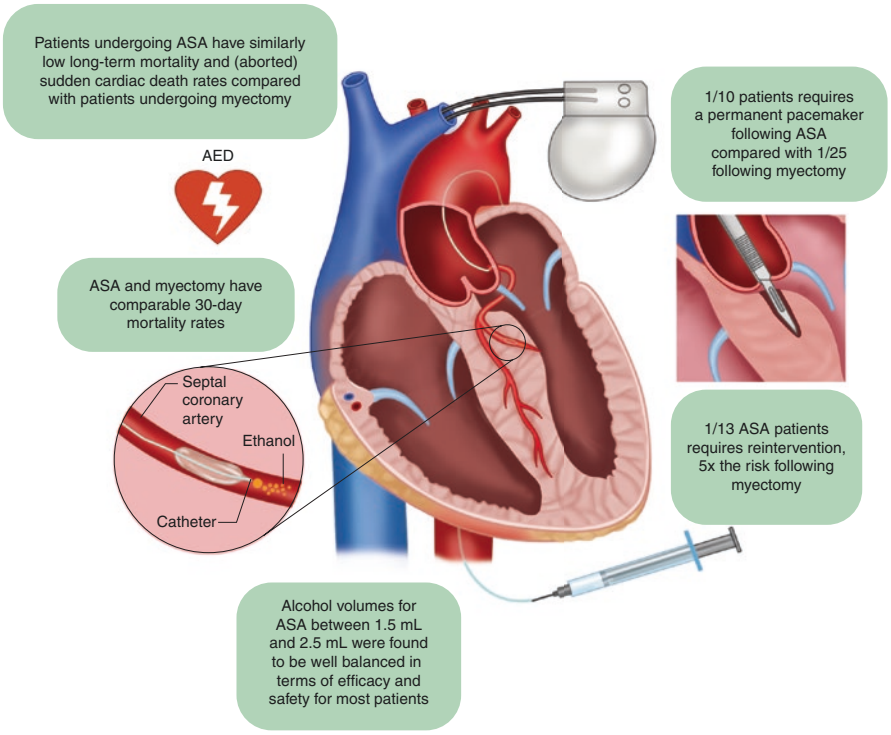

Fig. 32.1 Central Illustration of the alcohol septal ablation (ASA) procedure and key messages on outcomes following ASA to help facilitate shared decision-making

schematic description of the ASA procedure and key characteristics of outcomes following ASA to help facilitate shared decision-making [6].

Criteria

- Severe, drug-refractory cardiac symptoms (NYHA/CCS III/IV dyspnea/angina or syncope).
- Dynamic LVOT obstruction (gradient ≥50 mmHg at rest or ≥50 mmHg with provocation).

- Absence of significant intrinsic mitral valve disease.
- Ventricular septal thickness of ≥15 mm and systolic anterior motion (SAM) of the mitral valve.
- Sufficient basal septal thickness to safely and successfully perform ASA.

Access

- Right common femoral artery (alternatively, radial): 6-Fr sheath.
- Left common femoral artery (alternatively radial): 5-Fr sheath.
- Right internal jugular vein: temporary pacemaker (if the patient does not already have a permanent pacemaker (PPM)).

Equipment

- 6-Fr sheath and 6-Fr left coronary guide catheter of choice.
- 5-Fr multipurpose catheter (MP).
- Temporary pacemaker (with sheath and locking cover).
- 0.014″ guidewire (Fielder™ or Whisper™).
- 100% absolute alcohol.
- Compliant, (OTW) balloon (1.5 × 9 or 2.0 × 9 mm).
- Intraprocedural transthoracic echocardiography (TTE) and definity contrast injection available.

Hemodynamic Assessment

- Simultaneous pressure measurement of LV outflow region and ascending aorta (assess for gradient via pullback with MP catheter from LV apex to LV outflow tract).
- Assess for Brockenbrough-Braunwald-Morrow sign (see Fig. 32.2)—drop in post-extrasystolic aortic pulse pressure [7].

Technique

- Place a temporary pacemaker (if no permanent PM) via the right internal jugular vein. The temporary pacemaker should be left in place for at least 48 h post-ablation.
- Draw baseline CPK, CK-MB, and troponin levels.
- Perform coronary angiography to determine the most appropriate septal artery for ablation and to evaluate for CAD.
  - Both coronary arteries should be assessed as the proximal RCA can give rise to basal septal arteries.

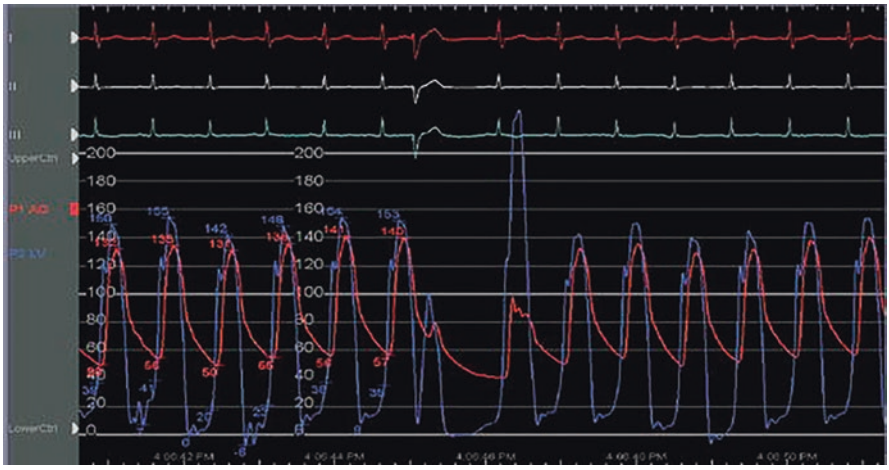

Fig. 32.2 Brockenbrough–Braunwald–Morrow sign

  - *RAO* (*straight and caudal*) projection: Angulation of origin of the septal artery; cranial projection: Length of septal and course of the septal artery.
- Use MP catheter in LV via second arterial access to measure resting and post-PVC LV gradient.
- Administer Angiomax with goal ACT >300 s.
- 6-Fr guide:
  - Wire the first major septal artery.
  - Negotiate OTW balloon to most basal septal branch.
  - Inflate over-the-wire balloon (size according to septal artery diameter) to 5–6 atm and perform cine angiography of LAD to confirm no compromise of LAD flow and balloon position.
  - Inject DEFINITY™ contrast (dilute 0.8 mL of DEFINITY into 10 cc saline, then draw up 1 cc into a small syringe and inject 1 cc at a time) while the balloon is inflated. The goal is to avoid enhancement of RV, free walls, or papillary muscles and to delineate the septum. Withdraw the guidewire out of the OTW balloon underwater seal.
- Inject absolute alcohol (100% absolute, 1–3 mL) through the lumen of the inflated balloon (no more than 1 mL/min with a timer). Slow the injections if the patient develops heart block, premature ventricular contractions (PVCs), or intraventricular conduction delay (IVCD). Usually, the gradient starts to decline with successful alcohol injection.
- Note: if a patient develops transient heart block or IVCD, HOLD injection for 3–5 min (balloon remains inflated) and restart if rhythm reverts to normal. STOP injection if the patient has developed persistent heart block or IVCD (even if 1, 2, or 3 cc of alcohol has been injected).
- Continue total balloon inflation time of at least 5 min (after stopping alcohol injection).
- Continue TTE monitoring and measure the LVOT gradient while the balloon is inflated and remove any residual alcohol from the lumen by an additional saline flush.

- If resting gradient <20 mmHg, deflate the balloon and finish after confirming LAD patency.
- If resting LVOT gradient >20 mmHg, consider injecting another septal artery or the same septal artery more proximally [8].
- The average number of arteries injected = 1.7; the average volume of alcohol injected = 3 mL.

Post-Procedure

- Acute procedural success (80–85%) is defined as a ≥50% reduction in peak resting or provoked LVOT gradient with a final residual resting gradient of <20 mmHg (see Fig. 32.3).
- Further reduction in LVOT gradient occurs over 3–6 months due to ventricular remodeling and basal septal thinning.

Complications

- Chest pain during the procedure (This can be usually treated with IV narcotics).
- Most common complication: 10–20% of patients develop new conduction abnormalities (RBBB, LAFB, and LBBB). 8–10% may develop complete heart block requiring permanent PM; this rate approaches 30% if baseline LBBB or RBBB is present [9].
- Ventricular fibrillation/tachycardia.
- Overall periprocedural mortality of 2%.

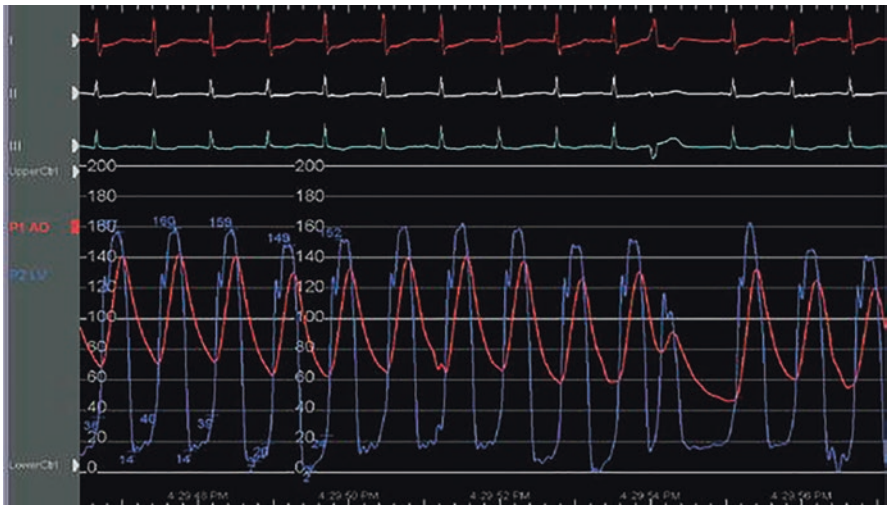

Fig. 32.3 Post-ASA gradients

Post-Procedure

- Observe in the ICU setting for at least 2 days. The temporary pacemaker is left in the patient for 48 h post-procedure.
- Post-procedural elevation of CPK (between 800 and 1200 U/L) usually occurs. The amount of elevation depends upon the amount of alcohol injected, vessel size, and method of enzyme measurement.
- The patient can be discharged home once the CK-MB level falls below <10× normal (<60 units).
- Aspirin 81 mg orally daily should be continued indefinitely.
- If a patient is on beta- and/or calcium-channel blockers, medical therapy can be reduced by discontinuation of one class if a patient is on both, or reduction in dosage if the patient is only on one class of medication.
- Repeat TTE the next day.
- Follow-up echocardiogram should be performed at 6 months and annually thereafter.
- There will be an additional 30% reduction in septal thickness over the next 4–6 weeks on follow-up echo.

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Pericardiocentesis and Balloon Pericardiotomy

33

Gurpreet S. Johal, Amit Hooda, Reza Masoomi, and Annapoorna Kini

Introduction

The pericardial space usually contains up to 50 ml of serous fluid and the pressure in this space varies with changes in intrathoracic pressure during respiration, ranging between −5 cm and +5 cm H_2O [1]. Typically, minor changes in volume and pressure are compensated for by pericardial compliance. However, rapid accumulation of even a small volume of fluid can lead to profound changes in pressure and tamponade. As the pressure in the pericardial space rises and near equalizes with the intracardiac pressure, ventricular filling is restricted, cardiac output is reduced, and hemodynamic instability ensues. This generally occurs when the intracardiac pressure reaches 15–20 mmHg [1].

Pericardiocentesis

Pericardiocentesis is percutaneous drainage of fluid from the pericardial space, and is performed for diagnostic and/or therapeutic purposes. The timing to perform the procedure ultimately depends on the clinical status of the patient.

Important hemodynamic and echocardiographic findings of cardiac tamponade are detailed below in Table 33.1.

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Table 33.1 Hemodynamic and echocardiographic findings in cardiac tamponade

Hemodynamic Findings in Cardiac Tamponade	Echocardiographic Findings in Cardiac Tamponade
Elevated right atrial pressure	Presence of pericardial effusion
Increased intra-pericardial pressure	Late diastolic collapse of RA
Equalization of left/right ventricular filling pressure	Early diastolic collapse of RV
Absent y-decent of the right atrial pressure waveform	Exaggerated respiratory variation across tricuspid/mitral valve
Paradoxical pulse	Plethoric inferior vena cava without inspiratory collapse

Adapted from Armstrong et al. [1]

Indications

- Emergent: Impending or existing cardiac tamponade.
- Elective: Diagnosis and/or therapeutic drainage to alleviate symptoms of pericardial effusion.

Contraindications

- Absolute: For emergency pericardiocentesis in hemodynamically compromised patients, there are no absolute contraindications, as withholding treatment will prove fatal.
- Relative: Aortic dissection, myocardial rupture, bleeding disorder (use of anticoagulants, platelet count <50,000), INR >3, and traumatic pericardial effusion.
- Consider a surgical approach in the following situations [1].
  - Hemopericardium due to Type A aortic dissection.
  - Hemopericardium due to traumatic injury.
  - Hemopericardium due to myocardial rupture (i.e., Post MI free wall rupture).
  - Recurrent pericardial effusion.
  - Purulent pericardial effusion.
  - Loculated pericardial effusion.
  - Small pericardial effusion.
  - Posteriorly located pericardial effusion.

Etiologies

- Most common outpatient etiologies: Metastatic cancer, tuberculosis, and uremia.
- Most common inpatient etiologies: Post-cardiac surgery and iatrogenic (following an invasive cardiac procedure).
- Other etiologies: Trauma, infection, cardiac failure, collagen vascular disease, mediastinal radiation, and hypothyroidism.

Equipment

- Local anesthetic, pericardial drainage kit which contains an 18 G needle, 10 and 20 cc syringe, 3-way stop cock, extension tubing, drainage bag, scalpel, puncture needle, 0.35 in. ×80 cm J guidewire, dilator, 8.3 F multihole pigtail or straight catheter, 2-0 silk suture, and sterile bottles for sample collection (Fig. 33.1).
- Monitoring equipment (heart rate, blood pressure, oxygen saturation, and continuous electrocardiography/telemetry).
- Code resuscitation cart.
- Combined Fluoroscopic and Echocardiography guidance is preferred.
- In life-threatening situations, pericardiocentesis can be performed at the bedside preferably under echocardiographic guidance.

Step-By-Step Approach

- Obtain informed consent (required for all elective procedures).
- Review relevant patient information (medications, allergies, labs especially CBC, coagulation parameters, etc.).

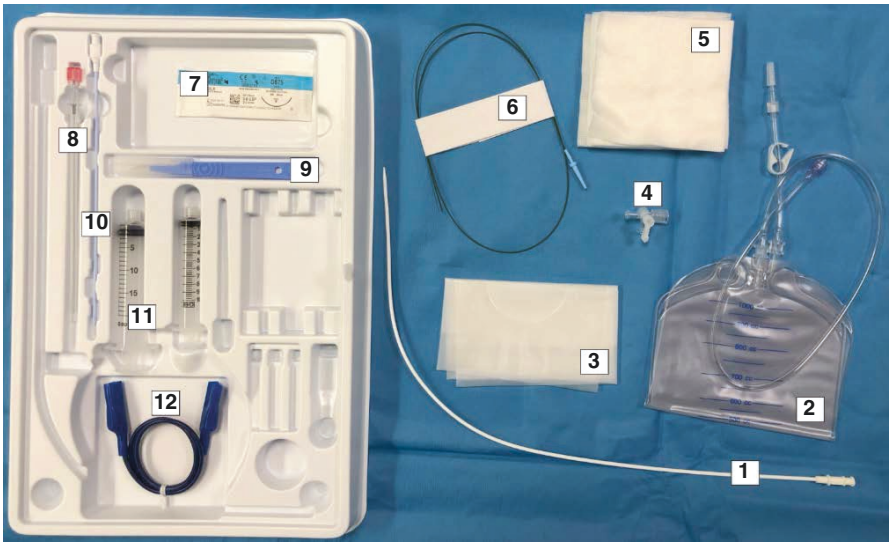

Fig. 33.1 Pericardiocentesis kit. (1) 8.3 Fr straight drainage catheter; (2) Extension tubing and 1000 cc collection bag; (3) Sterile drape; (4) 3-way stopcock; (5) Sterile gauge; (6) 0.035-inch × 80 cm J wire; (7) Silk suture; (8) 18-G Pericardiocentesis needle with stylet; (9) 11 # Scalpel; (10) Dilator; (11) 10, 20 cc Leur-lock syringe; (12) Sterile alligator clip

- Place the patient on the table or bed in a supine position. A 30–40° elevation of the head of the bed is suggested as this position brings the pericardial space closer to the anterior chest wall and allows the fluid to accumulate inferiorly.
- Clean and drape the operative area using aseptic precautions.
- Echocardiography helps identify the distance of the effusion from the skin and which entry site is most appropriate. The best site for access is where the fluid is most superficial, and safely accessible [1].
- The conventional approach is through the subxiphoid route [1]. Inject a generous amount of local anesthesia in the area about an inch below and left lateral to the xiphoid process.
- Entry occurs usually left lateral and 1–2 cm below the xiphoid process. Make a small skin incision with the #11 scalpel (Fig. 33.2).
- Use a 15 cm, 18-gauge Cook needle with a mandrel (or a 21-gauge micropuncture needle).
- Advance the needle with the mandrel in place at a 90° angle to the skin for 2–3 cm.
- Drop the angle to 15–30° and direct the needle toward the left mid clavicle under echocardiographic guidance.
- As the needle enters the pericardium, a giveaway sensation or characteristic "pop" is felt.
- Mandrel is removed, a 10 cc syringe is attached, and fluid is aspirated into the syringe.

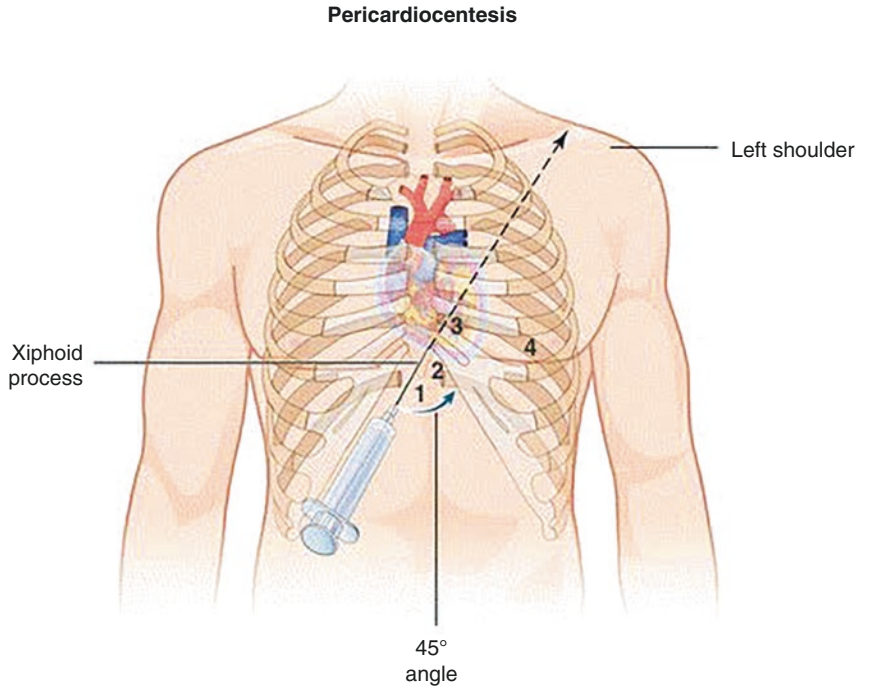

Fig. 33.2 Sites of entry: (1) Xiphoid approach. (2) Paraxiphoid approach. (3) Parasternal. (4) Apical approach

- Once the fluid is aspirated into the syringe, confirm the needle position (Table 33.2).
- Advance a 0.035-inch J wire into the pericardial space (stop if there is resistance).
- Confirm 0.035-inch J wire posterior position in the LAO view. The wire will be seen curling around the heart border toward the root (Table 33.2). Confirm guidewire position in a second orthogonal view.
- Remove the needle leaving the 0.035-inch J wire in place.
- Advance a 6–8 Fr dilator over the guidewire to make a tract in the subcutaneous tissue (give additional local anesthesia at this point if appropriate).
- A multihole 6–8 Fr pigtail drainage catheter or straight catheter is advanced over the wire into the pericardial space.
- Connect the distal end of the pigtail catheter, a 50 ml syringe, and a drainage bag to the 3-way stopcock.
- Aspirate the fluid using a 50 ml syringe and empty it in the drainage bag that is attached to the 3-way stopcock. Remember to aspirate slowly as rapid removal of fluid can precipitate acute post-procedure ventricular dysfunction.

Table 33.2 Techniques used to confirm needle position when performing pericardiocentesis

Method	Description
Echocardiography	Direct visualization of the needle position in the pericardial space
EKG signal from aspiration needle	Attaching a sterile alligator clip to the needle and V lead of an EKG monitor allows for electrocardiographic monitoring • Isoelectric ST-segment confirms the needle is in the pericardial space • ST-elevation/PVCs suggest RV entry/epicardium irritation • PR-elevation/APC's suggest RA entry
Pressure monitoring	Connect the aspiration needle, 10 ml syringe, and pressure tubing to a three-way stopcock, and transduce the pressure tubing for intrapericardial pressure waveform monitoring. • Right ventricular waveform suggests entry into the RV
Injection of agitated saline in the pericardial space	Inject 5–10 ml of agitated saline in the pericardial space and observe for the presence of bubbles in the pericardial cavity using echocardiography. • If bubbles appear in any of the cardiac chambers, it means the needle has been advanced too far. • If no bubbles are seen, it may suggest the needle is in an extra-cardiac location or the effusion is large. The needle should be repositioned and agitated saline reinjected.
Contrast injection	Outlines the border of the myocardium. • If contrast is seen within any of the cardiac chambers, it means the needle has been advanced too far.
0.035-inch J wire advancement under fluoroscopy	Wire seen wrapping around the heart within the myocardial silhouette. • The wire should advance without resistence and position confirmed in two different orthogonal views (i.e., LAO and AP).
Needle fluid aspiration	Assess the fluid; frank bloody fluid may be a sign you are in the myocardium versus a possible hemopericardium. • Features suggesting hemorrhagic pericardial fluid rather than frank blood: fluid with lower hematocrit than intravascular blood and fluid does not clot easily when placed in a Z Serum Clot Activator tube.

- Adapted from Armstrong et al. [1]

- Aspirate until there is no or minimal fluid in the pericardial cavity under echocardiography and remove the pigtail catheter. Apply a dressing at the insertion site.
- If there is a chance of re-accumulation, secure the pigtail catheter in place using a 2-0 silk suture. Place a dry gauze underneath the catheter hub to prevent skin injury and cover the skin entry site of the drain with a clear adhesive dressing. Usually, the pigtail is removed when the drainage has decreased to <50 ml over a 24 hr period.
- Send aseptically obtained pericardial fluid for biochemical, hematological, immunological, cytological, and microbiological (gram-stain, Ziehl–Neelsen stain, culture, and sensitivity) analysis if clinically indicated [1].
- Plan for pericardial window or pericardiotomy if indicated.

Post-Procedure Management

- Closely monitor the patient for clinical status changes, development of procedural complications, and record vital signs at regular intervals.
- Obtain a chest radiograph immediately after the procedure to rule out pneumothorax.
- Assure catheter drainage is set up appropriately as per one of the following methods: free drainage with gravity, intermittent manual aspiration, or continuous/intermittent suction using a water-seal device.
- Monitor the volume of pericardial fluid drainage at regular intervals.
- Flush the drainage catheter with sterile heparinized saline every 6 to 8 hr to maintain its patency.
- Follow strict aseptic protocol when manipulating the drain at all times.
- Treat pericardial pain with non-steroidal anti-inflammatory drugs or narcotic medications, unless contraindicated. If the patient is experiencing severe pain, 50 ml sterile saline or 10–20 ml of 1% Xylocaine can be injected into the pericardial space.
- Removal of the drain depends on the clinical status of the patient, volume draining, and follow-up echocardiography findings.
- If a patient develops fever/sepsis, remove the drain immediately.
- A drain should be removed when fluid draining is <25–50 ml in 24 hr.
- If fluid draining after 3 days is >75–100 ml in 24 hr, more definitive therapy may be required to prevent fluid re-accumulation after the drain is removed.
- Periodic echocardiography should be performed to screen for the re-accumulation of pericardial fluid.

Complications

- The rate of major and minor complications with echocardiographic guided pericardiocentesis is 0.7–3% and 2–3.5%, respectively.
- Refer to Table 33.3 for a list of major and minor complications.

Table 33.3 Minor and major complications of pericardiocentesis

Minor	Major
Vasovagal reaction	Ventricular tachycardia (VT)
Nonsustained VT	Infection
Catheter occlusion	Bleeding (hemothorax, hemopericardium, hemoperitoneum, or liver hematoma)
Small pneumothorax	Large pneumothorax
Chamber entry	Chamber laceration/perforation
Pleuropericardial fistula	Laceration of coronary or intercostal artery
	Injury/perforation of the diaphragm, liver, stomach, or spleen
	Death

Adapted from Armstrong et al. [1]

Pearl

- Pericardial Decompression Syndrome: Rarely, in chronic pericardial effusions, once sufficient volume of fluid has been drained to clinically alleviate cardiac tamponade, the rate at which the residual pericardial fluid is drained should be reduced to a volume not exceeding one liter per 24 hr period because a rapid rate of drainage can precipitate acute pulmonary edema, left ventricular dysfunction, and cardiogenic shock [1].

Percutaneous Balloon Pericardiotomy

Balloon pericardiotomy is a palliative procedure reserved for the management of recurrent pericardial effusions. It is performed using fluoroscopy and echocardiography as an alternative to a surgical pericardial window [2].

Indications

- Palliative procedure for recurrent pericardial effusions.
- Continuous pericardial drainage of >100 ml for 3 days.

Contraindications

- Infectious etiology of pericardial effusion.
- Major coagulation disorders.
- Effusive–constrictive pericarditis.
- Large left pleural effusion.
- Advanced respiratory insufficiency.
- History of pneumonectomy.
- If a pericardial effusion is loculated, surgical drainage may be required.

Equipment

- Local anesthetic
- Pericardiocentesis kit (as detailed above).
- Balloon 18–25 mm in size.

Procedure

- Ensure adequate local anesthesia and analgesia is given, as the procedure is painful.
- First, perform pericardiocentesis and evacuate at least 50% of the pericardial fluid.
- Exchange for an Amplatz stiff wire over a 7F dilator and ensure its position in the pericardial space.
- Advance serial dilators (up to 14 F to create a good tract in the skin and subcutaneous tissue to allow for easy passage of the balloon) over the Amplatz stiff wire and then position the balloon (18 to 25 mm in diameter/30 to 40 mm in length) in the pericardium well beyond the entry point.
- A 60 cc syringe with 1:3 diluted contrast is used to inflate the balloon under fluoroscopic guidance.
- The balloon is partially inflated to 2 atm and pulled back until the distal tip is squeezed, forming a waist.
- Pull the balloon back further so the waist is at the center of the balloon and across the inferior pericardial border in the AP or lateral view. While doing this, the entire balloon should remain under the skin.
- The balloon is slowly inflated to its nominal pressure so the waist disappears and the balloon is fully inflated (Figs. 33.3 and 33.4).
- Pericardial fluid may be blood-stained following balloon pericardiotomy and a pigtail catheter may be reintroduced to drain the remaining fluid.
- Remove the deflated balloon and guidewire.
- Apply an occlusive sterile dressing at the operative site.
- Modification of this technique involving the use of a double-balloon and Inoue balloon has been described in the literature.

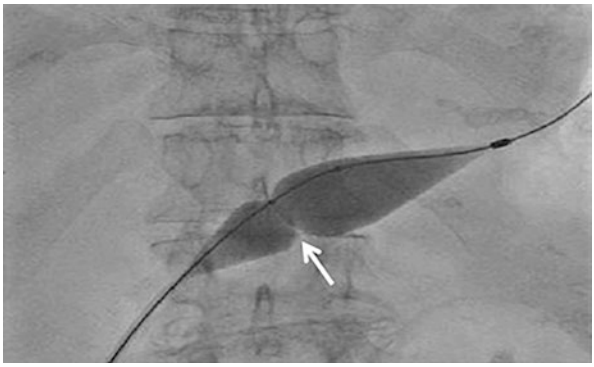

Fig. 33.3 Tight waist at the pericardium (*arrow*) is seen during initial balloon inflation of a 26 mm/40 mm ATLAS balloon

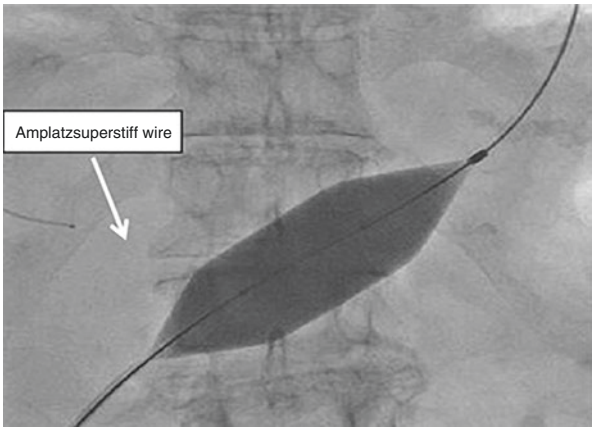

Fig. 33.4 Disappearance of the waist with full inflation of a 26 mm/40 mm ATLAS balloon

Post-Procedure

- Monitor patients on telemetry. Request for a CXR 24 hr after and an echocardiogram 48 hr after the procedure to rule out significant left-sided pleural effusion due to pericardial fluid drainage in the pleural space and recurrent pericardial effusion, respectively.

Pericardial Window

A surgical pericardial window is usually created for drainage of symptomatic, recurrent pericardial effusions especially in cases of malignancy and tuberculosis. It involves the excision of a portion of the pericardium, which allows the effusion to drain continuously into the peritoneum or chest. It is a safe technique with a low complication rate (1.1%), and a low recurrence rate [1]. It can be done in several ways:

- Via a small subxiphoid incision.
- Thoracotomy.
- Thoracotomy video-assisted thoracoscopy (VATS).

References

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